

Threshold Dynamics and Stability of Epidemic Models with Periodically Varying Contact Rates

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Abstract:

The fluctuations in human contact patterns can become a significant factor in the transmission and persistence of infectious diseases. In this work, we have developed a nonlinear SEIQR epidemic model to study the dynamics of epidemic with periodically varying contact rates. We break down the population into susceptible, exposed, infectious, quarantined, and recovered bodies and we consider demographic turnover, disease-causing mortality, disease progression from exposure to infection, and isolation of the infectious population. The contact rate is assumed to vary in time, and this model can be used to explain seasonal changes in population interaction. We have established the properties of the model, such as positivity and boundedness of solutions. A threshold quantity for disease invasions and extinction is derived from the linearized infection subsystem, and the stability of the disease-free state in the periodic setting is investigated. The impact of periodic forcing, isolation, progression, and recovery parameters on the persistence of the epidemic is investigated. We conduct numerical simulations to illustrate some of our analytical results and to show how changes in the amplitude and frequency of contact-rate fluctuations have the effect of time-dependent changes in the persistence of infection. Our findings give us a mathematical framework for how the periodic change in contact patterns and intervention through isolation can affect the persistence of the epidemic and its stability to a certain extent.

Keywords: SEIQR epidemic model; Periodic contact rate; Threshold dynamics; Stability analysis; Periodic forcing; Disease persistence; Quarantine; Epidemic modeling; Mathematical epidemiology; Floquet theory.

1. Introduction

Mathematical models have become a fundamental tool for understanding the transmission and development of infectious diseases and for understanding the factors that can affect its persistence, decline, or disappearance. Epidemic models provide a systematic way of describing interactions between individuals, to measure the mechanisms of disease transmission and to study the impact of epidemiological interventions. Since the development of the classical susceptible-infectious-recovered (SIR) model, many new compartmental models have been developed with additional epidemiological aspects like latency, demographic turnover, vaccination, quarantine, hospitalization, temporary immunity, and disease-induced mortality. These new features made mathematical models more and more useful to model epidemic processes in populations where disease transmission is influenced by multiple interacting mechanisms.

A key assumption in most classical epidemic models is that the transmission or contact rate is constant over time. This assumption simplifies the way that they are done, but it may not account for the temporal variability that we may observe in real epidemic systems. Human contact can change dramatically due to seasonal conditions, climate variations, school and work schedules, public gatherings, behavioral adaptation, intervention policies, and changes in population mobility. So the effective contact rate between susceptible

and infectious people may change over time as well. This temporal change can have a major impact on the growth, persistence, and long-term behavior of an infectious disease in a biological way. In order to have more realistic disease dynamics, it is important to incorporate time-dependent transmission into epidemic models.

Periodic variation provides a simple mathematical framework to describe recurrent changes in contact patterns. A periodic variation of contact rate can capture such phenomena as seasonal increases of social interaction, recurring patterns of behavior, and regular population mixing. A common formulation is

$$\beta(t) = \beta_0[1 + \varepsilon \cos(\omega t + \phi)],$$

where β_0 is the mean contact transmission rate, ε the amplitude of periodic variation, ω is the forcing frequency, and ϕ is the phase. Such a formulation allows the epidemic system to have periods of relatively high and low transmission but still have a well-defined long-term structure of periodic variation. The mathematical system is nonautonomous, and it is always difficult to analyze without tools not used when developing autonomous epidemic models.

Another key characteristic of epidemic transmission is the delay in which people become infectious. Infected people are not easily infected by a pathogen, and so an exposed compartment is a more realistic representation of the disease process. In addition, isolation or quarantine may affect the contagious individuals' contribution to the spread. Once an infectious person is identified and isolated, their contact with susceptible persons will be much less. The interaction between exposure, infectiousness, isolation, and recovery can therefore influence the threshold conditions for disease invasion and persistence.

Motivated by these considerations, we develop a nonlinear susceptible-exposed-infectious-quarantined-recovered (SEIQR) epidemic model with a periodically varying contact rate. Unlike the simplest SIR model, our model includes an exposed population, isolation of the infectious population, recruitment and natural mortality, recovery, and disease-induced mortality. These processes help us differentiate between people who have acquired infection but are not yet infectious, and those who are actively infectious and who are under isolation from the disease. The periodic contact rate also provides another time frame within which the interaction between the population and the disease can influence the epidemic.

The mathematical analysis of epidemic models with periodic coefficients differs from the approach for autonomous systems. In an autonomous model, equilibrium points are often used to describe the long-term behavior of the system. In the case of a periodically forced epidemic system, the disease-free state and, if present, endemic behavior may be periodic rather than stationary. Therefore, the stability and invasion properties of the system must be analyzed in terms of its time scale. Linearizing around the disease-free state leads to a periodic infection subsystem for which the long-term growth or decay of infection can be characterized via threshold concepts and Floquet theory. This is a natural mathematical basis for deciding if a small introduction of infection can invade the susceptible population for periodic contact patterns.

Threshold quantities play an important role in epidemic modeling because they make a mathematical distinction between different disease cases. In constant-parameter models, the basic reproduction number is generally used to determine if infection is going to disappear or establish itself in a population. Under periodic forcing, however, the invasion process is affected by the whole time profile of the transmission coefficient and not only by a single constant value. The threshold of the infection subsystem has to be related to the periodic structure of the infection subsystem. Establishing such a threshold and relating it to the stability of the disease-free periodic state is one of the main mathematical objectives of the present work. We also see that quarantine can be used to adapt the threshold behavior. Increasing the rate at which infectious individuals are isolated may lead to less interaction time with susceptible individuals and thus alter the invasion characteristics of the disease. And the effectiveness of isolation may also depend on the frequency of contact. For example, if it seems to be effective when you have a constant contact rate, the long-term behavior changes

when the contact is more or less intermittent. The interaction of the interventions in time is therefore critical in order to determine the control of an epidemic in different environments.

We now discuss the analytical and numerical aspects of this problem. First, the basic properties of our new SEIQR system (positivity and boundedness of solutions) are established so that the mathematical model can be used to characterize epidemiology. Second, the infection subsystem around the disease-free state is formulated and the appropriate threshold quantity is investigated. Third, we study the stability of the disease-free state according to periodic differential system theory. We particularly study the role of periodic forcing in controlling the growth or decay of infection. Finally, numerical simulations are used to complement the analytical results and to illustrate the effects of the amplitude and frequency of periodic contact variation, as well as epidemiological parameters such as the isolation and recovery rates. The main contribution of this work is thus to integrate periodic contact variation, latent infection, isolation, demographic turnover, and threshold stability in a unified SEIQR framework. Rather than treating time variation as a numerical perturbation of a constant transmission rate, the periodic structure is considered to be intrinsic to the epidemic dynamics. This approach provides a mathematical basis to investigate how a pattern of population contact variations with the disease progression and isolation is coupled to determine the invasion, persistence and stability of the epidemic.

The rest of the paper is organized as follows. The next section develops the periodically forced SEIQR model and establishes the basic mathematical properties. The threshold dynamics and disease invasion and extinction dynamics are developed in the next section. The stability properties of disease-free and relevant periodic states are studied in the appropriate way based on the theory for periodic systems. Numerical simulations are then presented to illustrate the theoretical results and the parameter-dependent epidemic behavior, followed by a discussion of the main findings and the conclusion.

2. Mathematical Model Formulation and Basic Properties

Consider a closed heterogeneous population in which there are 5 epidemiological compartments, namely susceptible people ($S(t)$), exposed people ($E(t)$), infectious people ($I(t)$), quarantined people ($Q(t)$), and recovered people ($R(t)$). The total population at time (t) is given by

$$N(t) = S(t) + E(t) + I(t) + Q(t) + R(t).$$

The susceptible population is recruited at a constant rate λ . Susceptible individuals may get infected when they are in contact with infectious individuals and come into the exposed compartment. The exposed individuals have acquired the infection but are not yet capable of transmitting it at the same level as infectious individuals. After an average latent period, exposed individuals progress to the infectious compartment. Infectious individuals may either recover naturally, be isolated, or die as a result of the disease. People in quarantine are later found to recover or die from the disease. Recovered patients are assumed to acquire immunity over the period of time considered in the model. To incorporate temporal variation in human contact patterns, we assume that the effective contact rate is periodic. We consider

$$\beta(t) = \beta_0[1 + \varepsilon \cos(\omega t + \phi)],$$

where $(\beta_0 > 0)$ is the mean contact-related transmission coefficient, $(0 \leq \varepsilon < 1)$ is the amplitude of periodic variation, $(\omega > 0)$ is the angular frequency of contact fluctuations, and (ϕ) is the phase. The restriction $(0 \leq \varepsilon < 1)$ guarantees that $(\beta(t))$ remains positive for all (t) . The period of the forcing is

$$T = \frac{2\pi}{\omega}.$$

Thus,

$$\beta(t + T) = \beta(t),$$

and the epidemic system becomes a (T) –periodic nonautonomous dynamical system.

We assume that new infections occur through frequency-dependent transmission. Therefore, the force of infection is represented by

$$\lambda(t) = \beta(t) \frac{I(t)}{N(t)}.$$

Accordingly, the incidence of new infections is

$$\lambda(t)S(t) = \beta(t) \frac{S(t)I(t)}{N(t)}.$$

The parameter ($\sigma > 0$) is the rate at which exposed individuals become infectious, and ($1/\sigma$) is the average time until the exposed individuals become infectious. Infectious individuals are isolated at **rate(κ)**, **while(γ)and(γ_q)** denote the recovery rates of infectious and quarantined individuals, respectively. The parameters (δ) and (δ_q) represent disease-induced mortality rates in the infectious and quarantined compartments, and (μ) is the natural mortality rate.

Given these assumptions, the periodically forced SEIQR model is given by

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \beta(t) \frac{SI}{N} - \mu S, \\ \frac{dE}{dt} &= \beta(t) \frac{SI}{N} - (\sigma + \mu)E, \\ \frac{dI}{dt} &= \sigma E - (\gamma + \kappa + \delta + \mu)I, \\ \frac{dQ}{dt} &= \kappa I - (\gamma_q + \delta_q + \mu)Q, \\ \frac{dR}{dt} &= \gamma I + \gamma_q Q - \mu R.\end{aligned}$$

For convenience, define

$$\begin{aligned}a &= \sigma + \mu, \\ b &= \gamma + \kappa + \delta + \mu, \\ \text{and} \\ c &= \gamma_q + \delta_q + \mu.\end{aligned}$$

The model can therefore be written as

$$\begin{aligned}\frac{dS}{dt} &= \Lambda - \beta(t) \frac{SI}{N} - \mu S, \\ \frac{dE}{dt} &= \beta(t) \frac{SI}{N} - aE, \\ \frac{dI}{dt} &= \sigma E - bI, \\ \frac{dQ}{dt} &= \kappa I - cQ, \\ \frac{dR}{dt} &= \gamma I + \gamma_q Q - \mu R.\end{aligned}$$

Model Assumptions

We make the following assumptions. The population is sufficiently well mixed so that the frequency-dependent incidence term provides an appropriate approximation to infection-producing contacts. Infected individuals are introduced into the population as susceptible and are subject to natural mortality in all compartments. Newly infected individuals first enter the exposed compartment and subsequently progress to infectiousness at rate σ . Infectious individuals can be identified and transferred to quarantine at rate κ . Quarantined individuals do not contribute directly to the principal infection term, reflecting the

assumption that isolation substantially reduces their effective contact with susceptible individuals. Recovery from the infectious and quarantined classes provides immunity during the period under consideration. Disease-induced mortality is allowed in both infectious and quarantined classes.

The periodic function $(\beta(t))$ represents recurrent variation in the intensity of effective contacts. Such variation may mathematically represent seasonal changes, recurring social activity, periodic population mixing, or other regularly fluctuating factors. The model does not require the periodic variation to correspond to a particular biological mechanism; rather, it provides a general deterministic representation of recurrent changes in transmission opportunities.

Positivity of Solutions

For an epidemiologically meaningful model, all compartment populations must remain nonnegative whenever the initial population is nonnegative. Let

$$S(0) > 0, \quad E(0) \geq 0, \quad I(0) \geq 0, \quad Q(0) \geq 0, \quad R(0) \geq 0.$$

On the boundary $(S=0)$, equation (1) gives

$$\left. \frac{dS}{dt} \right|_{S=0} = \Lambda > 0.$$

Similarly, when $(E = 0)$,

$$\left. \frac{dE}{dt} \right|_{E=0} = \beta(t) \frac{SI}{N} \geq 0.$$

For $(I = 0)$,

$$\left. \frac{dI}{dt} \right|_{I=0} = \sigma E \geq 0,$$

while for $(Q = 0)$,

$$\left. \frac{dQ}{dt} \right|_{Q=0} = \kappa I \geq 0.$$

Finally, when $(R = 0)$,

$$\left. \frac{dR}{dt} \right|_{R=0} = \gamma I + \gamma_q Q \geq 0.$$

Thus, the vector field points inward on every boundary of the nonnegative region. Consequently, solutions originating in the nonnegative orthant remain nonnegative for all subsequent times for which the solution exists. Hence,

$$S(t), E(t), I(t), Q(t), R(t) \geq 0$$

for all $(t \geq 0)$.

Boundedness of the Total Population

Adding equations (1)–(5) gives

$$\frac{dN}{dt} = \Lambda - \mu N - \delta I - \delta_q Q.$$

Since $(I, Q \geq 0)$,

$$\frac{dN}{dt} \leq \Lambda - \mu N.$$

The comparison equation

$$\frac{dN}{dt} = \Lambda - \mu N$$

has solution

$$N(t) = N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}).$$

Hence,

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t}).$$

In particular,

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Thus, the total population remains bounded for all future time. The biologically feasible region can therefore be defined as

$$\Omega = \left\{ (S, E, I, Q, R) \in \mathbb{R}_+^5 : N \leq \frac{\Lambda}{\mu} \right\}.$$

The set (Ω) is positively invariant under the dynamics of system (1)–(5). This establishes that the model is both mathematically well posed within the biologically relevant region and epidemiologically meaningful.

Periodic Structure of the Model

Because

$$\beta(t + T) = \beta(t), \quad T = \frac{2\pi}{\omega},$$

the right-hand side of system (1)–(5) is periodic in time. Therefore, the system is a nonlinear periodic differential system rather than an autonomous epidemic system.

This distinction is important for the subsequent threshold analysis. In particular, the long-term infection dynamics cannot, in general, be characterized solely by the eigenvalues of a constant Jacobian matrix evaluated at an equilibrium. Instead, the linearized infection subsystem around the disease-free state is itself periodic, and its stability is naturally investigated through the fundamental solution matrix and its associated Floquet multipliers. The periodic formulation also allows us to model two different effects of time contact variation. The parameter ε controls the magnitude of fluctuations around the mean contact rate, whereas ω controls how rapidly they occur. As a result, two epidemic systems with the same mean contact rate β_0 will have different dynamical behavior when their amplitudes or frequencies are different, and this will be in the focus of our threshold and stability analysis in the following sections. The model formulation thus provides a unified framework in which disease progression, demographic processes, isolation, and periodic variation in contact patterns interact. The next step is to find the disease-free periodic state and derive an appropriate threshold quantity that determines whether infection can invade the population.

Threshold Dynamics and Disease Invasion

The purpose of this section is to determine when an introduced infection disappears from the population or successfully enters and stays there. Since the contact rate is periodic, the infection dynamics are driven by a time-dependent linear system near the disease-free state. The appropriate threshold must take into account the complete periodic transmission profile rather than the mean value of the contact rate.

Disease-Free State

In the absence of infection,

$$E(t) = I(t) = Q(t) = 0.$$

From equation (1), the susceptible population satisfies

$$\frac{dS}{dt} = \Lambda - \mu S.$$

Hence,

$$S(t) \rightarrow S^0 = \frac{\Lambda}{\mu}$$

as $(t \rightarrow \infty)$. Since $(R(t))$ also approaches zero in the absence of infection, the disease-free state is

$$\mathcal{E}_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \right).$$

Although the transmission coefficient $(\beta(t))$ is periodic, the disease-free population itself is constant because no infection is present. Thus, $(\mathcal{E}_0)$ is the reference state around which the invasion dynamics can be explored. At the disease-free state,

$$N^0 = \frac{\Lambda}{\mu}.$$

Therefore,

$$\frac{S^0}{N^0} = 1.$$

The force of infection near the disease-free state reduces to

$$\lambda(t) \approx \beta(t)I(t).$$

This observation greatly simplifies the linearization of the infected compartments.

Relation Between the Threshold and Epidemic Control

The threshold quantity is affected by several epidemiological mechanisms. An increase in the mean contact rate (β_0) generally increases the generation of new infections and consequently increases the invasion potential of the disease. Similarly, a larger amplitude (ϵ) produces stronger fluctuations between high-contact and low-contact phases. The resulting effect on the threshold is determined by the interaction between the nonlinear epidemic dynamics and the temporal structure of transmission and therefore should not be inferred solely from the average value of $(\beta(t))$. The isolation parameter (κ) has a direct effect on the infectious compartment through

$$\frac{dI}{dt} = \sigma E - (\gamma + \kappa + \delta + \mu)I.$$

An increase in (κ) increases the rate at which infectious individuals leave the actively infectious compartment. Consequently, their average time available for effective transmission is reduced. This mechanism can shift the system toward the disease-free regime. Similarly, an increase in the recovery rate (γ) decreases the duration of infectiousness, whereas an increase in the progression rate (σ) changes the rate at which exposed individuals enter the infectious class. The mortality parameters also influence the residence time of infected individuals and therefore contribute to the overall threshold behavior. The periodic forcing frequency (ω) introduces another important mechanism. Two systems may have identical values of (β_0) and (ϵ) but different forcing frequencies. In one system, the period of transmission fluctuation may be comparable to the characteristic epidemic time scale, while in another it may be much shorter or much longer. Consequently, the epidemic response depends not only on the magnitude of periodic forcing but also on its temporal scale.

Disease Extinction Regime

Suppose that the periodic threshold satisfies

$$\mathcal{R}_p < 1.$$

Equivalently, suppose that the dominant Floquet multiplier satisfies

$$\rho_* < 1.$$

Then small perturbations in the infection-free population decay under the linearized dynamics. In epidemiological terms, each successive transmission cycle leads to insufficient infection growth to compensate for removal through recovery, isolation, natural mortality, and disease-induced mortality.

Under the stability conditions for the whole nonlinear system, the disease-free state is locally asymptotically stable. Thus,

$$\begin{aligned} E(t) &\rightarrow 0, \\ I(t) &\rightarrow 0, \\ Q(t) &\rightarrow 0 \end{aligned}$$

as ($t \rightarrow \infty$) for certain initial conditions that are close to the disease-free state.

The susceptible population approaches

$$S(t) \rightarrow \frac{\Lambda}{\mu},$$

once the infected compartments disappear, and the population returns asymptotically to the disease-free configuration.

Hence, the condition

$$\mathcal{R}_p < 1$$

provides a mathematical criterion for the failure of infection to establish itself in the population.

Disease Invasion Regime

In contrast, suppose that

$$\mathcal{R}_p > 1$$

or, equivalently, that

$$\rho_* > 1.$$

Then a sufficiently small introduction of infection can increase over time. The disease-free state is unstable with respect to infection, so that the susceptible population can support initial epidemic growth. This does not imply that infection grows without bound. The nonlinear feedback produced by depletion of susceptible individuals, recovery, isolation, and mortality eventually modifies the growth rate. Instead, the threshold condition indicates the ability of infection to invade the disease-free population. For a persistent epidemic, the nonlinear system may approach a positive periodic solution or a bounded long-term one. The existence and stability of a periodic endemic state need to be considered beyond the linear invasion criterion and is considered in the stability analysis.

Threshold Interpretation

The threshold structure can therefore be summarized as

$$\mathcal{R}_p < 1$$

$\Rightarrow$

infection extinction near the disease-free state

and

$$\mathcal{R}_p > 1$$

$\Rightarrow$

disease-free state loses stability and invasion is possible.

The equality case,

$$\mathcal{R}_p = 1,$$

represents the critical threshold at which the dominant infection mode neither grows nor decays exponentially over one forcing cycle. In this case, small changes in contact intensity, isolation, recovery, or forcing characteristics may move the system from one dynamical regime to the other. The threshold analysis shows that epidemic control in a periodically varying environment cannot be defined solely by the average transmission rate. The amplitude and frequency of the recurrent contact fluctuations, along with the rates of progression, isolation, recovery, and removal, together determine the invasion behavior. This provides the mathematical basis for the stability analysis in the next section, where the Floquet structure of the periodic system and the stability of the disease-free and persistent regimes are studied in more detail.

Stability Analysis

The threshold condition in the previous section determines whether infection can invade the disease-free population. In this section we study the corresponding stability properties of the periodically forced SEIQR system. Since the transmission coefficient is periodic, the disease-free state must be studied in the language of periodic differential systems instead of simply using the eigenvalues of a constant Jacobian matrix. At the disease-free state, the susceptible population is at its demographic equilibrium and the infected compartments disappear. The dynamics of a small infection introduced into this population is governed by the periodic infection subsystem obtained by linearizing the model around the disease-free state. The resulting coefficient matrix is periodic with the same period as the contact rate. This periodic structure is responsible for the characteristic differences between the present model and an autonomous epidemic model.

Local Stability of the Disease-Free State

Let the infection-free state be denoted by $(\mathcal{E}_0)$. Let us consider a small perturbation in the exposed and infectious populations. The linearized system is then related to whether the perturbation increases or decreases with time. Since the coefficient matrix is $(T) - \text{periodic}$, the stability is determined by the Floquet multipliers of one full forcing cycle. If every Floquet multiplier has a modulus less than one, the small infection perturbation decreases after subsequent periods. In fact, the disease-free state is locally asymptotically stable. The dominant Floquet multiplier is the most important stability measure. If its modulus is below unity, the infection cannot replace itself over successive transmission cycles. At that point the exposed and infectious populations gradually decline and the system returns to the disease-free state. If the dominant Floquet multiplier is greater than one, an initial small infection perturbation grows in the next few cycles. The disease-free state becomes unstable with respect to the infection. This is the invasion regime we found through threshold analysis. Therefore, the threshold and stability criteria are closely related. The threshold below the critical value is stability of the disease-free state, while a threshold above the critical value is stability loss.

Stability Under Periodic Contact Variation

The periodic contact function plays a crucial role in stability. When the amplitude of the periodic variation is small, the epidemic system remains relatively close to the corresponding constant-contact model. As the amplitude increases, the difference between high-contact and low-contact phases becomes more pronounced. The mass production of new exposed individuals increases during high-contact periods so that the infectious population is increased for a short time. During low-contact periods the transmission is reduced and the removal mechanisms of isolation and recovery are much more important. The long-term stability of the system depends on the cumulative effect of these alternating phases rather than on any single instantaneous value of the contact rate. An epidemic can have very large short-run increases in infection, but still be asymptotically stable if the overall periodic growth factor is not above the stability threshold. The forcing frequency is also important here. If the contact conditions change slowly compared to the typical epidemic time scale, the

infection dynamics have sufficient time to respond to individual high- and low-contact phases. Rapid fluctuations may have a different response because the epidemic compartments cannot immediately follow every variation in the contact environment. Thus the same average contact rate can lead to different transient and long-term behavior when the amplitude or frequency of periodic forcing changes.

Influence of Isolation on Stability

Isolation is a crucial control mechanism in the proposed SEIQR system. Infectious individuals leave the actively infectious compartment at a rate (κ), thereby reducing the duration during which they can contribute directly to new infections. As (κ) increases, infectious individuals are more removed from the transmission process. From the point of view of the linearized infection subsystem, this increases the effective removal rate from the infectious class and reduces infection growth potential. This mechanism is especially important during high-contact phases. When the contact rate is maximum, fast isolation can limit the time that infectious individuals are involved in transmission. Hence, isolation can reduce the amplification caused by periodic increases in contact intensity. The model thus provides a mathematical basis for studying the relationship between recurring transmission pressure and intervention intensity. Rather than considering isolation independently of seasonal or behavioral variation, we now allow both mechanisms to operate in parallel.

Stability and Recovery

The recovery rate also directly affects the stability of the infection-free state. A higher recovery rate reduces the average residence time of infectious individuals and increases the rate at which infection is removed from the actively infectious compartment. When recovery is sufficiently fast relative to infection generation, the infection perturbation is lost from one forcing cycle to the next. Slow recovery in contrast allows for infectious individuals to linger in the transmission process longer and may help them persist when contact rates rise at a time. The combined effects of recovery and isolation are therefore important. Both mechanisms remove individuals from the infectious class, but they do so in different epidemiological ways. Recovery removes infectious individuals from the contagious class by disease resolution, and isolation reduces their opportunity for effective contact while they remain infected.

Stability of Persistent Periodic Dynamics

When the invasion threshold exceeds its critical value, the disease-free state is unstable. The nonlinear nature of the model prevents the infection from growing forever in which susceptible individuals are progressively depleted and infected individuals are continuously removed from the disease through isolation, recovery, and mortality. As long as the parameters are suitable, the system can therefore approach a positive periodic regime in which the epidemiological variables oscillate in time with the forcing period or integer multiple of that period. This is a persistent epidemic whose prevalence varies in time with periodic contact conditions. The stability of a positive periodic solution can be studied again using Floquet theory. A periodic endemic solution is locally asymptotically stable when the nontrivial Floquet multipliers associated with perturbations around that solution lie inside the unit circle. In this case small deviations from the periodic epidemic trajectory decay over time. This behavior is epidemiologically significant as it indicates that periodic fluctuations in contact patterns need not eliminate the disease. Instead, they can generate sustained oscillations around a positive mean level of infection.

Combined Role of Model Parameters

The stability behaviour of the model depends on the interaction of several parameters rather than the single transmission coefficient. The mean contact rate determines the overall transmission intensity and the amplitude and frequency determine the temporal structure of contact variation. The progression rate

determines how rapidly individuals exposed to contact become infectious, while recovery and isolation determine how quickly infectious individuals leave the main transmission path. Disease-induced mortality also changes the residence time of infected people, while demographic recruitment and natural mortality determine the size and turnover of the susceptible population. Such mechanisms can vary in competing directions. For example, a high mean contact rate may favor disease invasion, but a high isolation rate can counter the effect. Likewise, a strong periodic fluctuation can produce large temporary outbreaks without necessarily being long-term if recovery and isolation are effective enough. And of course this is all of course why we should focus on the complete periodic system rather than each parameter separately.

Stability Interpretation

The mathematical results can be interpreted in terms of two main dynamical regimes. In the case when the dominant Floquet multiplier is below unity, the infection perturbations decay and the disease-free state is locally stable. If the dominant multiplier is above unity, however, then the disease-free state becomes unstable and the infection can invade the population. The transition between these regimes is governed by the periodic threshold identified previously. The mean contact intensity, forcing amplitude, forcing frequency, isolation, progression, and recovery can shift the system across this threshold. Thus, periodic variation in contact patterns is not merely a source of fluctuations around an otherwise unchanged epidemic trajectory. It can change the stability structure of the system itself and therefore change whether infection dies out or creates persistent dynamics. The numerical analysis presented in the next section will illustrate these theoretical results by examining the time behavior of susceptible, exposed, infectious, quarantined, and recovered populations in various periodic forcing scenarios.

Numerical Experiments and Discussion

The analytical results show that the long-term behaviour of the proposed SEIQR epidemic model is determined by the interaction between periodic contact variation and epidemiological removal mechanisms. We now use numerical simulations to illustrate these results, and to study the effect of periodic contact variation on epidemic trajectories. The simulations also demonstrate how isolation and recovery affect disease persistence. For numerical experiments we chose these parameters to represent a biologically plausible epidemic setting and to be consistent with the mathematical assumptions. The numerical values are not for a specific disease or geographic population, but rather to show the qualitative behavior that we can expect from this analytical model. The initial population is assumed to have a small infectious introduction, and the remaining population is generally divided into susceptible and exposed classes. The periodically varying contact rate is given by

$$\beta(t) = \beta_0[1 + \epsilon \cos(\omega t + \phi)],$$

where the mean contact intensity is (β_0), the strength of periodic variation is (ϵ), and the temporal frequency is (ω). Numerical solutions are obtained over a long-time interval so that transient and asymptotic behavior can be observed.

Baseline Epidemic Dynamics

The first simulation considers the baseline parameter set with a moderate amplitude of periodic contact variation. The trajectories of the five epidemiological compartments show the typical response of the system to recurrent changes in contact intensity. The susceptible population decreases as infection spreads through the population. The exposed population increases as newly infected individuals enter the latent stage. After the corresponding progression period, the infectious population starts to increase. The quarantined population follows the infectious population with a delay determined by the isolation rate. As the infection is removed through recovery and isolation, the growth of the infected compartments is eventually limited. Whenever

periodic forcing occurs, the trajectories generally do not converge monotonically to constant values. Instead, the infected compartments could exhibit recurrent oscillations reflecting the periodic changes in the contact environment. These oscillations are an example of the nonautonomous nature of our model. Higher contact intensity leads to a higher rate of infection, whereas low contact intensity leads to a lesser rate of new infections. The resulting epidemic trajectory thus contains both transient epidemic behavior and periodic variation.

Effect of Periodic Amplitude

The amplitude (ϵ) determines the magnitude of variation around the mean contact rate. When

$$\epsilon = 0,$$

the contact rate is constant and the model reduces to the corresponding autonomous SEIQR system. This case provides an important reference solution to evaluate the effect of periodic forcing. If the positive value of (ϵ) is small, the epidemic trajectories have relatively weak periodic oscillations. When (ϵ) is large, the difference between high and low contact phases is more pronounced and hence the infectious and exposed populations might experience larger recurrent peaks. The simulations are expected to show that the maximum prevalence can increase significantly during high-contact phases even if the mean contact rate is unchanged. This highlights an important feature of periodic epidemic systems: two populations with the same average transmission intensity can experience different temporal epidemic profiles when their contact variability differs. However, a larger amplitude does not imply permanent disease persistence. In any case, the ultimate outcome is determined by the threshold and stability of the whole periodic system. So numerical oscillations should be interpreted together with the analytical threshold results.

Effect of Forcing Frequency

The frequency parameter (ω) determines how fast the contact rate changes. Numerical experiments with different values of (ω) provide insight into the relationship between the time scale of contact variation and the intrinsic time scales of disease progression and removal. The epidemic system has enough time to respond to individual high-contact and low-contact phases at relatively slow forcing. This can generate waves in the infectious population and peaks occur after periods of elevated contact. As the forcing grows faster, the epidemic compartments may respond less strongly to individual fluctuations. Instead, the system can exhibit a smoother averaged response. However, the exact behavior depends on the relationship between the forcing period and the characteristic time scales associated with exposure, infection, recovery, and isolation. This result shows that periodicity should not only be characterized by its amplitude. The temporal frequency of the forcing can also influence epidemic behavior and may change the stability characteristics of the system.

Effect of Isolation

We next consider the isolation rate (κ), thus examining how quickly identifying and isolating infectious individuals can affect the progression of an epidemic. For a relatively low isolation rate, infectious individuals remain in the active infectious compartment for a longer period of time. The transmission can continue longer during high-contact phases, leading to higher infection peaks. As (κ) increases, more infectious individuals are transferred to the quarantined area. The infectious population decreases more quickly, and the quarantined population increases. Since quarantined individuals are not expected to be responsible for the main transmission process, this process reduces the effective time of contact between the infectious individuals. The simulations thus illustrate the stabilizing role of isolation. Under otherwise identical periodic forcing, stronger isolation can lower the amplitude of epidemic oscillations and can shift the system towards the disease-free regime when the corresponding threshold condition is satisfied. A key feature is that isolation is

most beneficial when the contact rate is high as well. Rapid removal of infectious individuals during this time period reduces the time when high contact intensity can lead to additional infections.

Effect of Recovery

The recovery rate is another important mechanism for controlling infection. A higher recovery rate reduces the average duration of the infectious stage and hence the transmission. We performed numerical simulations with different values of (γ), and found that faster recovery reduces the magnitude and duration of infection peaks. As a result, the exposed and infectious populations return to low levels more rapidly. When recovery is fast enough, the removal of infectious individuals can compensate for the higher transmission during periodic high-contact phases. In this case, the infection perturbation decreases from one cycle to the next. On the other hand, a low recovery rate leads to long-term stay in the infectious compartment. This can lead to periodic contact peaks and thus longer epidemic oscillations.

Comparison Between Constant and Periodic Contact Rates

A particularly meaningful numerical experiment compares the model with the constant contact rate and with the periodically varying contact rate.

For the constant-contact case,

$$\varepsilon = 0,$$

and therefore

$$\beta(t) = \beta_0.$$

For the periodic case,

$$\varepsilon > 0.$$

For example, keeping the mean contact rate fixed means that we can isolate temporal variability. The simulations show that the constant-contact model generally leads to a smoother epidemic trajectory, while the periodically forced model can lead to recurrent fluctuations in infection prevalence. The magnitude and persistence of these oscillations will depend on the forcing amplitude and frequency as well as the epidemiological parameters. This comparison demonstrates why replacing a periodically varying contact process by its mean value can overlook important temporal features of epidemic dynamics. Although the mean transmission intensity will be useful information, it does not necessarily represent the timing and magnitude of infection peaks.

Threshold Regimes

To illustrate the theoretical threshold results, numerical experiments can be performed for parameter sets that represent three regimes:

$$\mathcal{R}_p < 1,$$

$$\mathcal{R}_p \approx 1,$$

$$\mathcal{R}_p > 1.$$

When the threshold is below unity, the exposed, infectious and quarantined populations gradually decline despite periodic change in contact intensity. The oscillations become smaller, showing local stability of the disease-free state. Near the critical threshold, the infection dynamics becomes particularly sensitive to parameter changes. Small variations in contact intensity, isolation or recovery can lead to significant changes in the long-term trajectory. This regime is particularly relevant to understand the boundary between disease extinction and persistence. When the threshold is higher than unity the disease-free state becomes unstable. The infection starts from a small initial dose and nonlinear feedback then restricts the growth. Depending on the parameter regime the system may become a persistent periodic pattern with recurrent fluctuations in

infection prevalence. These numerical regimes provide a direct visualization of the threshold structure established analytically.

Epidemiological Interpretation

The numerical findings indicate the importance of temporal contact variability in epidemic control. Regular contact intensity can lead to large infection peaks, even if the average contact rate is unchanged. Therefore, interventions based on average transmission levels may not address periods of increased contact. The model also shows that isolation and recovery can alter the response of the epidemic to periodic forcing. Increasing the isolation rate reduces the time infectious individuals remain in the main transmission pathway, and faster recovery decreases the infectious duration. Both mechanisms can thus reduce the amplification associated with high-contact phases. The interaction with these processes is more important than the effects of each individual parameter in isolation. For example, a moderate isolation rate may be sufficient when periodic fluctuations are weak but less effective when contact variability is strong. Similarly, a system near the threshold may respond much differently to relatively small changes in either contact intensity or removal rate.

Overall Discussion

The numerical results support the analytical threshold and stability approach described in the previous sections. The fluctuating contact rate generates recurrent increases in infection prevalence and the threshold determines whether these fluctuations eventually disappear or remain as an epidemic. The simulations demonstrate that the amplitude and frequency of periodic forcing should be considered separately. The amplitude influences the strength of the temporal variation, whereas frequency determines its time scale. Their effects are mediated by the intrinsic time scales of exposure, infection, isolation, and recovery. Overall, the numerical study shows that including periodic contact patterns for epidemic dynamics in an SEIQR model is more accurate than a constant contact model. The model is able to explain more than whether an infection can invade but also how recurrent changes in population interaction affect the timing, magnitude and persistence of epidemic waves.

Conclusion

In this study, we developed and analyzed a nonlinear SEIQR epidemic model that includes periodically varying contact rates to explore the threshold behavior and stability of infectious disease dynamics in a temporally changing environment. Unlike autonomous epidemic models with constant transmission parameters, our proposed framework accounts for recurrent variations in population contact patterns while simultaneously incorporating exposed individuals, isolation, recovery, demographic turnover and disease-induced mortality.

The mathematical analysis has confirmed the positivity and boundedness of the solutions and the proposed model is still relevant for nonnegative initial conditions. We identified the disease-free state and the infection subsystem obtained through linearization around it is defined as a periodic differential system. So we have a periodic threshold to characterize the ability of the infection to invade the susceptible population. We observed that the threshold distinguishes between two completely different dynamical regimes: below the threshold the infection perturbations decay and the disease-free state is locally stable, and above the threshold the disease-free state loses stability and disease invasion becomes possible.

Floquet theory provides a suitable mathematical framework for understanding the stability of the periodically forced system. The major Floquet multiplier determines the long-term amplification or attenuation of small infection perturbations over successive contact cycles. This shows that the stability of an epidemic is not only on the average contact intensity of the system, but also on the time series of contact change. The numerical

study also showed the effects of periodic forcing on epidemic trajectories. Increasing the amplitude of contact variation can have stronger recurrent infection peaks, while changing the frequency of contact changes affects the nature of the disease progression and elimination time scales. Comparison between a constant and periodic contact rate revealed that even systems with the same mean contact intensity can have different epidemic patterns over time. The isolation and recovery mechanisms were also found to play a key role in reducing the duration and magnitude of infectious episodes and moving the system towards the disease-free state. One important result of this study is that the average transmission or contact rate alone may not be sufficient to characterize epidemic risk in populations with recurrent changes in social interaction. The timing, magnitude and frequency of contact events may influence the development of epidemic waves and long-term stability. Mathematical epidemic models that address the periodic contact phenomena can be a more informative tool for studying disease persistence and control in different environments. The present model can be extended in several directions. For example, vaccination, waning immunity, multiple infectious stages, demographic heterogeneity, time delays, stochastic contact processes or imperfect isolation may be considered. Another promising extension is the development of fractional-order versions of the periodically forced SEIQR model to study memory-dependent epidemic dynamics. Such extensions could provide insights into the interplay between temporal variability, intervention strategies and long-term disease persistence.

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