

Floquet Stability Analysis of Periodic Epidemic Models

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Abstract:

Seasonally forced epidemic models are nonautonomous dynamical systems, in which the contact rate and the transmission process change in time. For these models, the constant-coefficient eigenvalue analysis of autonomous theory no longer applies, and stability should be read off from the behaviour of the solutions in one forcing period. In this article we develop Floquet theory, the spectral theory of linear periodic differential equations, as the natural language for stability analysis of periodic epidemic models. We introduce the fundamental matrix, the monodromy matrix, and the Floquet multipliers, together with the classical criterion that every Floquet multiplier must remain inside the unit circle. Linearizing the infected subsystem in terms of the disease-free state leads to a periodic linear system whose monodromy matrix determines the invasion. We now describe the periodic next-generation operator and the associated periodic basic reproduction number R_0 , and we state the central correspondence: $R_0 < 1$, $= 1$, > 1 exactly when the dominant Floquet multiplier ρ satisfies $\rho < 1$, $= 1$, > 1 , respectively. This yields the threshold theorem: the disease-free periodic state is locally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$, in which case the disease is uniformly persistent. We also discuss the Floquet stability of positive periodic (endemic) solutions and the resonance phenomena that periodic forcing can produce. A seasonally forced SIR model is worked out in closed form to illustrate the theory. The results surveyed are classical; detailed proofs may be found in the references.

Keywords: Floquet theory; Floquet multipliers; monodromy matrix; periodic epidemic models; seasonal forcing; basic reproduction number; threshold dynamics; stability analysis.

1. Introduction

Many infectious diseases exhibit pronounced seasonality. Influenza surges in winter, cholera tracks the rains, and childhood infections follow the school calendar; Altizer et al. [9] survey the ecological and epidemiological evidence. The classical compartmental models of Kermack and McKendrick, and the modern threshold theory built upon them – notably the next-generation framework of van den Driessche and Watmough [5] – were developed for autonomous systems with constant parameters, and they are summarized in the authoritative review of Hethcote [4]. When the contact rate varies periodically in time, however, the model becomes a nonautonomous periodic differential system, and the familiar autonomous toolkit breaks down: equilibria are replaced by periodic solutions, and eigenvalues of a constant Jacobian are replaced by a subtler spectral object.

That object is the subject of Floquet theory. In 1883, Floquet [1] showed that a linear differential equation with periodic coefficients admits a distinguished set of solutions whose behaviour over one period is captured by finitely many numbers – the Floquet multipliers – and that these multipliers determine stability exactly as eigenvalues do for constant-coefficient systems. The theory is presented in textbook form by Hale [2] and Coppel [3]. For epidemic models with periodic coefficients, Floquet theory is therefore not a luxury but the natural language: linearizing about the disease-free state produces a periodic infection subsystem, and its Floquet multipliers decide whether a small introduction of infection grows or decays from one season to the next.

A parallel development extended the basic reproduction number to periodic environments. Bacaër and Guernaoui [8] treated seasonal vector-borne disease, Bacaër and Abdurahman [10] uncovered resonance of the epidemic threshold, and Wang and Zhao [11] gave the definitive threshold theory for compartmental models in periodic environments, defining the periodic reproduction number via a next-generation operator and proving that it stands in exact correspondence with the dominant Floquet multiplier of the linearized infection subsystem. Related contributions include the seasonal SEIR thresholds of Ma and Ma [7], periodic demographics in Wesley and Allen [12], the Malthusian-parameter perspective of Inaba [14, 15], age-structured periodic models of Kuniya and Inaba [16], and periodic cholera models of Posny and Wang [17].

This article is expository. Its aim is to assemble, in one place and with modest prerequisites, the Floquet-theoretic core of periodic epidemic modelling: the stability criterion for periodic linear systems, the periodic next-generation operator and its reproduction number, and the threshold theorems that link the two. Short proofs are included when they are illuminating; for the deeper persistence theory we give precise references, above all to Wang and Zhao [11] and Zhao [6].

There is a classical precedent for this point of view. Floquet's original motivation lay in the stability of periodic solutions of differential equations – the same circle of problems that produced Hill's equation for the lunar perigee and, later, the Mathieu equation, whose tongues of parametric instability are the textbook illustration of how periodic coefficients can destabilize what a frozen-coefficient analysis would deem safe. Epidemic models with seasonal forcing sit squarely in this tradition: freezing the transmission rate at its mean value and computing eigenvalues can declare a disease-free state stable while the true periodic system amplifies infection from season to season. The monodromy matrix is the object that refuses this simplification – it integrates the full seasonal profile into a single linear map, and its dominant multiplier reports, without approximation, the fate of a small infection after one complete cycle.

Our exposition follows the logical order of the theory rather than its history. We first develop the Floquet machinery for its own sake, since the stability criterion for periodic linear systems is used almost verbatim at every later stage. We then show how a periodic epidemic model, linearized about its disease-free state, produces exactly such a system, and how the periodic next-generation operator of Wang and Zhao extracts from it a reproduction number with the same threshold meaning as in the autonomous case. The correspondence between this number and the dominant Floquet multiplier is the hinge of the paper: it turns the abstract stability criterion into an epidemiological threshold theorem. The

remaining sections apply the same criterion to endemic periodic solutions, where it plays the role the Jacobian eigenvalue test plays for endemic equilibria, and work out the one case – the seasonally forced SIR model – in which the monodromy multiplier can be written down in closed form.

The paper is organized as follows. Section 2 is a self-contained primer on Floquet theory: the fundamental matrix, the monodromy matrix, multipliers and exponents, and the stability theorem. Section 3 introduces periodic compartmental epidemic models and linearizes the infected subsystem about the disease-free state. Section 4 defines the periodic basic reproduction number through the next-generation operator and states the threshold–multiplier correspondence. Section 5 develops the Floquet stability analysis of the disease-free state and of positive periodic solutions. Section 6 works out a seasonally forced SIR model in closed form, and Section 7 offers concluding remarks.

2. Floquet theory: a primer

We consider the linear homogeneous system

$$\dot{x}_t = A(t)x, x \in \mathbb{R}^n \quad (1)$$

where $A : \mathbb{R} \rightarrow \mathbb{R}^{n \times n}$ is continuous and T -periodic for some $T > 0$, i.e. $A(t + T) = A(t)$ for all t . Equation (1) is the linearization that will appear throughout: whenever a nonlinear periodic epidemic model is linearized about one of its periodic solutions, the result has exactly this form. The question Floquet theory answers is: how does the periodicity of A constrain the solutions, and what finite spectral data determine their long-time behaviour?

2.1. The fundamental matrix and the monodromy matrix

Let $\Phi(t)$ be the principal fundamental matrix of (1), defined by

$$\dot{\Phi}_t = A(t)\Phi(t), \Phi(0) = I_n \quad (2)$$

so that the solution with initial value x_0 is $x(t) = \Phi(t)x_0$. By Liouville's formula $\Phi(t)$ is invertible for every t . Periodicity of A implies a crucial algebraic relation. Indeed, both $\Phi(t + T)$ and $\Phi(t)\Phi(T)$ satisfy the same matrix differential equation $X' = A(t)X$; by uniqueness of solutions,

$$\Phi(t + T) = \Phi(t)M, M := \Phi(T) \quad (3)$$

The constant matrix $M = \Phi(T)$ is called the monodromy matrix of (1): it advances solutions by one full period, $x(t + T) = \Phi(t)Mx_0$. Iterating,

$$x(kT) = M^k x_0, k = 0, 1, 2, \dots \quad (4)$$

so the long-time behaviour of solutions is governed by the powers of the single matrix M . Its eigenvalues $\mu_1 \dots \mu_n$, counted with algebraic multiplicity, are the Floquet multipliers of (1). The dominant multiplier, and the single most important number in what follows, is the spectral radius

$$\rho(M) = \max_{1 \leq i \leq n} |\mu_i| \quad (5)$$

it is the per-period growth factor of the system: a generic solution grows like $\rho(M)^k$ after k periods.

2.2. Multipliers, exponents, and the stability theorem

Floquet's fundamental representation theorem [1, 2] refines (3). There exist a T -periodic matrix function $P : \mathbb{R} \rightarrow \mathbb{R}^{n \times n}$ and a constant matrix $B \in \mathbb{R}^{n \times n}$ such that

$$\Phi(t) = P(t)e^{tB} \quad (6)$$

with $P(t + T) = P(t)$. The eigenvalues λ_i of B are the characteristic (or Floquet) exponents, related to the multipliers by $\mu_i = e^{\lambda_i T}$. Equation (6) is the periodic analogue of the diagonalization of a constant-coefficient system: it separates the bounded periodic modulation $P(t)$ from the exponential trend e^{tB} . In particular, if μ is a multiplier with eigenvector v (so $Mv = \mu v$), then $\psi(t) = P(t)e^{\lambda t}v$ with $\mu = e^{\lambda T}$ satisfies $\psi(t + T) = \mu\psi(t)$: a Floquet solution, reproducing itself up to the scalar factor μ after each period.

The stability theorem is now an immediate consequence of (4) and (6).

Theorem 1 (Floquet stability criterion). *The zero solution of (1) is (i) asymptotically stable if and only if $|\mu_i| < 1$ for every Floquet multiplier, i.e. $\rho(M) < 1$; (ii) unstable if $|\mu_i| > 1$ for some multiplier, i.e. $\rho(M) > 1$; (iii) Lyapunov stable, but not asymptotically, if $|\mu_i| \leq 1$ for all i and every multiplier on the unit circle is semisimple (its Jordan blocks are trivial).*

The proof is the matrix analogue of the constant-coefficient case: M^k decays exponentially iff $\rho(M) < 1$, by the spectral radius formula, and (4) transfers this to $x(t)$. The borderline case $\rho(M) = 1$ is the periodic analogue of a zero eigenvalue: the system is at threshold, and the nonlinear terms decide the outcome. This is exactly the situation in which the epidemic threshold will place us.

3. Periodic epidemic models

The mathematical modeling of infectious diseases has traditionally been based on autonomous compartmental systems in which epidemiological parameters are assumed to be constant over time. However, many infectious diseases are subject to high temporal variation in transmission due to seasonal changes in climate, human behaviour, school calendars, population mixing, and many other variables in the environment or demographic change. Autonomous models may not be able to describe the dynamics of an epidemic as transmission is often random. This led to the development of seasonally forced epidemic models in which one or more epidemiological parameters are explicitly represented as periodic functions of time. One of the classic developments in this direction was the incorporation of seasonal variation in susceptible-infected-recovered (SIR) models. Hethcote and Tudor showed that demographic and epidemiological processes could be incorporated into nonlinear epidemic systems and laid the groundwork for further periodic models [1]. In later studies seasonal forcing can generate recurrent epidemics and significantly affect the stability properties of endemic and disease-free states [2,3].

The role of seasonal variation was particularly emphasized in studies of childhood diseases. Schenzle [4] proposed a stochastic model of the dynamics of measles and demonstrated that seasonal variation in transmission and demographic factors could produce complex epidemic patterns. Earn et al. [5] studied the mechanism of seasonality and synchrony of measles epidemics and found that periodic variation in transmission can lead to rich temporal behaviour even in relatively simple epidemic systems.

Seasonally forced epidemic models have also been studied from the perspective of nonlinear dynamics and bifurcation theory. Olsen and Schaffer [6] showed that periodic variation of transmission can lead to complex dynamical phenomena, such as subharmonic responses and chaotic behaviour. These results showed that periodic forcing is not just a minor perturbation of an autonomous epidemic model but can qualitatively change the long-term dynamics of the system. A particularly important development was

the use of periodically varying transmission rates to represent seasonal contact patterns. Dushoff et al. [7] investigated the relation between seasonality and the dynamics of infectious diseases and showed that the interaction between demographic turnover, transmission seasonality and intrinsic epidemic time scales can determine the persistence and recurrence of infection. Such models can explain the recurring annual or multiannual epidemic patterns of several infectious diseases.

Periodic epidemic systems have been studied in the theory of nonautonomous dynamical systems. However, as with autonomous models, a periodically forced epidemic system in most cases does not have an equilibrium representing a time-independent endemic state. Instead, the relevant invariant object may be a periodic solution whose stability is determined by the corresponding linearized periodic system. This naturally leads to Floquet theory and to the use of Floquet multipliers to study the stability of disease-free periodic solutions. The next-generation approach has also been extended to periodically changing epidemic systems. In autonomous models, the basic reproduction number is usually related to the spectral radius of a next-generation matrix. For periodic epidemic systems, however, transmission and removal processes will vary with time and invasion will depend on the cumulative effect of these processes over one forcing period. This has motivated the development of reproduction numbers for periodic and more general nonautonomous epidemic models [8-10]. Wang and Zhao [10] in particular developed a next-infection operator framework for periodic epidemic systems and related the reproduction number to the spectral properties of the corresponding evolution operator. These developments provide the mathematical basis for studying epidemic models with periodic changes in contact or transmission rates. In such systems the disease-free periodic state replaces the disease-free equilibrium of an autonomous model and the dominant Floquet multiplier is a natural criterion to see if small infections decay or grow from one forcing period to the next. When the dominant multiplier lies inside the unit circle infection perturbations decay, and when it exceeds unity in modulus, infection can invade the disease-free periodic state. The threshold case when the dominant multiplier lies on the unit circle is even harder to understand.

So periodic epidemic models provide a general framework for studying diseases whose transmission is strongly influenced by seasonal or otherwise recurrent environmental and behavioural influences. The general formulation used in the present work is presented below.

3.1. A general compartmental framework

We consider a compartmental epidemic model with T -periodic coefficients, written in the general nonautonomous form

$$\dot{x}_t = f(t, x) \quad (7)$$

where $f(t + T, x) = f(t, x)$ for all t, x . The state $x \in \mathbb{R}^n$ is partitioned into m infected compartments $x_I = (x_1, \dots, x_m)^T$ and $n - m$ uninfected compartments x_U (susceptible, recovered, and so on). Periodicity enters through time-dependent parameters: the transmission coefficient $\beta(t)$, the recovery rate $\gamma(t)$, demographic rates, and the like. A standard seasonal forcing, used throughout the literature [7, 8, 11], is

$$\beta(t) = \beta \left(1 + \varepsilon \sin \left(\frac{2\pi t}{T} + \phi \right) \right) \quad (8)$$

with mean $\beta > 0$, relative amplitude $0 \leq \varepsilon < 1$, and phase ϕ . The amplitude ε controls the contrast between high- and low-transmission seasons, while the period T (typically one year) sets the forcing time scale against the intrinsic epidemic time scales.

3.2. The disease-free periodic state and linearization

Assume that (7) admits a disease-free periodic solution

$$E_0(t) = (0, x_U^0(t)) \quad (9)$$

with $x_U^0 T$ -periodic; in the usual demographic setting the uninfected compartments settle to a constant or periodic demographic steady state while infection is absent. The invasion question is whether a small introduction of infection near E_0 grows or decays. Linearizing the infected compartments about E_0 gives, following the periodic extension of the van den Driessche–Watmough splitting [5] due to Wang and Zhao [11],

$$x_t = (F(t) - V(t))x \quad (10)$$

where $x \in \mathbb{R}^m$ now denotes the infected compartments, $F(t)$ is the T -periodic matrix of rates of new infections, and $V(t)$ is the T -periodic matrix of transfer rates between and out of the infected compartments (recovery, progression, mortality). The matrix $F(t)$ is nonnegative, and $-V(t)$ is cooperative with spectral abscissa nonpositive, uniformly in t [11]. Equation (10) is precisely of the form (1) with $A(t) = F(t) - V(t)$: its monodromy matrix $\Phi_{F-V}(T)$, and in particular its dominant Floquet multiplier $\rho(\Phi_{F-V}(T))$, govern the linear invasion dynamics.

4. The periodic reproduction number and the dominant multiplier

For autonomous models the basic reproduction number R_0 is the spectral radius of the next-generation matrix FV^{-1} [5]. In a periodic environment there is no constant matrix to invert; instead, Wang and Zhao [11] define R_0 as the spectral radius of a next-generation operator acting on T -periodic functions.

4.1. The next-generation operator in periodic environments

Let $Y(t, s)$, $t \geq s$, be the evolution operator of the linear system $y' = -V(t)y$, describing how an initial infection cohort survives and moves between infected compartments in the absence of new infections. For a given T -periodic distribution φ of infectives, the total infective output produced over all future generations is

$$(L\varphi)(t) = \int_0^\infty Y(t, t-a)F(t-a)\varphi(t-a) da \quad (11)$$

The operator L is positive, compact, and maps the cone of nonnegative T -periodic functions into itself; by the Krein–Rutman theorem its spectral radius is a simple eigenvalue with a strictly positive eigenfunction. The periodic basic reproduction number is defined [11] by

$$R_0 = \rho(L) \quad (12)$$

the asymptotic per-generation growth factor of the infection in the periodic environment. Inaba [14] later gave this definition a unified generation-evolution interpretation, showing that it extends the classical autonomous definition to arbitrary heterogeneous environments, while Inaba [15] connected it with the Malthusian parameter of the periodic system.

4.2. The threshold–multiplier correspondence

The bridge between the reproduction number and Floquet theory is the following fundamental result.

Theorem 2 (Wang and Zhao [11]). *Let R_0 be defined by (12) and let $\Phi_{F-V}(T)$ be the monodromy matrix of (10). Then*

$$R_0 < 1 \Leftrightarrow \rho(\Phi_{F-V}(T)) < 1; R_0 = 1 \Leftrightarrow \rho(\Phi_{F-V}(T)) = 1; R_0 > 1 \Leftrightarrow \rho(\Phi_{F-V}(T)) > 1 \quad (13)$$

that means the disease can invade the disease-free state ($R_0 > 1$) if and only if the dominant Floquet multiplier of the linearized infection subsystem exceeds unity, i.e. if and only if a small infection is amplified from one forcing period to the next. The threshold value $R_0 = 1$ corresponds exactly to the borderline Floquet case $\rho = 1$ of Theorem 1. This correspondence is the periodic analogue of the familiar autonomous statement that $R_0 - 1$ and the spectral abscissa of $F - V$ have the same sign [5]. The correspondence (13) is sharp, but it carries a warning that Ma and Ma [7] made explicit for seasonally forced SEIR models: the reproduction number of the time-averaged autonomous system, R_0 , need not decide the outcome. One may have $R_0 < 1$ while the true periodic R_0 exceeds 1, and vice versa. Seasonality is not a perturbation of the mean: the whole periodic profile of $F(t)$ and $V(t)$ enters the monodromy matrix, and with it the threshold.

5. Floquet stability of disease-free and endemic periodic states

Periodic epidemic models have been studied more and more using Floquet theory in order to characterize the stability of the disease-free and endemic periodic systems. In periodically forced systems, the disease-free equilibrium of an autonomous model is replaced by a disease-free periodic orbit and its stability is determined by the Floquet multipliers of the corresponding linearized periodic system [1,2]. Bacaër and Ait Dads [3] discovered the connection between the principal eigenvalue of periodic epidemic models and their invasion properties. Wang and Zhao [4] introduced a spectral approach for defining reproduction thresholds for time-periodic epidemic systems. They showed that the primary Floquet multiplier is a natural criterion for deciding whether the small infection perturbation decays or grows over successive forcing periods. This framework is the basis for our disease-free stability analysis in the following subsection.

5.1. Stability of the disease-free state

Linearizing the full system (7) about the disease-free periodic solution $E_0(t)$ yields a block-triangular periodic system: the infected block is exactly (10), while the uninfected block describes demographic relaxation toward x_U^0 and is asymptotically stable by assumption. The Floquet multipliers of the full linearization are therefore the union of the multipliers of the two blocks.

Theorem 3 (Threshold stability of the disease-free state). *Assume the uninfected subsystem is asymptotically stable at x_U^0 . If $R_0 < 1$, then the disease-free periodic solution $E_0(t)$ of (7) is locally asymptotically stable; if $R_0 > 1$, it is unstable.*

The proof is a direct application of Theorem 1: by Theorem 1, $R_0 < 1$ is equivalent to every Floquet multiplier of (10) lying inside the unit circle, and together with the stable uninfected block this places all multipliers of the full linearization inside the unit disc. Linearized asymptotic stability then implies nonlinear local asymptotic stability by the standard periodic version of Lyapunov's first method [2, 3]. In

epidemiological terms, each successive transmission season multiplies a small infection by a factor below one, so the infection decays geometrically from period to period.

5.2. Instability and uniform persistence

When $R_0 > 1$, the dominant multiplier satisfies $\rho(\Phi_{F-V}(T)) > 1$, and the disease-free state loses stability through the Floquet mechanism: the associated Floquet solution grows by the factor ρ every period. For the nonlinear system this instability is not the end of the story but the beginning of the endemic theory. Wang and Zhao [11] prove that $R_0 > 1$ implies uniform persistence of the disease – the infected compartments stay bounded away from zero – and the existence of at least one positive T -periodic solution, the periodic endemic state. The monograph of Zhao [6] develops the abstract persistence machinery behind this conclusion. Global threshold results in this spirit were obtained for age-structured periodic SIS models by Kuniya and Inaba [16] and for periodic cholera models by Posny and Wang [17].

5.3. Stability of positive periodic solutions

Let $x^*(t)$ be a positive T -periodic solution of (7), for instance the endemic periodic state whose existence follows from $R_0 > 1$. Linearizing (7) about $x^*(t)$ gives the periodic variational system

$$y_t = D_x f(t, x^*(t))y \quad (14)$$

again of the form (1). Its monodromy matrix always has the multiplier $\mu = 1$, corresponding to perturbations along the periodic orbit itself – the periodic analogue of the zero eigenvalue produced by translation invariance for traveling waves. The periodic solution is locally asymptotically stable (orbitally, with asymptotic phase) if every remaining Floquet multiplier lies strictly inside the unit circle, and unstable if any multiplier lies outside it. This is the exact periodic counterpart of the eigenvalue criterion for equilibria, and it is how the stability of seasonal endemic cycles is assessed in practice: compute the fundamental matrix of (14) over one period and inspect the spectrum of the monodromy matrix.

Resonance and the limits of averaging: Periodic forcing can do more than modulate a fixed threshold. Bacaër and Abdurahman [10] showed that the epidemic threshold itself can resonate: when the forcing period is commensurate with the intrinsic infectious period, the dominant multiplier – and hence R_0 – responds non-monotonically to the forcing amplitude. Katriel and Stone [13] found related resonance structure in the attack rates of seasonal epidemics. The lesson, already noted in previous section, is that the mean parameters do not tell the whole story: the timing of transmission within the period, encoded in the monodromy matrix, can move multipliers across the unit circle even when all averaged quantities are held fixed.

6. Worked example: the seasonally forced SIR model

To fix ideas we work out the simplest nontrivial case in closed form. Consider the SIR model with periodic transmission and demographic turnover,

$$S_t = \mu N - \frac{\beta(t)SI}{N} - \mu S, I_t = \frac{\beta(t)SI}{N} - (\gamma + \mu)I, R_t = \gamma I - \mu R \quad (15)$$

with $\beta(t + T) = \beta(t)$ as in (8), constant total population N , recovery rate γ , and natural mortality μ . The disease-free state is the constant

$$E_0 = (N, 0, 0) \quad (16)$$

The infected subsystem is one-dimensional, and linearization about E_0 gives the scalar periodic equation

$$I_t = (\beta(t) - (\gamma + \mu))I \quad (17)$$

For a scalar equation the monodromy “matrix” is just a number, explicitly computable as in previous section

$$\rho = \exp(\int \beta(t) - (\gamma + \mu) dt) = e^{(\beta - (\gamma + \mu))T} \quad (18)$$

where $\beta = \frac{1}{T} \int \beta(t) dt$ is the mean transmission rate. The single Floquet multiplier ρ lies inside the unit circle if and only if

$$\beta < \gamma + \mu \Leftrightarrow R_0 := \frac{\beta}{\gamma + \mu} < 1 \quad (19)$$

and Theorem 3 gives local asymptotic stability of E_0 for $R_0 < 1$ and instability for $R_0 > 1$. In this one-dimensional case the periodic reproduction number coincides with the reproduction number R_0 of the time-averaged autonomous system – a coincidence that is deceptive. Already for the SEIR model, whose infected subsystem is two-dimensional, no such closed form exists: the monodromy matrix must be computed numerically from (2), and the dominant multiplier depends on the amplitude ε , the phase ϕ , and the relation between the forcing period and the latent and infectious periods, in general differing from the averaged threshold [7, 10]. The scalar computation above is therefore best read as the base case from which the genuinely periodic phenomena – resonance, phase sensitivity, and the failure of averaging – depart as the dimension of the infected subsystem grows.

7. Conclusion

Floquet theory replaces eigenvalues by Floquet multipliers wherever periodicity replaces autonomy. For a periodic epidemic model the monodromy matrix of the linearized infection subsystem plays exactly the role the Jacobian plays at an endemic equilibrium: its dominant multiplier ρ is the per-period growth factor of a small infection, and the periodic basic reproduction number R_0 , defined as the spectral radius of the next-generation operator (12), satisfies the exact correspondence $R_0 < 1 \Leftrightarrow \rho < 1$ of Theorem 1 [11]. The threshold theorem follows at once: the disease-free periodic state is locally asymptotically stable for $R_0 < 1$ and unstable, with the disease uniformly persistent, for $R_0 > 1$. The same Floquet machinery governs the stability of positive periodic (endemic) solutions via the variational system (14). Two lessons deserve emphasis. First, the threshold is a genuinely periodic object: it depends on the full time profile of transmission and removal, not merely on their averages, and seasonal forcing can produce resonance phenomena invisible to averaged models [7, 10, 13]. Second, the theory is constructive: the monodromy matrix is obtained by integrating the fundamental matrix over a single period, so the dominant multiplier – and with it the stability verdict – is numerically accessible for any concrete model. Extensions of this framework to age-structured [16], vector-borne [8], and environmentally driven [17] periodic models, and to the Malthusian-parameter viewpoint [15], continue to be an active area of research.

On the practical side, the theory is constructive rather than merely qualitative. Because the monodromy matrix is obtained by integrating the fundamental matrix over a single forcing period, the dominant multiplier – and with it the stability verdict – is numerically accessible for any concrete model, however complicated its compartmental structure. This is how the threshold is actually evaluated in applications: one integrates the linearized infection subsystem over one year, computes the eigenvalues of the

resulting matrix, and reads off the per-season growth factor. Analytical insight and numerical computation thus meet at the same object, and the Floquet multiplier serves as a common language between the two.

Several natural extensions lie beyond the scope of this exposition. Pulse vaccination strategies superimpose impulsive periodic forcing on the seasonal background; time delays in latency or immunity turn the monodromy operator into an infinite-dimensional object while preserving the Floquet philosophy; and stochastic or almost-periodic forcing asks how much of the periodic picture survives when strict periodicity is relaxed. The interplay between forcing amplitude, forcing frequency, and the intrinsic epidemic time scales – visible already in numerical studies of seasonally forced models – remains a rich source of concrete problems, from the resonance of the epidemic threshold to the design of interventions timed to the seasonal cycle.

In the end, Floquet theory earns its place in mathematical epidemiology for a simple reason: it is the exact linear stability theory of the periodic world, just as eigenvalue analysis is the exact linear stability theory of the autonomous world. Wherever transmission varies periodically – with the seasons, the school terms, or the agricultural calendar – the questions of invasion, extinction, and persistence are questions about Floquet multipliers, and the basic reproduction number of the periodic environment is their epidemiological face.

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