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A comparison of traveling wave properties in minimal and biophysically detailed models of cardiac tissue using a novel software tool

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Vincent Lovero  ; Stephanie Dodson  ; Timothy J. Lewis  



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ABSTRACT

Models of cardiac tissue span a wide range of complexity, from minimal models to biophysically detailed models. It is often assumed that minimal models accurately capture the core behaviors of propagating action potentials in biophysically detailed models. However, it remains unclear whether additional physiological detail introduces qualitatively new behaviors. Dispersion relations quantify the existence, stability, and bifurcations of traveling waves, but their computation for biophysically detailed models has been limited by system complexity and computational cost. To overcome these challenges, we develop Traveling Wave Investigation Software Tool (TWIST), a numerical framework for the systematic computation of traveling wave solutions and their spectra. Using TWIST, we compute and compare dispersion relations in the FitzHugh–Nagumo, Beeler–Reuter, and nine-variable ten Tusscher–Panfilov models. Our analysis shows that increasing physiological detail can introduce substantial additional structure in dispersion relations. In particular, the interaction of multiple activation processes in the ten Tusscher–Panfilov model gives rise to additional solution branches, including families of slow waves that are absent in minimal models and may play important roles in abnormal wave dynamics. Our comparisons across models of increasing complexity distinguish robust features of wave propagation from those arising specifically from detailed ionic interactions and represent a step toward validating that the observed behaviors reflect genuine cardiac phenomena rather than artifacts of specific model formulations.

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Regular cardiac function depends on the reliable propagation of electrical waves, or action potentials, through the heart. Disruptions to these waves can initiate or sustain life-threatening arrhythmias, making it essential to understand how wave propagation depends on physiological conditions. A powerful framework for this is provided by dispersion relations, which describe how wave speed, existence, and stability vary with model parameters. However, cardiac models span a wide range of complexity from minimal models that capture general features of excitability to detailed models that incorporate the dynamics of specific ionic currents, and analyzing wave behavior across this spectrum of models remains challenging. In particular, the nonlinear dynamics and multiple interacting timescales in detailed models have made the systematic computation of dispersion relations largely impractical. Here, we introduce TWIST, a novel software

framework designed to compute dispersion relations in cardiac models. Using TWIST, we compare wave dynamics across models of increasing complexity and identify both robust features of propagation and qualitatively new behaviors that emerge from additional physiological detail.

I. INTRODUCTION

The rhythmic contraction of the heart is driven by the propagation of electrochemical waves of excitation, or action potentials (APs), through cardiac tissue. These propagated waves coordinate the mechanical contraction to ensure effective pumping of blood. Disturbances to AP propagation, such as reduced conduction velocity, conduction block, or the formation of reentrant circuits, can

disrupt this coordination and precipitate life-threatening arrhythmias such as ventricular tachycardia and ventricular fibrillation.¹ Understanding the mechanisms that govern the initiation, propagation velocity, and stability of these waves is, therefore, central to cardiac electrophysiology and to the prevention of arrhythmias.

Mathematical models play a critical role in elucidating the mechanisms underlying wave propagation. A broad spectrum of cardiac models exists, ranging from low-dimensional minimal phenomenological models (e.g., FitzHugh–Nagumo,² Morris–Lecar³) that capture qualitative features of excitation, to high-dimensional biophysically detailed ionic models (for example, Beeler–Reuter,⁴ Luo–Rudy,⁵ ten Tusscher,⁶ Grandi–Pasqualini–Bers⁷) that explicitly represent individual ionic currents, gating variables, and intracellular processes. Minimal models offer analytical and computational tractability, enabling the identification of general principles governing wave propagation, stability, and bifurcation structure. However, their simplified descriptions of ionic processes limit their ability to capture quantitatively accurate or physiologically specific behaviors. In contrast, biophysically detailed models provide a more faithful representation of cardiac electrophysiology and can reproduce experimentally observed dynamics, but their high dimensionality and complex coupling between ionic processes often obscure the underlying dynamical mechanisms and complicate systematic analysis. Resolving these trade-offs and leveraging the complementary strengths of different models requires comparing these model classes to identify which features of wave dynamics are robust across levels of complexity and which arise specifically from detailed ionic interactions.

Wave propagation in both minimal and biophysically detailed models can exhibit rich bifurcation structures and complex spatiotemporal dynamics as parameters vary, often in ways that are difficult to predict *a priori*. Characterizing these dependencies is essential for model comparison and for understanding the onset of conduction block, the formation of reentrant circuits, and related arrhythmogenic phenomena. A common approach to quantifying parameter dependencies is to idealize the propagating action potential as a traveling wave and compute dispersion relations, which describe how properties of the traveling wave such as conduction velocity and action potential duration depend on parameters including spatial period and maximal conductances.

Analytical solutions for traveling waves are rarely available, making numerical methods essential for computing solutions and assessing their stability. However, most existing simulation frameworks are not designed for the systematic computation of traveling wave solutions and their spectra. As a result, researchers often rely on *ad hoc* workflows that combine multiple solvers, custom scripts, and extensive parameter sweeps. These approaches become particularly burdensome in high-dimensional, stiff biophysically detailed models, where direct simulation is computationally expensive and may fail to detect subtle changes in wave structure. Moreover, simulation-based methods are generally unable to capture unstable traveling waves, which play critical roles in organizing wave behavior. These limitations hinder comprehensive studies of dispersion, stability, and bifurcation structures in biophysically detailed cardiac models.

In this work, we overcome the challenges of computing dispersion curves and spectral stability in biophysically detailed cardiac models by developing TWIST, a numerical framework for the systematic computation of traveling wave solutions, their spectra, and associated bifurcation structures in reaction–diffusion systems. Using this approach, we compute dispersion curves and spectra in both minimal and biophysically detailed models, enabling a direct comparison across levels of model complexity and revealing both robust organizing principles and qualitative differences in wave dynamics. We apply TWIST to a minimal excitable system (FitzHugh–Nagumo) and to biophysically detailed cardiac models (Beeler–Reuter and the nine-variable ten Tusscher–Panfilov model⁶), uncovering phenomena including cascades of Hopf bifurcations, modulated traveling waves, and the emergence of multiple slow-wave branches.

The remainder of the paper is organized as follows. Sections II and III summarize the cardiac models considered and review traveling wave theory and dispersion relations. Section IV describes the numerical methods underlying TWIST and benchmarks its performance against AUTO. Section V presents the computed dispersion curves and spectra across models. Section VI investigates threshold dynamics for wave propagation and the conditions under which wave reflection can occur. Finally, Sec. VII summarizes key results and discusses their implications for wave dynamics in cardiac tissue, and outlines directions for future work.

II. MINIMAL AND BIOPHYSICALLY DETAILED MODELS OF CARDIAC TISSUE

Cardiac tissue can be viewed as a spatially extended excitable medium composed of electrically coupled myocytes that form a functional syncytium. At the cellular level, the excitation and recovery processes that underlie cardiac APs arise from the complex nonlinear interactions of ion channels, pumps, exchangers, intracellular calcium dynamics, and the concentrations of key ion species. Electrical coupling through gap junctions allows current to electrotonically spread between cells, enabling APs to propagate through the myocardium. The cellular processes, together with intercellular coupling, operate over a wide range of time scales and collectively determine key macroscopic electrophysiological properties, including the rate-dependence of action potential duration (APD) and conduction velocity (CV). The resulting APD and CV restitution properties play central roles in governing wave stability and the susceptibility of cardiac tissue to arrhythmogenic behaviors such as conduction block, reentry, and fibrillation.

Computational models of cardiac action potentials span a wide range of complexity.^{8,9} At one end, minimal models intentionally omit most biophysical detail and retain only the core dynamical processes of excitability and recovery. These models are particularly valuable for dynamical-systems analysis, conceptual understanding, and large-scale tissue simulations. At the other end, highly detailed conductance-based models explicitly describe ion-channel gating, pumps and exchangers, intracellular calcium handling, and spatial sub-compartments. The physiological detail of these models enables mechanistic investigations of the initiation and maintenance of arrhythmias, as well as effects of pharmacological agents.

Between minimal models and highly detailed models, there is a hierarchy of models with intermediate complexity that trade off physiological detail for computational efficiency and analytical tractability. The appropriate level of model complexity depends on the scientific question being addressed. However, it is essential to establish that dynamical behaviors and mechanisms identified in minimal models persist in more detailed descriptions, and to assess whether additional physiological detail introduces qualitatively new behavior. Cross-model validation is, therefore, critical for ensuring that insights obtained from minimal models reflect genuine physiological mechanisms rather than artifacts of oversimplification and for identifying and characterizing phenomena that arise from biophysical processes present only in more detailed models.

A. Minimal models

The simplest minimal models of cardiac electrophysiology reduce dynamics to two state variables: a single fast excitation variable v and a single slow recovery variable w . One of the most widely used canonical models of excitable media is the FitzHugh–Nagumo (FHN) model,² which captures threshold behavior, excitability, and refractoriness in a highly simplified form. In a spatially extended setting, the FHN model can be written as

$$\begin{aligned}\frac{\partial v}{\partial t} &= -v(v - \alpha)(v - 1) - w + D\Delta v, \\ \frac{\partial w}{\partial t} &= \varepsilon(v - \gamma w),\end{aligned}\quad (1)$$

where D is the scalar diffusion coefficient representing intercellular coupling, α sets the threshold for excitation, γ modulates the recovery process, and $0 < \varepsilon \ll 1$ sets the separation of time scales. Despite its simplicity and lack of direct biophysical interpretation, the FHN model has played a key role in developing a deep qualitative understanding of wave propagation, as well as spiral wave dynamics and defibrillation, in cardiac tissue.

To better understand specific cardiac properties, several alternative low-dimensional models have been developed. Two-variable models such as the Aliev–Panfilov model¹⁰ and Karma model^{11,12} modify the recovery dynamics to better capture restitution and excitability control, while still remaining computationally efficient and theoretically tractable. Other two-variable models, such as the Morris–Lecar model,³ are formulated in terms of ionic currents and gating variables and therefore can be used to hint at biophysical mechanisms underlying phenomena observed in cardiac tissue. Three-variable models, such as the Fenton–Karma model,¹³ introduce additional recovery or gating variables to reproduce key electrophysiological features and restitution properties of cardiac tissue, improving predictions of alternans and spiral-wave breakup while maintaining a relatively small state space.

B. Biophysically detailed conductance-based models

In contrast to minimal models, biophysically detailed conductance-based models involve dozens of variables and hundreds of parameters, and they provide mechanistic descriptions of the myriad of interacting molecular- and cellular-scale processes governing excitation, recovery, modulation, and long-term ionic homeostasis.

The O’Hara–Rudy model¹⁴ for human ventricular tissue, for example, has 41 state variables and explicitly represents a wide range of ionic currents, transporters, calcium-handling, and homeostatic processes. However, even the nine-variable ten Tusscher–Panfilov (TP06) model¹⁵ and the eight-variable Beeler–Reuter (BR) model⁴ for human ventricular tissue are substantially complex. Both the TP06 and the BR models incorporate multiple excitation processes, primarily mediated by the fast sodium current and the L-type calcium current, and a diverse set of recovery and refractory mechanisms including activation of several potassium channels and inactivation of sodium and calcium channels. The time scales associated with these processes span several orders of magnitude. We will show later that the interaction between the distinct excitation processes gives rise to important differences in the traveling-wave structure and stability.

As for all conductance-based models, the current-balance equation in the TP06 and BR models serves as the structural core and explicitly governs the evolution of the membrane potential v ,

$$C_m \frac{\partial v}{\partial t} = - \sum_j I_j(v, \mathbf{y}) + D\Delta v, \quad (2)$$

where C_m is the membrane capacitance, $I_j(v, \mathbf{y})$ denotes the j th ionic membrane current density, and $\mathbf{y} = (y_1, \dots, y_k)$ is the vector of gating variables. The diffusion term represents electrical coupling between neighboring cells via gap junctions. As in the FHN model above, spatial coupling enters exclusively through the effective diffusion of v that represents electrotonic coupling between neighboring cells. The diffusion coefficient is $D = (S_V \rho C_m)^{-1}$, where ρ is the internal resistivity and S_V is the surface-to-volume ratio of the cells. (Note: We are considering only homogeneous and isotropic monodomain models.)

Each ionic current I_j depends on the membrane voltage and on a subset of the gating variables and, in some cases, on ionic concentrations. In both the TP06 and BR models, the gating variables y_i , which govern the activation or inactivation of the corresponding ion channels, obey first-order kinetic equations of the form,

$$\frac{\partial y_i}{\partial t} = \frac{1}{\tau_i(v)} (y_{i\infty}(v) - y_i). \quad (3)$$

More generally, gating in conductance-based models can be governed by more complicated “Markov state” transition models and can involve ion-concentration-dependent transition rates.^{16,17} The BR model also includes intracellular calcium as a dynamic variable, but it lacks detailed intracellular calcium handling.

It is important to note that the specific formulations of biophysical processes in detailed conductance-based models can vary substantially, even when the models are intended to represent the same tissue type in the same species. As a consequence, the resulting dynamical properties, such as APD and CV restitution, bifurcation structure, and wave stability, can differ quantitatively and, in some cases, qualitatively across models. The results obtained in one model, therefore, do not necessarily generalize to others.^{8,18} The additional excitation and recovery pathways present in biophysically detailed models raise the possibility of qualitatively new traveling-wave phenomena that cannot occur in minimal two-variable excitable systems. One of the central goals of this work is

to determine which features of traveling-wave dynamics are robust across model hierarchies and which emerge only with increased physiological detail.

To begin a comparison of propagated wave dynamics across the model hierarchy, we use the two-variable FHN model as a canonical minimal model, and the eight-variable BR model and nine-variable ten Tusscher–Panfilov (TP06) model as representative detailed conductance-based descriptions. Although the BR model is no longer commonly used as a primary electrophysiological model, it remains a foundational first-generation ventricular cell model and continues to appear in methodological and comparative studies. The TP06 model is a reduced version of the 19-variable ten Tusscher–Noble–Noble–Panfilov (TNNP) model^{6,15} and preserves key features of human ventricular action potential morphology and restitution. While it is less detailed than the full TNNP formulation and therefore less frequently employed in clinically oriented simulations, its balance between physiological realism and computational efficiency makes it well suited for theoretical studies and large-scale tissue simulations.

III. TRAVELING WAVES IN EXCITABLE MEDIA

The results presented later in this paper rely on several concepts, including unstable traveling waves, spectral stability, and dispersion relations, that are central to the analysis of traveling waves but are not routinely encountered in studies based on direct numerical simulation. Because these concepts play an essential role in interpreting the results, we briefly review them here. For more comprehensive reviews, see Refs. 19–22.

In cardiac tissue, action potentials spread through a complex three-dimensional structure with heterogeneous properties, fiber anisotropy, and curvature effects. Nevertheless, many fundamental aspects of action potential propagation can be understood by considering idealized settings in which waves propagate steadily as planar waves through homogeneous media.

Traveling wave solutions provide a powerful mathematical idealization of action potential propagation in cardiac tissue. In this framework, a planar wave maintains a fixed spatial profile and propagates at a constant velocity determined by the interaction between local cellular dynamics and diffusive coupling. Despite their idealized nature, traveling waves capture essential features of action potential conduction in cardiac tissue, including how conduction velocity, wavefront steepness, refractory structure, and wave stability depend on tissue and cellular properties, while remaining amenable to systematic mathematical analysis.^{19,23,24} Moreover, traveling waves act as organizing structures for more complex spatiotemporal dynamics: Their existence, stability, and bifurcation structure often determine how tissue responds to perturbations, pacing, or parameter changes, and underlie phenomena such as threshold behavior, conduction block, alternans, and spiral wave breakup.^{6,11,12,25–31}

Importantly, traveling waves provide an ideal framework for systematic comparisons across model hierarchies. By reducing wave propagation to an idealized setting, traveling waves provide a common mathematical framework in which minimal and biophysically detailed models can be directly compared through quantitative measures such as wave speed, wave profile, spectral stability, and their associated bifurcation structure.

Despite substantial differences in model formulation between minimal models and biophysically detailed conductance-based descriptions, both classes of models can be expressed as reaction–diffusion systems in which spatial coupling acts through the membrane potential. This common mathematical framework allows traveling waves in models of very different complexity to be analyzed using the same mathematical tools and compared on an equal footing. In one spatial dimension, they take the general form,

$$\begin{aligned}\frac{\partial \mathbf{u}}{\partial t} &= D \frac{\partial^2 \mathbf{u}}{\partial x^2} + F(\mathbf{u}, \mathbf{p}), \\ \mathbf{u}(t, 0) &= \mathbf{u}(t, \Lambda), \\ \frac{\partial \mathbf{u}}{\partial x}(t, 0) &= \frac{\partial \mathbf{u}}{\partial x}(t, \Lambda),\end{aligned}\quad (4)$$

where $\mathbf{u}(t, x) \in \mathbb{R}^n$ is the state vector, $\mathbf{p} \in \mathbb{R}^p$ is a vector of system parameters, $F(\mathbf{u}, \mathbf{p}) \in \mathbb{R}^n$ governs the local reaction dynamics, and D is a nonnegative diagonal matrix of diffusion coefficients. In tissue-level cardiac models, electrical coupling occurs through gap junctions between neighboring cells. Consequently, only the membrane potential (or voltage-like variable) diffuses, and D , therefore, has a single nonzero entry.

We focus on traveling waves that propagate on a ring of tissue with circumference Λ , which imposes periodic boundary conditions. Ring geometry is widely used in both modeling^{26,29,32–36} and experiments^{32,35,37–39} to study the onset of alternans and reentrant arrhythmias. In addition, ring geometry allows both stable and unstable traveling waves to be computed over a wide range of wave periods, making it especially well suited to systematic comparisons across models.

To analyze propagating waves, we introduce a coordinate system moving at speed c by defining $(\tau = t, \xi = x + ct)$ and write $\mathbf{u}(t, x) = \mathbf{U}(\tau, \xi)$. In these coordinates, the governing equations become

$$\begin{aligned}\frac{\partial \mathbf{U}}{\partial \tau} &= D \frac{\partial^2 \mathbf{U}}{\partial \xi^2} - c \frac{\partial \mathbf{U}}{\partial \xi} + F(\mathbf{U}, \mathbf{p}), \\ \mathbf{U}(\tau, 0) &= \mathbf{U}(\tau, \Lambda), \\ \frac{\partial \mathbf{U}}{\partial \xi}(\tau, 0) &= \frac{\partial \mathbf{U}}{\partial \xi}(\tau, \Lambda).\end{aligned}\quad (5)$$

A. Traveling wave solutions as steady state solutions

Traveling waves are non-trivial solutions of the reaction–diffusion system (4) that propagate with a fixed shape and constant speed c . In the comoving coordinate system (5), these propagated waves correspond to steady state solutions. Consequently, the problem of computing traveling-wave solutions reduces to solving the boundary value problem (BVP),

$$\begin{aligned}0 &= D \frac{\partial^2 \mathbf{U}}{\partial \xi^2} - c \frac{\partial \mathbf{U}}{\partial \xi} + F(\mathbf{U}, \mathbf{p}), \\ \mathbf{U}(0) &= \mathbf{U}(\Lambda), \\ \frac{\partial \mathbf{U}}{\partial \xi}(0) &= \frac{\partial \mathbf{U}}{\partial \xi}(\Lambda).\end{aligned}\quad (6)$$

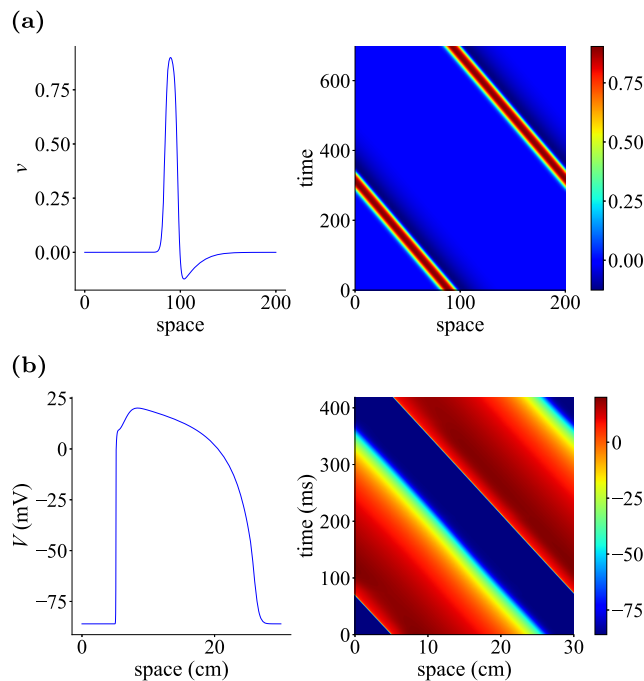


FIG. 1. Traveling waves in the (a) FitzHugh–Nagumo equations using $\Lambda = 200$ (b) nine-variable ten Tusscher–Panfilov model using $\Lambda = 30$ cm. The left column presents snapshots of the action potential profiles. The kymographs in the right column show traveling waves propagating through space over time. The colorbar units for the nine-variable ten Tusscher–Panfilov model are mV.

Note that the wave speed c must be determined simultaneously with the wave profile $\mathbf{U}$. Each traveling-wave solution is, therefore, associated with a unique propagation speed, a relationship that underlies the dispersion curves presented in later sections.²⁰

Figure 1 shows examples of traveling waves propagating on a ring in the FHN and the TP06 models. The left column shows snapshots of the traveling wave profiles for the voltage variable, and the right column presents space–time kymographs illustrating the constant leftward propagation of the waves in the periodic domain. These examples highlight the marked differences in wave profile that arise with increasing physiological complexity and foreshadow the more complex traveling-wave dynamics explored in later sections.

Although the FHN model can be tuned to reproduce basic features of the TP06 action potential, such as action potential duration and approximate upstroke velocity, its waveform remains relatively simple. In particular, the FHN AP has a roughly symmetric rise and fall reflecting a single fast excitation process and a single slow recovery process. In contrast, the TP06 action potential is distinctly multiphasic, exhibiting a very sharp upstroke, an early notch, a prolonged plateau, and gradual multi-sloped repolarization, indicative of multiple depolarizing and repolarizing processes with distinct kinetics acting over a wide range of timescales. The ultrafast sodium current produces the steep wavefront, while slower calcium and potassium currents shape the plateau and extended repolarization. The absence of such timescale separation in the FHN model enforces

similar steepness of the wavefront and waveback, as seen by comparing Figs. 1(a) and 1(b). As shown in Secs. V and VI, these differences are not limited to wave profile but extend to the existence, stability, and bifurcation structure of traveling-wave solutions.

B. Stability of traveling waves

Once a traveling-wave solution $\tilde{\mathbf{U}}$ has been computed, linear stability analysis characterizes how it responds to small perturbations. As shown later, changes in stability identify the bifurcations that organize the families of traveling-wave solutions and the transitions between different propagation regimes.

Stability analysis is performed by considering perturbations to $\tilde{\mathbf{U}}$ of the form,

$$\mathbf{U}(\tau, \xi) = \tilde{\mathbf{U}}(\xi) + e^{\lambda\tau} \mathbf{V}(\xi),$$

which, upon substitution into (5) and linearizing about $\tilde{\mathbf{U}}$, yields the eigenvalue problem,

$$\begin{aligned} \lambda \mathbf{V} &= D \frac{\partial^2 \mathbf{V}}{\partial \xi^2} - c \frac{\partial \mathbf{V}}{\partial \xi} + F_U(\tilde{\mathbf{U}}(\xi), \mathbf{p}) \mathbf{V}, \\ \mathbf{V}(0) &= \mathbf{V}(\Lambda), \\ \frac{\partial \mathbf{V}}{\partial \xi}(0) &= \frac{\partial \mathbf{V}}{\partial \xi}(\Lambda), \end{aligned} \quad (7)$$

where $\mathbf{V} \in \mathbb{C}^n$ is an eigenfunction with eigenvalue $\lambda \in \mathbb{C}$, and F_U corresponds to the Jacobian matrix of the reaction dynamics F linearized about $\tilde{\mathbf{U}}$. The eigenvalues determine how perturbations evolve over time: eigenvalues with negative real part correspond to decaying perturbations, whereas eigenvalues with positive real part indicate growing perturbations and instability. Because a traveling wave may be shifted to any position on the ring without changing the solution, the system always possesses a zero eigenvalue with eigenfunction $\mathbf{V}(\xi) = \partial_\xi \tilde{\mathbf{U}}(\xi)$. Aside from this zero eigenvalue, the traveling wave $\tilde{\mathbf{U}}$ is spectrally stable if all remaining eigenvalues have negative real part.

Remark. Throughout this paper, we focus on solutions posed on a finite periodic domain, for which the spectrum consists of discrete eigenvalues. On infinite domains, the relevant stability information is contained in the essential spectrum; see Refs. 40, 20, and 21 and references therein.

C. Fast waves, slow waves, and dispersion curves

For sufficiently high excitability and sufficiently large spatial period (e.g., ring circumference), models of cardiac tissue and excitable media generally admit a stable traveling wave solution that is observed in direct numerical simulations of the PDE system (4). In addition to this stable solution, there typically exists an unstable traveling wave that propagates more slowly and is linearly unstable. These two solutions are commonly referred to as the fast wave and slow wave, respectively.^{41–44} As excitability or spatial period is decreased, the fast and slow waves typically coalesce in a saddle-node bifurcation, beyond which no traveling wave solutions exist. While the slow wave is always unstable, the fast wave branch is stable for a large range of Λ . However, before disappearing via the saddle-node

bifurcation, the fast wave sometimes loses stability through secondary bifurcations, leading to phenomena such as alternans or the absence of stable propagation.^{33,45–47}

Although unstable traveling waves cannot be robustly observed in direct simulations of the PDE model (4), their existence and structure play a central role in organizing wave dynamics. In particular, the slow wave and its stable manifold define the threshold for successful wave propagation.⁴⁸ Moreover, properties of the slow wave have been linked to the conditions for wave reflection, which, in turn, are linked to the initiation of reentrant arrhythmias.^{30,49,50}

Bifurcation diagrams provide a compact summary of the parameter dependence of traveling wave existence, stability, and properties. In the cardiac electrophysiology literature, restitution curves that plot CV and APD as functions of pacing period or spatial wavelength are commonly computed from direct numerical simulations and characterize stable propagating waves.^{6,25,31,51} These curves can be extended to include unstable solutions, yielding bifurcation diagrams that relate wave speed to spatial or temporal period. Such diagrams are often referred to as dispersion relations in the context of traveling waves.^{20,21,33} Throughout the remainder of the paper, we use the term dispersion curves or dispersion relations to refer generically to bifurcation diagrams of traveling wave solutions, regardless of the specific bifurcation parameter employed.

IV. NUMERICAL APPROACHES FOR COMPUTING DISPERSION CURVES

A. Computational tasks

Analytical traveling-wave solutions and their spectra are generally unavailable. Therefore, computing a dispersion curve requires a sequence of computational steps, each presenting distinct numerical challenges related to accuracy, robustness, and computational efficiency. These steps are summarized below.

1. Solve for $U(\xi)$ and c

Computing a traveling-wave solution $U(\xi)$ and its wave speed c entails solving the BVP (6), together with a phase condition that eliminates the nonuniqueness arising from translational invariance.⁵² After discretization, the BVP becomes a large system of nonlinear algebraic equations that can be solved using a root-finding algorithm. Because Newton-type methods require a sufficiently accurate initial guess, a robust procedure for generating an initial traveling wave is also needed. Furthermore, biophysically detailed cardiac models often exhibit extremely stiff local dynamics, producing steep spatial gradients whose accurate numerical resolution requires fine spatial discretizations.

2. Estimate the spectrum

Once a traveling-wave solution $U(\xi)$ and its wave speed c have been computed, its stability is determined by solving the eigenvalue problem (7). As with the BVP, the spatial derivatives in the eigenvalue problem must be discretized, yielding a large matrix eigenvalue problem. For stiff cardiac models, the fine spatial meshes required to resolve steep wave fronts can make this eigenvalue problem computationally demanding. Moreover, eigenvalue computations are highly sensitive to discretization and rounding errors,

which can significantly degrade the accuracy of the computed spectrum.⁵³ Therefore, accurately determining stability is often the most computationally demanding step in computing complete dispersion curves for biophysically detailed models.

3. Perform numerical continuation

Computing a dispersion curve requires a path-following algorithm, such as arclength or Moore–Penrose continuation, to trace the family of traveling-wave solutions as the spatial period or another continuation parameter varies.⁵⁴ Performing numerical continuation requires repeatedly solving the traveling-wave BVP (6) and, when stability is computed, the associated eigenvalue problem (7). In addition, numerical continuation enables the detection and accurate localization of bifurcation points, such as saddle-node and Hopf bifurcations, that organize the structure and stability of the dispersion curve. Consequently, the overall computational efficiency of generating a dispersion curve depends critically on the efficiency and robustness of the underlying traveling-wave and stability computations.

B. Existing continuation software

Existing continuation and bifurcation software provides powerful and highly flexible frameworks for the numerical analysis of differential equations. These packages support a broad range of applications and have been successfully applied to traveling-wave problems. However, computing complete dispersion curves for stiff, conductance-based cardiac models requires combining several computational tasks into a single workflow, often with numerical methods tailored to the large spatial gradients and high dimensionality of these systems. We briefly review the capabilities of existing software before introducing TWIST.

AUTO⁵⁵ is one of the most widely used software packages for numerical continuation, providing robust path-following capabilities for traveling-wave boundary-value problems through orthogonal collocation.^{56,57} Because AUTO is designed as a general continuation package rather than an integrated traveling-wave environment, users typically combine it with additional software for generating initial waves and performing spectral stability analysis.⁴⁰ WAVE-TRAIN⁵⁸ simplifies the use of AUTO by providing a front-end for traveling-wave computations, although users must still provide much of the problem-specific setup, and large stiff cardiac models remain computationally demanding because of the fine spatial discretizations required for accurate stability calculations. Similarly, *pde2path*⁵⁹ provides a flexible MATLAB-based finite-element framework for continuation and bifurcation analysis of PDEs, but its flexibility requires users to formulate the model, specify phase conditions, choose discretizations, configure continuation parameters, and select appropriate eigensolvers. Other continuation environments, including *BifurcationKit.jl*,⁶⁰ *COCO*,⁶¹ and *MATCONT*,⁶² likewise provide powerful tools for numerical bifurcation analysis and have also been applied to traveling-wave problems.

Although these software packages provide powerful continuation and bifurcation capabilities, computing complete dispersion curves for biophysically detailed cardiac models still requires integrating the computational tasks outlined in Sec. IV A into an efficient and robust workflow. TWIST was developed to provide

this integrated workflow while employing numerical techniques specifically designed for stiff conductance-based reaction–diffusion systems.

C. TWIST: Traveling wave investigation software tool

We introduce the Traveling Wave Investigation Software Tool (TWIST), a standalone software package that automates the workflow for computing traveling-wave solutions, spectral stability, and dispersion curves in reaction–diffusion systems. Unlike general-purpose continuation software, TWIST was designed specifically for stiff conductance-based cardiac models and other systems with large spatial gradients. Here, we briefly outline the principal numerical methods employed by TWIST; a detailed description of the algorithms and user interface is provided in the TWIST manual⁶³ and in Ref. 64. The performance of TWIST is benchmarked against AUTO in the Appendix.

Importantly, TWIST largely automates this workflow from a single user-written JSON input file describing the reaction–diffusion system. No additional software is required to generate initial guesses, perform stability analysis, or continue solution branches. Furthermore, any computed traveling-wave solution can serve directly as the starting point for further continuation or time-dependent simulations.

Because Newton-type methods require a sufficiently accurate initial guess, TWIST automatically generates an initial traveling-wave solution through direct simulation of the reaction–diffusion system. The resulting propagating wave, together with an estimate of the wave speed, serves as the initial guess for the traveling-wave boundary-value solver.

To compute traveling-wave solutions, TWIST uses the Runge–Kutta representation of orthogonal collocation⁶⁵ to discretize the boundary value problem (6). In doing so, the second-order system is first reformulated as a first-order system in the spatial variable to avoid the approximation of second derivatives. The high-order collocation methods together with adaptive mesh refinement allows for accurate resolution of the steep gradients on relatively coarse meshes. The nonlinear collocation equations are solved for the wave profile U and wave speed c using a global Newton method with adaptive damping to improve convergence in models with large spatial gradients and narrow basins of attraction. The damping factor is selected using a combination of residual-based and error-oriented descent tests to prevent overshoot and drive residuals close to machine precision. Iterations of Newton's method require solving large linear systems arising from the collocation discretization of the Jacobian matrices. To do so efficiently, TWIST employs a novel tree-based factorization that is naturally parallelizable.^{63,64} The global Newton solver is embedded within approximate Moore–Penrose continuation⁵⁴ to efficiently track solutions through parameter space and trace out dispersion curves.

Stability is assessed by solving the eigenvalue problem (7) using the same collocation discretization employed for the traveling-wave BVP, which eliminates errors associated with switching numerical schemes. By utilizing high-order methods, this collocation approach achieves smaller global errors with relatively coarse meshes, ensuring that the resulting discretized operators remain computationally tractable. Furthermore, eigenvalues are computed using a novel

algorithm (xGGEV4) for the generalized eigenvalue problem that is faster and scalable for large matrices;^{63,64} the new algorithm builds upon the ideas from Steel and Vandebriel for the Hessenberg-triangular reduction.⁶⁶

Numerical continuation is performed using approximate Moore–Penrose continuation,⁵⁴ allowing TWIST to efficiently trace solution branches through parameter space while automatically detecting bifurcation points. Because each continuation step reuses the previously computed traveling-wave solution, complete dispersion curves can be computed efficiently with minimal user intervention.

V. A COMPARISON OF TRAVELING WAVES IN MINIMAL AND BIOPHYSICALLY DETAILED MODELS

Traveling wave solutions and their bifurcations are well characterized in minimal models of excitable media, but it remains unclear which elements of this structure persist in biophysically detailed cardiac models that incorporate multiple interacting activation and recovery processes. In this section, we use TWIST to systematically compare the existence, stability, and bifurcation structure of traveling waves across three models of increasing physiological complexity: the FitzHugh–Nagumo (FHN) equations, the Beeler–Reuter (BR) model, and the 9-variable ten Tusscher–Panfilov (TP06) model. By computing dispersion curves and spectra in each system, we identify both robust organizing principles shared across models and qualitative changes introduced by the rich ionic current structure of the cardiac models. We first summarize the overall structure of the dispersion curves in Subsection V A, then examine bifurcations of fast wave solutions in Subsection V B, and finally explore novel properties of slow waves in Subsection V C.

Default parameters for each model are as follows. For the FHN, $\alpha = 0.25$, $\gamma = 5.0$, $\varepsilon = 0.003$, and $D = 1$. Parameters for the BR model are taken from the original formulation,⁴ with diffusion coefficient $D = 0.001 \text{ cm}^2/\text{ms}$.²⁶ Parameters for the TP06 model correspond to the M-cells configuration in the original study with diffusion coefficient $D = 0.00154 \text{ cm}^2/\text{ms}$.¹⁵

A. Dispersion relations in FHN, BR, and TP06

Dispersion curves computed using TWIST for the FHN, BR, and TP06 models are shown in the top row of Fig. 2. These curves describe the existence, stability, and wave speed c (conduction velocity, CV) of traveling wave solutions to system (4) as a function of ring circumference Λ . The bottom row of Fig. 2 shows corresponding APD90 restitution curves. In this and subsequent figures, black curves denote stable solution branches and gray curves denote unstable solution branches. The temporal period T of the traveling waves is related to the spatial period by $\Lambda = cT$.

The FHN model exhibits the canonical dispersion curve structure described in Sec. III C [Fig. 2(a)]. For sufficiently large Λ , a branch of fast wave solutions and a branch of slow wave solutions exist. These two solution branches coalesce at a saddle–node bifurcation (pink square), below which traveling wave solutions do not exist.

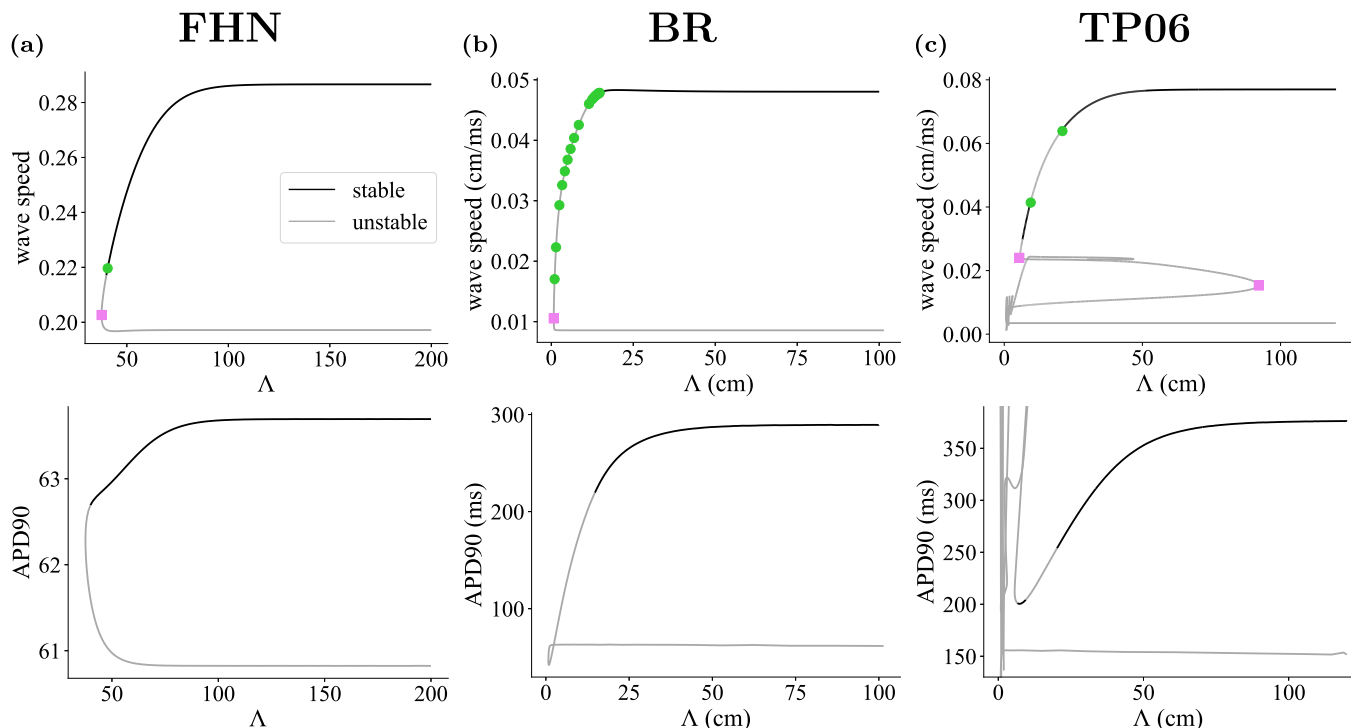


FIG. 2. Dispersion curves (top row) and APD90 restitution curves (bottom row) computed using TWIST. Black and gray lines denote stable and unstable solution branches, respectively. (a) FitzHugh–Nagumo (FHN) model; (b) Beeler–Reuter (BR) model; (c) ten Tusscher–Panfilov (TP06) model. Pink squares indicate a saddle-node bifurcation of the fast wave and slow wave branches. Green circles indicate Hopf bifurcations at which the fast-wave branch loses or regains stability. Although both the BR and TP06 models exhibit cascades of Hopf bifurcations, only the primary Hopf bifurcations are marked for the TP06 model to preserve the readability of the figure. The TP06 model has two disjoint solution branches: one connected to the fast wave and one connected to the sodium spike. The vertical axis of the TP06 APD90 restitution curve is truncated at 390 ms to highlight the changes associated with the destabilization of the fast-wave branch; the complete APD90 restitution curve is shown in Fig. 5(e).

As Λ decreases, the fast wave loses stability through a Hopf bifurcation (green circle) located on the steep portion of the dispersion curve, close to the saddle-node bifurcation and immediately preceding the steep region of the APD restitution curve. At this point, a complex-conjugate pair of eigenvalues crosses the imaginary axis, giving rise to an oscillatory instability. As Λ decreases further, this complex-conjugate pair becomes two positive real eigenvalues. At the saddle-node bifurcation, one of these eigenvalues passes through zero, leaving the slow-wave branch with a single unstable mode.

For large Λ , the propagation speed of the fast wave is approximately constant; however, it decreases sharply near the saddle-node bifurcation as the recovery window shrinks with decreasing ring circumference. These trends are reflected in the APD90 restitution curve, but the variation in APD is small: the fast wave APD changes by less than 1.5%, and the maximum difference between the APDs of the fast and slow waves is less than 5%.

Representative fast and slow wave solutions ($\Lambda = 200$) are shown in Fig. 3(a). In the FHN model, the slow wave is essentially a slightly scaled-down version of the fast wave. Along the fast-wave branch, the traveling-wave profile remains nearly unchanged for large Λ , with an amplitude of approximately 0.9. Near the Hopf and

saddle-node bifurcations, however, the amplitude decreases rapidly to approximately 0.7, while the overall shape is largely preserved. The slow-wave branch inherits this profile and maintains a very similar profile as Λ varies. Movie 1 in the [supplementary material](#) shows the smooth transition of the wave profile along the FHN dispersion curve.

The bifurcation structure for the FHN model described above is robust to changes in the excitability parameter ϵ . For decreasing ϵ , the qualitative structure of the dispersion curve is preserved. As ϵ increases, the Hopf bifurcation and the saddle-node bifurcation approach one another and coalesce in a Takens–Bogdanov bifurcation. Beyond this point, the Hopf bifurcation no longer exists, and the fast-wave branch does not undergo an oscillatory instability. As ϵ increases further, there is a critical value above which the system loses excitability and traveling wave solutions no longer exist.

Despite the considerably greater complexity of the BR model, many aspects of the FHN structure are preserved in its dispersion curve [Fig. 2(b)]. In particular, the fast and slow wave branches meet and annihilate at a saddle-node bifurcation (pink square), and the fast wave loses stability through a Hopf bifurcation (green circle) prior to the saddle-node bifurcation. Furthermore, the propagation

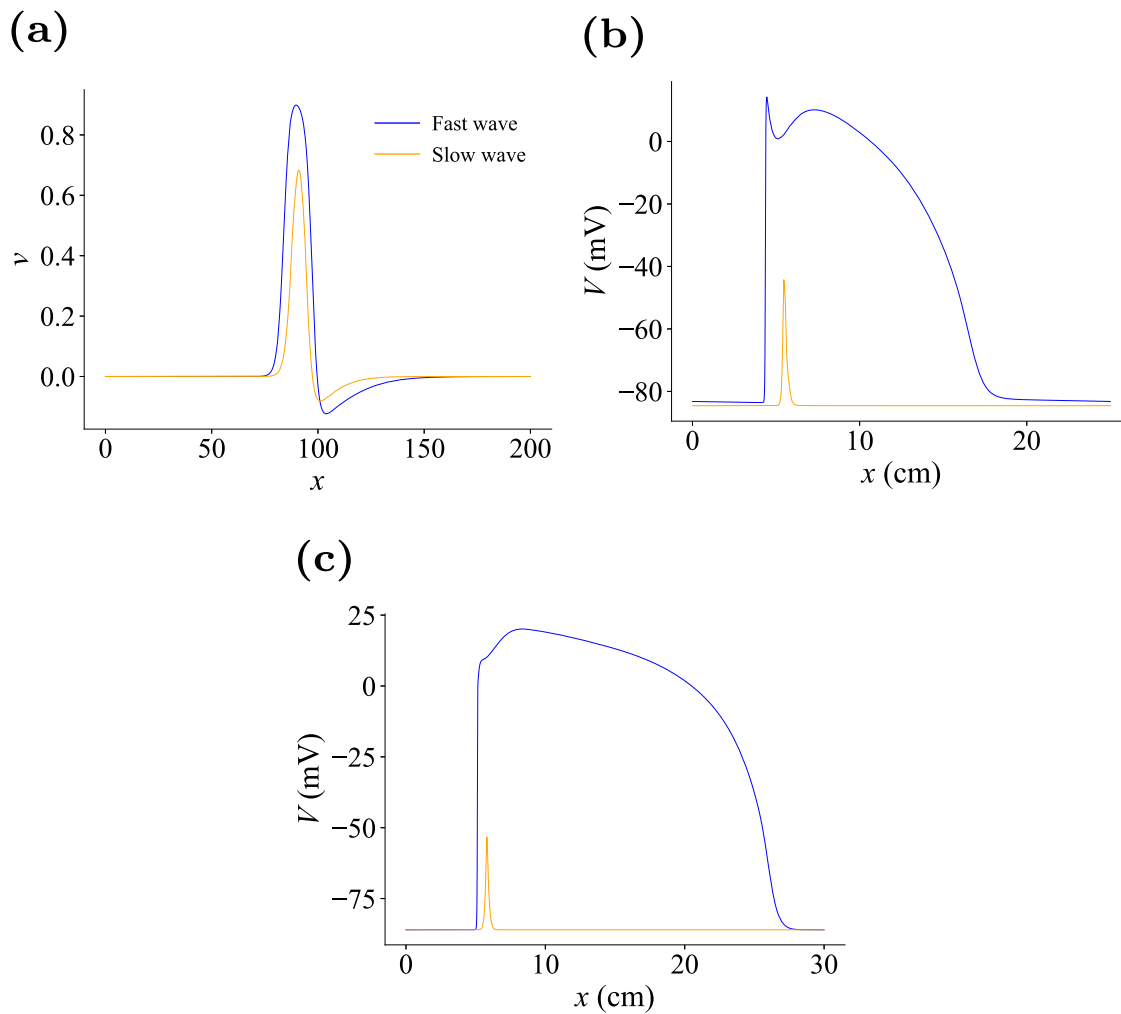


FIG. 3. Spatial profiles of the fast (blue curves) and slow (golden curves) traveling wave solutions. Panel (a) shows the profiles for the FHN system, where the slow wave appears as a scaled-down version of the fast wave with a roughly symmetric rise and fall. The spatial period is $\Lambda = 200$. Panels (b) and (c) display the membrane potential profiles for the BR and TP06 models, respectively. In these biophysically detailed models, the fast waves exhibit the spike-notch-dome morphology, featuring a sharp initial upstroke followed by an initial repolarization (BR) or delay (TP06) before a prolonged plateau; in contrast, the standard slow wave solutions manifest as narrow sodium spikes. The spatial periods for (b) and (c) are $\Lambda = 25$ cm and $\Lambda = 30$ cm, respectively.

speed of the fast wave exhibits a plateau for sufficiently large Λ , while the speed of the slow wave is largely independent of Λ .

Profiles of representative fast and slow wave solutions ($\Lambda = 25$ cm) are shown in Fig. 3(b). The fast wave exhibits the spike-notch-dome morphology characteristic of cardiac action potentials. Unlike the FHN model, the slow wave is not simply a scaled-down version of the fast wave; instead, it takes the form of a narrow spike. Analysis of the underlying activation and inactivation processes indicates that this slow wave is driven primarily by the sodium current. We, therefore, refer to this type of slow wave as a “sodium spike.”

Two notable differences from the FHN model arise along the fast wave branch. First, the Hopf bifurcation in the BR model occurs

at a much larger spatial period Λ and approximately coincides with the end of the fast wave CV plateau. In this range of Λ , the propagation speed remains nearly constant, but the APD decreases rapidly [Fig. 2(b)]. As a result, the fast wave loses stability through an APD restitution-driven instability associated with the steep APD restitution curve, consistent with prior studies of the BR model.^{25,33,51}

Second, beyond the initial Hopf bifurcation, the fast-wave branch undergoes a cascade of additional Hopf bifurcations, leading to a substantial increase in the dimension of the unstable subspace (up to 16). This loss of stability is subsequently reversed through a corresponding sequence of Hopf bifurcations, which restabilize these modes. The remaining unstable pair of complex-conjugate eigenvalues then splits into two positive real eigenvalues. As in the

FHN model, at the saddle-node bifurcation one of these eigenvalues passes through zero, leaving the slow-wave branch with a single unstable mode.

Along the fast-wave branch, the waveform remains largely unchanged for large Λ , with APD ~ 290 ms. As Λ decreases, the height of the action potential plateau, the APD, and the wave speed decrease smoothly, including beyond the Hopf bifurcation where the fast wave is unstable. At the saddle-node bifurcation, the action potential approaches that of a sodium spike. Along the slow-wave branch, in contrast, the sodium-spike morphology and propagation speed remain largely unchanged across Λ . Movie 2 in the [supplementary material](#) shows the smooth transition of the wave profile along the BR dispersion curve.

The TP06 model exhibits a fundamentally richer and more complex bifurcation structure than either the FHN or BR models. TWIST identifies a total of six branches of traveling wave solutions, giving the dispersion curve [Fig. 2(c)] a qualitatively different structure from those of the FHN and BR models. Two of these solution branches correspond to the expected fast wave (top branch) and the sodium spike slow wave (bottom-most branch). These are the only two solutions found for spatial periods of $\Lambda \gtrsim 100$, and they have profiles similar to those in the BR model. Representative profiles of the fast wave and the sodium-spike slow wave for $\Lambda = 30$ cm are shown in Fig. 3(c). Moreover, the dependence of CV and APD on Λ for these waves is similar to that in the BR model.

As Λ is decreased, the stability of the fast wave initially mirrors that of the BR model and a Hopf bifurcation (green circle) occurs just beyond the CV plateau, consistent with the steep APD90 restitution curve at this point. As in the BR model, this is followed by a cascade of Hopf bifurcations. However, unlike the BR model, the fast wave branch in the TP06 model re-stabilizes over a range of Λ before undergoing a second sequence of destabilizing Hopf bifurcations and ultimately terminating at the saddle-node bifurcation. As a result, the TP06 model supports stable propagating waves over a broader and more complex range of spatial periods.

Perhaps the most striking difference between the TP06 model and the FHN and BR models is the organization of the slow-wave branches. In the FHN and BR models, the fast wave branch directly connects through a saddle-node bifurcation to a single slow wave branch, i.e., the branch of sodium spike slow waves. In contrast, the TP06 model supports multiple distinct slow-wave branches. At the saddle-node bifurcation (pink square), the fast wave branch coalesces with a slow wave branch whose solutions are largely depolarized. This depolarized slow wave branch undergoes additional bifurcations and terminates at low Λ , and it does not appear to directly connect to the branches containing the sodium spike slow waves. The structures of these slow waves and their solution branches are investigated further in Sec. V C.

B. Instability of fast waves

The fast wave solutions in the FHN (with default parameters), BR, and TP06 models all lose stability via a Hopf bifurcation as the ring circumference Λ is decreased. The dynamical consequences of this instability, however, are model dependent.

Figure 4 shows kymographs obtained from simulations using TWIST. The top row displays stable fast waves for values of Λ

slightly larger than the corresponding Hopf bifurcation points. In this regime, waves propagate with a constant profile and conduction velocity. The second row of Fig. 4 illustrates the spatiotemporal behaviors for Λ just below the Hopf bifurcation, where the fast wave is unstable. In each case, simulations are initialized with a small perturbation of the corresponding unstable fast wave solution.

The kymograph in Fig. 4(a) (bottom) indicates that the Hopf bifurcation in the FHN model is subcritical. For Λ below the Hopf bifurcation, no stable propagating structures are observed. Instead, perturbations of the unstable fast wave produce transient propagation that ultimately decays to the resting state, which appears to be the unique stable state in this regime. This behavior is observed for all ϵ for which a Hopf bifurcation exists.

In contrast, the Hopf bifurcations in the BR and TP06 models are supercritical and give rise to stable time-periodic solutions. Figures 4(b) (bottom) and 4(c) (bottom) show that perturbations of the unstable fast wave lead to the emergence of alternans, characterized by alternating long and short action potentials. Long-duration simulations using TWIST indicate that these alternans solutions are stable over a finite range of Λ near the Hopf bifurcation. These findings are consistent with prior studies of restitution-driven alternans in both the BR and TP06 models.^{6,25,33,51}

C. Slow waves and their ionic mechanisms of the TP06 model

Unlike the FHN and BR models, which each exhibit a single slow-wave branch, the TP06 model has five distinct branches of unstable slow-wave solutions [Fig. 2(c)]. In addition to its more complex dispersion curve structure, the TP06 model gives rise to a much broader range of waveform morphologies than in the FHN and BR models. The branch with the slowest wave speed corresponds to the sodium-spike slow waves observed in the BR model; a representative example is shown in Fig. 3(c). The remaining four branches consist of solutions that are largely depolarized across their profiles. Representative solutions from these four branches are shown in panels (a)-(d) of Fig. 5, ordered by decreasing conduction velocity at $\Lambda = 30$. The full APD90 diagram for all six branches (including the fast-wave branch) is shown in Fig. 5(e). The smooth transition of the wave profile along the branches of the TP06 dispersion curve is described below and shown in Movie 3 of the [supplementary material](#).

Along the fast-wave branch, passing through the saddle-node bifurcation and onto the associated slow wave branch, the notch of the action potential (phase 1) broadens and effectively becomes a plateau. That is, the interval between the rapid upstroke (phase 0) and the dome (phase 2) increases dramatically. Figure 5(a) provides the waveform midway along this slow wave branch: the upstroke is located at $x \sim 5$ cm, the APD90 exceeds 1500 ms, phase 1 now comprises the majority of the waveform (approximately 80%), and the dome resembles an early afterdepolarization (EAD). Further along the branch, APD90 exceeds 6000 ms. Beyond a second saddle-node bifurcation ($\Lambda \sim 95$), the upstroke amplitude decreases, phase 1 develops a positive slope, and the overall APD decreases. The dome (or EAD-like feature) sharpens and develops a secondary oscillation, as shown in Fig. 5(b). Further along the branch, this

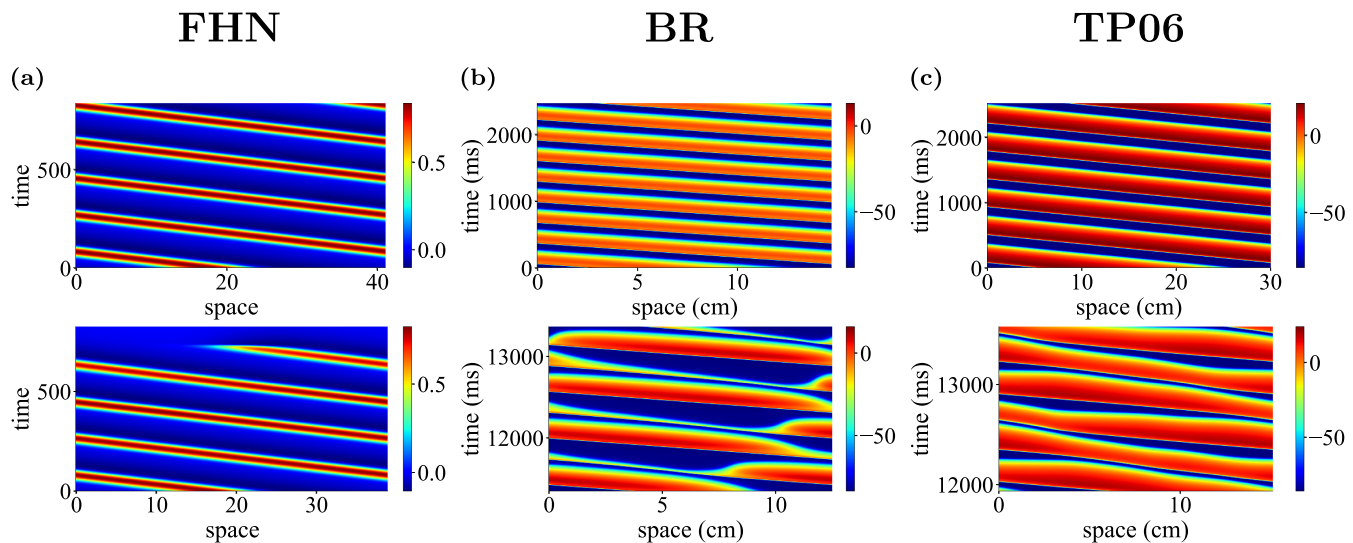


FIG. 4. Spatiotemporal kymographs of wave simulations generated using TWIST. (a) FitzHugh–Nagumo (FHN) model; (b) Beeler–Reuter (BR) model; (c) ten Tusscher–Panfilov (TP06) model. The top row shows stable fast wave propagation for values of Λ prior to the initial Hopf bifurcation [(a) $\Lambda = 41.16$, (b) $\Lambda = 14.72$, (c) $\Lambda = 20.21$]. The bottom row shows the dynamics for Λ just below the Hopf bifurcations [(a) $\Lambda = 38.96$, (b) $\Lambda = 12.5$, (c) $\Lambda = 15.1$], with initial conditions given by small perturbations of the unstable fast-wave solution. In the FHN model, the system decays to the resting state following transient propagation. In the BR and TP06 models, the instability gives rise to alternans, characterized by alternating long and short action potentials. The color bars for panels (b) and (c) indicate the membrane potential in mV.

oscillation deepens, and in the most complex region of the dispersion curve at very small Λ (approximately 0–3 cm), the waveform transitions to a pattern resembling a pair of closely spaced sodium spike slow waves with amplitudes of ~ 30 mV.

A second set of slow-wave branches is connected to the sodium-spike slow wave branch. Along this branch, the waveform retains the characteristic sodium-spike shape, with amplitudes of approximately 30 mV, over a wide range of Λ . Only for very small Λ (0–3 cm), corresponding to the most complex region of the dispersion curve, does the profile begin to change significantly. In this complex region, the waveform develops a two-component structure: the primary peak increases in amplitude toward that of a fast wave (although it lacks a sharp upstroke), while a notch forms on the upstroke but then separates to produce a secondary structure resembling a sodium-spike slow wave.

As the branch emerges from this low- Λ region, the primary peak sharpens and develops a fast-wave-like upstroke, while the secondary peak grows into a box-like waveform with a sharp upstroke and a plateau near -25 mV as in Fig. 5(c). Further along the branch, beyond a saddle-node bifurcation, the box-like waveform develops an EAD-like structure, resembling the phase-1 plateau–EAD slow wave shown in Fig. 5(a), while co-propagating with a second waveform that resembles a fast wave [Fig. 5(d)]. As Λ increases further, the plateau of the EAD-like wave shortens, and the two components become increasingly similar. Eventually, the solution approaches a pair of fast waves propagating on a ring, thus the branch connects to a period-doubled (second-harmonic) fast-wave solution, consisting of two waves over twice the spatial period.

We hypothesize that the increased complexity of the dispersion-curve structure in the TP06 model, as well as the diversity of slow-wave morphologies, arises largely from the interaction between two distinct excitation processes: the fast sodium current and the L-type calcium current. In both the BR and TP06 models, sodium activation is extremely rapid (timescales of 0.01–0.1 ms) with inactivation occurring over 1–10 ms. In contrast, the slow inward calcium current in the BR model has activation on the order of tens of milliseconds and inactivation extending up to ~ 1 s, yielding a relatively simple two-timescale structure. In the TP06 model, however, activation of the L-type calcium current is taken to be instantaneous and the two inactivation processes span tens to hundreds of milliseconds, consistent with experimental measurements in human ventricular myocytes. In particular, the effectively instantaneous activation of the calcium current allows it to participate on the same timescale as sodium-driven excitation, promoting direct interaction between the two excitation processes and enabling more complex wave dynamics.

To test this hypothesis, we vary the maximum conductance of the L-type calcium current, g_{CaL} , which controls the strength of the calcium-mediated excitation process. Figure 6(a) shows the resulting continuation curves. As g_{CaL} is decreased, the unstable branches associated with the complex slow-wave structures [Figs. 5(a)–5(d)] merge with the sodium-spike slow-wave branch and progressively deform toward the fast-wave branch. In the limit $g_{\text{CaL}} = 0$, when the L-type calcium current is eliminated, only the fast-wave and sodium-spike slow-wave branches remain. Thus, removing the calcium-mediated excitation process eliminates the

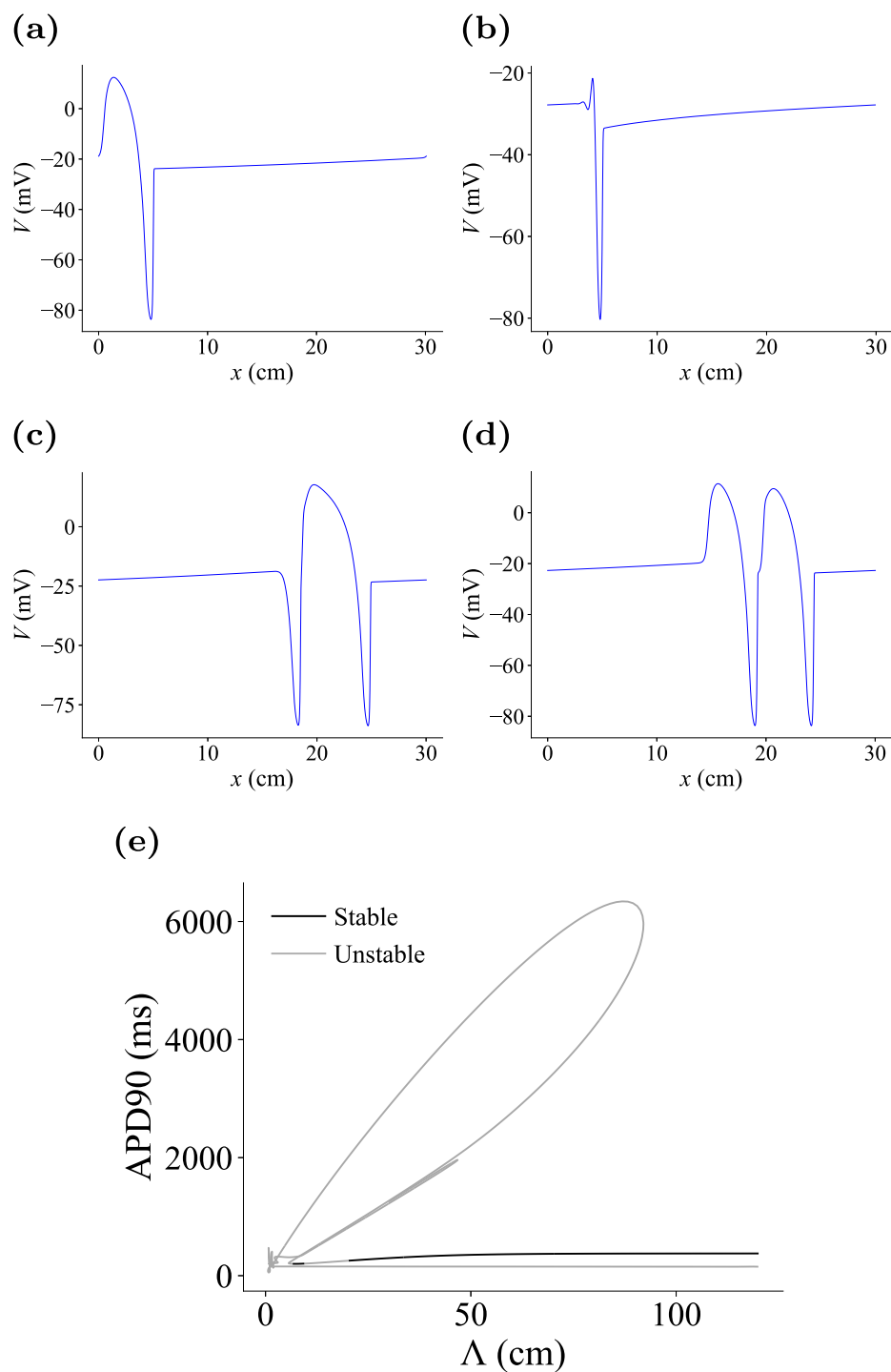


FIG. 5. Spatial profiles and APD90 curves for the additional unstable slow wave branches in the TP06 model. Panels (a)–(d) display representative wave profiles for the four depolarized slow wave branches computed at a spatial period of $\Lambda = 30$, organized with columns ordered from faster to slower wave speeds and rows ordered based on their connected solution branch. Specifically, the top row [panels (a) and (b)] displays slow waves that are connected to the branch of fast wave solutions, while the bottom row [panels (c) and (d)] displays slow waves that are connected to the sodium spike slow wave branch. These profiles feature sharp upstrokes, heavily depolarized lengths, and structures resembling early and delayed afterdepolarizations. Panel (e) shows the complete APD90 curves corresponding to the TP06 dispersion relation, reflecting the large APD90 values (reaching up to 6000 ms) of these depolarized branches.

additional branches and restores the canonical dispersion-curve structure observed in the FHN and BR models.

Figure 6(b) provides further evidence that the relative strengths of the excitation processes govern the structure of the dispersion

relation. By performing numerical continuation in the maximum conductance of the sodium calcium current g_{Na} at $\Lambda = 30$ cm, we find that number and organization of unstable solution branches depend sensitively on the magnitude of the sodium current. In

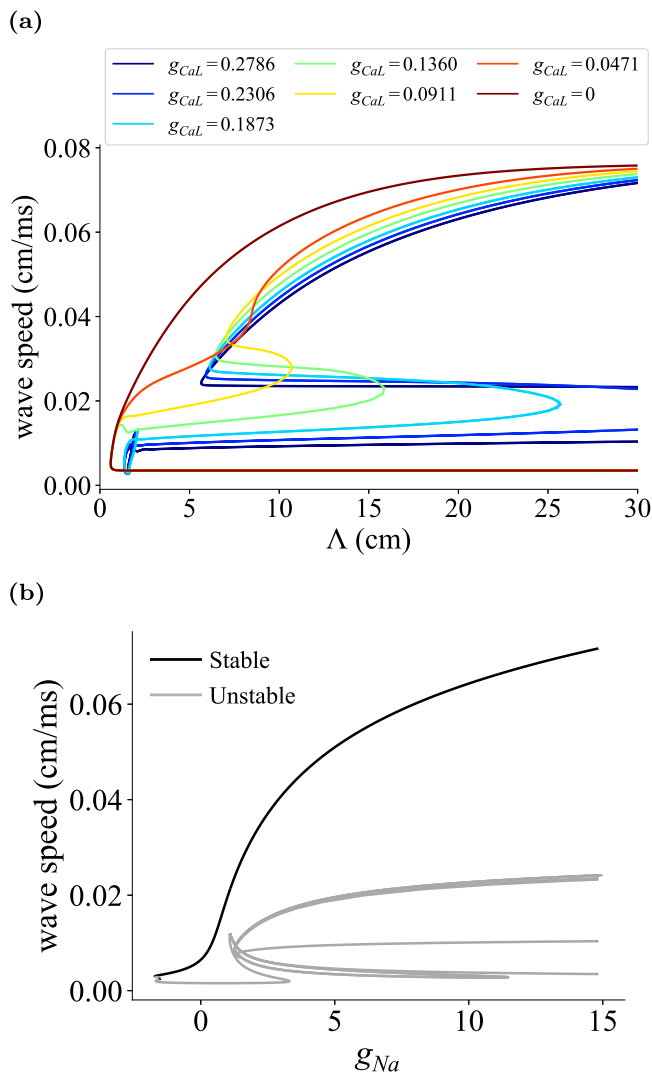


FIG. 6. (a) A family of dispersion curves for the TP06 model computed using TWIST across varying the maximum conductance of the L-type calcium current (g_{CaL} in mS/cm²). The plot illustrates the evolution of the wave speed solution branches as g_{CaL} is incrementally decreased from its baseline value of 0.2786 mS/cm² down to zero. The branches associated with the depolarized slow waves are shown morphing into the fast wave branch, culminating in a simplified two-branch structure (dark brown curve) when $g_{CaL} = 0$. (b) Dispersion relation for the TP06 model using the maximum conductance of the sodium calcium current g_{Na} in mS/cm² as the continuation parameter. The black and gray curves indicate stable and unstable solutions, respectively. The stable fast wave persists even for nonphysical, negative values. The number of unstable wave branches is dependent on the value of g_{Na} .

particular, a stable propagating wave persists even when $g_{Na} = 0$, in which case excitation and propagation are mediated entirely by the calcium current. Thus, the TP06 model supports wave propagation through either excitation mechanism individually, while the coexistence and interaction of both mechanisms give rise to the

additional branches and increased complexity observed in the full model.

VI. SLOW WAVES, THRESHOLD DYNAMICS, AND WAVE REFLECTION IN MINIMAL AND BIOPHYSICALLY DETAILED MODELS

Although slow waves are unstable, their existence and structure play a central role in organizing wave dynamics. In particular, slow waves and their stable manifolds define thresholds for successful wave propagation:⁴⁸ initial conditions below the stable manifold decay to the resting state, whereas those above it evolve into propagating fast waves.

Moreover, threshold properties of slow waves have been linked to the conditions that permit wave reflection,^{30,50} i.e., when an action potential traverses a structural or functional heterogeneity and subsequently generates a new wave that propagates in the retrograde direction.⁴⁹ Such reflected waves are a key mechanism underlying the initiation of reentrant arrhythmias.^{30,67–69} In this framework, the outcome of perturbations near the slow wave determines whether reflections are possible: if perturbations above the stable manifold evolve into a single forward-propagating fast wave, reflections cannot occur, whereas evolution into two counter-propagating waves indicates that reflections are supported.

Here, we use TWIST to demonstrate that the slow wave in the FHN model and the sodium-spike slow waves in the BR and TP06 models act as threshold structures separating propagation failure from successful wave propagation. To assess each model's ability to generate reflections, we initialize simulations near the slow-wave solution and perturb along the eigenfunction spanning its one-dimensional unstable manifold. Perturbations in the negative direction lead to decay to rest, while perturbations in the positive direction lead to fast wave propagation. The resulting dynamics determine whether the system supports reflections.

Figure 7 shows the evolution of perturbed slow waves in each model. In all cases, negative perturbations result in conduction failure, with activity decaying rapidly to the resting state (top row); positive perturbations lead to propagating waves (bottom row) but in a model-dependent manner. In the FHN and BR models, the perturbed slow wave evolves into a single fast wave propagating, indicating that reflections are not supported under the default parameterizations. In contrast, in the TP06 model, the perturbed slow wave evolves into two counter-propagating fast waves, indicating that reflections can occur under the default parameter set.

The transition between regimes that do not support reflections and those that do occurs through a global bifurcation and cannot be identified through linear stability analysis alone. To approximate the location of this transition, we use direct numerical simulations in TWIST to track the evolution of perturbations along the unstable manifold of the slow wave.

Figure 8 shows this transition as key excitability parameters are varied. In the FHN model, the transition occurs with a modest decrease in the excitability parameter ε (from $\varepsilon = 0.0013$ to $\varepsilon = 0.0012$), indicating that reflection-supporting dynamics are accessible within small parameter variations. In the BR model, the transition occurs only when the sodium conductance

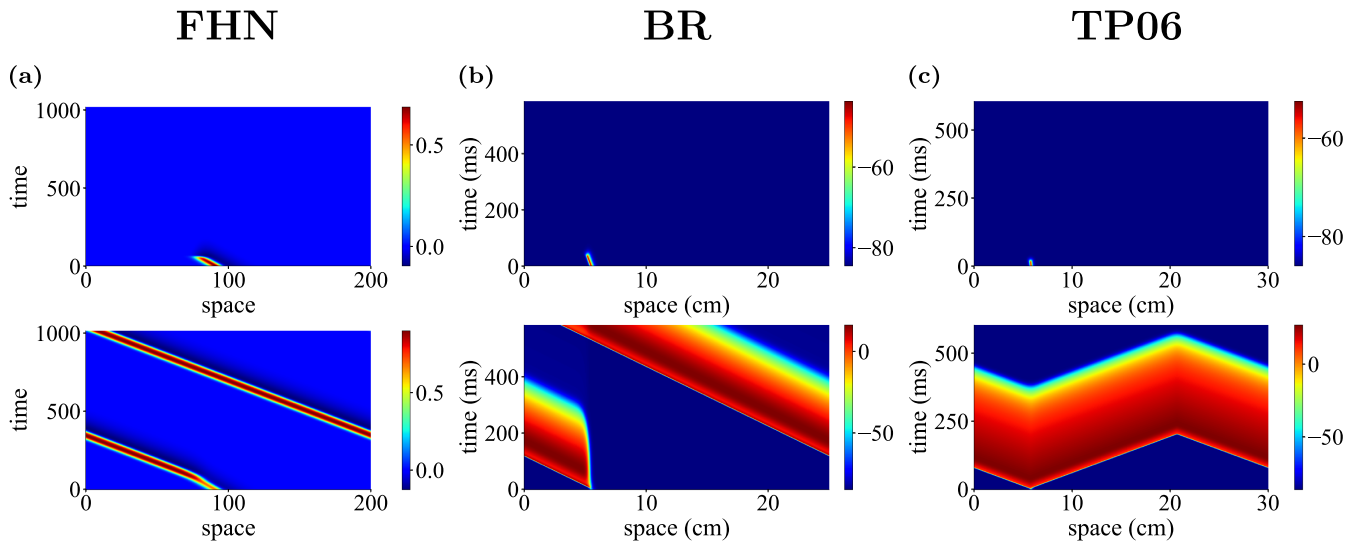


FIG. 7. Simulations run using TWIST to investigate threshold behavior of the slow waves. The initial conditions are a slow wave solution perturbed in the direction of the unstable eigenmode. The top row shows the result of negative perturbations; all propagating behavior rapidly decays to the rest state. Simulations in the bottom row are the result of a positive perturbation which leads to the sustained propagation of a fast wave in (a) and (b) and the emergence of oppositely propagating waves in (c). The spatial periods are (a) $\Lambda = 200$, (b) $\Lambda = 25$ cm, (c) $\Lambda = 30$ cm. The color bar units for (b) and (c) are mV.

is increased to $g_{Na} \approx 11.518 \text{ mS/cm}^2$, nearly three times its standard value ($g_{Na} = 4 \text{ mS/cm}^2$), suggesting that reflections are not supported under physiologically relevant conditions. In contrast, the TP06 model exhibits reflection-supporting behavior under its

default parameter set. Suppressing reflections requires reducing g_{Na} to an unphysical value ($g_{Na} \approx -0.4232 \text{ mS/cm}^2$), suggesting that the TP06 dynamics support wave reflection across physiologically relevant regimes.

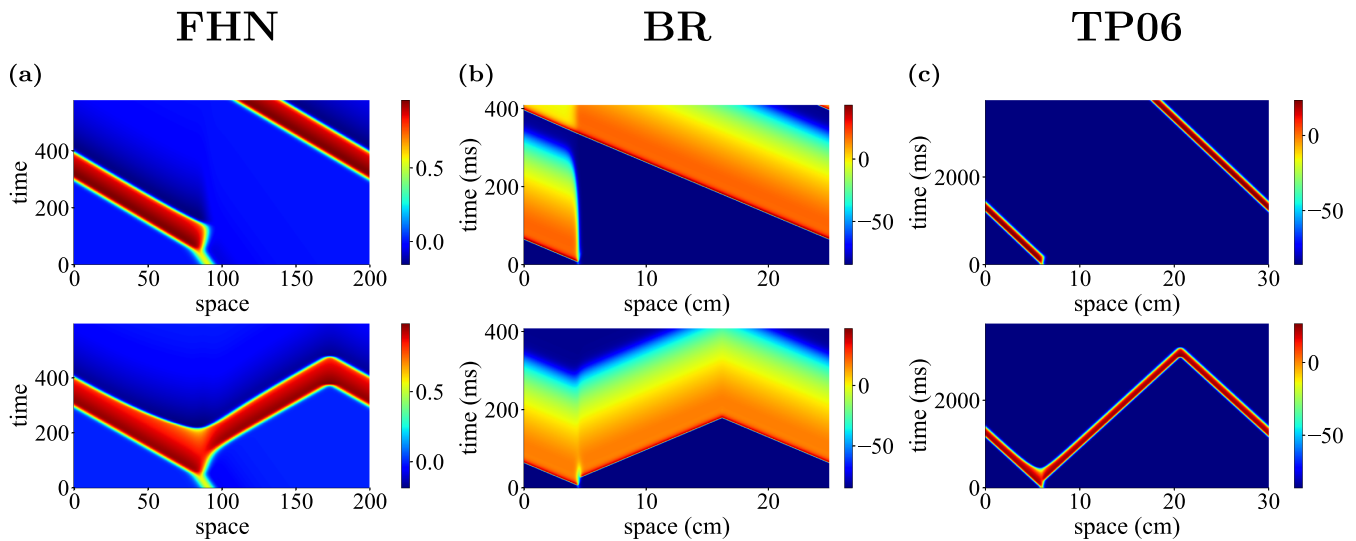


FIG. 8. Simulations of the slow waves near a possible global bifurcation associated with wave reflections. The top row shows the growth of the slow wave into a single propagating fast wave (supportive of no reflections) and whereas the development of counter-propagating fast wave in the bottom row provides evidence of the existence of wave reflection. (a) FHN model parameter values of (top) $\varepsilon = 0.0013$ and (bottom) $\varepsilon = 0.0012$. (b) BR model parameter values of (top) $g_{Na} = 11.518 \text{ mS/cm}^2$ and (bottom) $g_{Na} = 11.527 \text{ mS/cm}^2$. (c) TP06 model parameter values of (top) $g_{Na} = -0.4232 \text{ mS/cm}^2$ and (bottom) $g_{Na} = -0.3617$. The spatial periods are (a) $\Lambda = 200$, (b) $\Lambda = 25$ cm, (c) $\Lambda = 30$ cm. The color bar units for (b) and (c) are mV.

VII. DISCUSSION

In this study, we compare and contrast the behavior of propagating waves across a hierarchy of cardiac models and within models of similar complexity. Our central objective is to determine which features of traveling waves are robust across model classes and which arise from additional physiological detail. Our results demonstrate that several core organizing principles identified in minimal models persist in more detailed descriptions. In particular, all models considered here exhibit the canonical fast-slow wave structure, with propagation failure occurring through a saddle-node bifurcation and loss of stability of the fast wave through Hopf bifurcations. On the other hand, increasing physiological detail introduces substantial qualitative complexity into the structure of dispersion relations. While both the BR and TP06 models exhibit instabilities such as cascades of Hopf bifurcations and restitution-driven alternans,^{25,33,34} the TP06 model also displays a markedly richer structure, including multiple distinct branches of slow wave solutions that are absent in the FHN and BR models. These results highlight both the robustness of fundamental wave mechanisms and the emergence of qualitatively new behaviors arising from detailed ionic interactions.

A. TWIST: Traveling wave investigation software tool

Enabling this comparison required overcoming significant computational challenges associated with the analysis of traveling waves in biophysically detailed models. In particular, the high dimensionality, stiffness, and steep spatial gradients in conductance-based models make the systematic computation of dispersion relations and stability spectra difficult using existing tools. To address these limitations, we developed TWIST,⁶³ which integrates robust continuation methods, high-order discretization, and accurate stability analysis within a single framework. TWIST makes it possible to compute both stable and unstable solutions in models where such analyses were previously impractical. This capability is essential for uncovering the full structure of dispersion relations and enabling the cross-model comparisons presented in this work. We note that, although the development of TWIST was motivated by the specific challenges of biophysically detailed cardiac models, it is broadly applicable for the investigation traveling waves and complex spatiotemporal dynamics in reaction-diffusion systems.

B. Slow waves

One of the most striking results is the emergence of multiple slow-wave branches in the TP06 model. Unlike the single slow-wave branch observed in the FHN and BR models, the TP06 model supports several distinct families of unstable traveling waves with qualitatively different waveform morphologies. This increased complexity appears to arise from interactions between multiple excitation mechanisms, which differ across models. Both BR and TP06 include a fast sodium current and a calcium-mediated depolarizing current, but their relative timescales are markedly different. In the BR model, the slow inward calcium current I_s activates much more slowly than the sodium current and therefore contributes little to the rapid upstroke that drives wave propagation. In contrast, the TP06 model includes an L-type calcium current I_{CaL} with a rapidly activating component, allowing it to interact dynamically with the

sodium current during the upstroke and promote more complex wave structures.

Differences in repolarizing currents may further enhance the dispersion relation complexity. In the BR model, the time-dependent potassium current I_x evolves on relatively slow timescales (100–1000 ms) and primarily influences recovery. By contrast, the TP06 model includes a transient outward current I_{to} that activates rapidly enough to interact with both the sodium and calcium currents during the upstroke and thus could also be partially responsible for the richer bifurcation structure and the emergence of additional slow-wave branches observed in the TP06 model. Although the precise roles of I_{Na} , I_{CaL} , and I_{to} remain to be determined, our results suggest that fast secondary currents can qualitatively alter dispersion relations by enabling interactions between multiple excitation pathways.

C. Threshold dynamics and reflections

The importance of slow waves as structures that organize threshold dynamics for wave propagation is highlighted by our simulations. Across all models, the stable manifolds of slow waves act as separatrices that determine whether perturbations lead to propagation or decay, consistent with prior theoretical predictions.^{30,48} In our analysis of these threshold dynamics, we focused on the sodium-spike slow wave, as it possesses the dynamical structure expected of a threshold solution—a single unstable mode corresponding to a one-dimensional unstable manifold. This property makes it a natural candidate for organizing threshold behavior in the phase space of the system. Within this framework, we also assessed the ability of perturbations near this slow wave to generate wave reflection. The capacity for reflection differs across models. In the FHN and BR models, perturbations near the slow wave produce at most a single forward-propagating wave, whereas in the TP06 model, similar perturbations can generate counter-propagating waves. This implies that reflection-supporting dynamics, and thus the potential for reflection induced arrhythmias, are present in the TP06 model but not in the BR or FHN under default parameters.

The role of the additional, largely depolarized slow-wave branches observed in the TP06 model remains less clear. These solutions do not appear to have the same simple threshold structure, and their stability properties suggest higher-dimensional unstable manifolds. Preliminary simulations indicate that perturbations near these branches evolve into fast waves following a complex transient, but the dynamical significance of this behavior is not yet understood. Determining whether these solutions play a role in organizing wave dynamics, or whether they represent dynamically less relevant branches, is an important topic for future study.

D. Relation to previous work

Previous work on the existence and stability of action potentials on one-dimensional rings with varying circumference has focused primarily on the destabilization of fast waves and the onset of alternans (see, e.g., Refs. 6, 25, 26, 29, 31, 33, 34, and 45). Our findings are broadly consistent with and extend this body of work.

Early studies by Courtemanche *et al.* examined wave propagation in the Beeler-Reuter (BR) model on rings of decreasing circumference and observed the onset of alternans in numerical

simulations.^{25,26} Using analytical arguments based on dispersion relations and action potential duration (APD) restitution, they linked this instability to a Hopf bifurcation. Subsequent work extended these ideas to reduced and minimal models, where similar instabilities were found.^{28,29,34,45,70} In these settings, the onset of alternans was associated with either Hopf bifurcations in the point spectrum or, in spatially extended limits, Eckhaus-type instabilities arising from the essential spectrum.⁷⁰

Perhaps the closest study to the work presented here is that of Bauer *et al.*,³³ who used numerical continuation to compute dispersion relations and stability properties of periodic fast waves in a modified BR model.³³ This study provided further insight into the onset of alternans and its spectral origin.

The present work builds on and extends these results in several ways. First, we compute dispersion relations and stability spectra for both minimal and biophysically detailed cardiac models within a unified framework, enabling direct comparison across model classes. Second, in contrast to prior dispersion-relation studies of the BR model,³³ we explicitly compute and analyze the associated slow-wave branches and their role in threshold dynamics. Because our analysis is performed on a bounded ring, the instability manifests as a cascade of Hopf bifurcations associated with discrete modes, rather than a classical Eckhaus instability of the essential spectrum.

Finally, to our knowledge, this work presents the first systematic computation of the dispersion relation for the ten Tusscher–Panfilov (TP06) model, as well as the first detailed characterization of slow-wave branches in this setting. Notably, we observe that the TP06 model exhibits restabilization of the fast wave at small Λ following the onset of alternans [Fig. 2(c)]. It would be of interest to determine whether this restabilization is captured by classical APD restitution theory.

E. Limitations and scope

The results presented here are based on dispersion relations for traveling wave solutions, which represent a highly idealized description of wave propagation in cardiac tissue, even when biophysically detailed electrophysiological models are used. Our analysis is restricted to one-dimensional, homogeneous media with periodic boundary conditions, whereas real cardiac tissue exhibits spatial heterogeneity, anisotropy, curvature, and complex three-dimensional structure. However, traveling wave solutions and their associated bifurcation structure offer a systematic framework for characterizing the existence, stability, and transitions of propagating waves, and thus provide insight into the core dynamical mechanisms governing cardiac excitation. In this sense, the idealized framework used here complements more detailed simulations by isolating and revealing the structures that underlie complex spatiotemporal behavior.

F. Conclusions and future directions

Our results underscore the importance of systematically comparing wave dynamics both across model hierarchies and within the same level. Qualitatively similar behaviors observed in a wide variety of models suggest robust physiological mechanisms, whereas discrepancies indicate dynamics that arise from specific modeling assumptions. As shown here, traveling wave solutions and

bifurcations present in one model may be absent in another comparable model, with potentially significant implications for predictions of normal and pathological cardiac function.

The results presented here represent only an initial step in this direction; in many respects, they capture only a small portion of a much larger landscape of dynamical behaviors that remain to be explored. TWIST provides a framework for systematically exploring dispersion relations, stability, and bifurcation structure across a wide range of cardiac models and parameter regimes. Extending this approach to additional models and parameter regimes will help determine which features of wave dynamics are truly universal and which are model-specific.

Beyond model comparison, TWIST enables the investigation of physiologically and clinically relevant questions. For example, dispersion relations can be used to assess how disease states, such as long QT or Brugada syndromes, or pharmacological interventions alter the existence, stability, and bifurcation structure of propagating waves, and thereby influence the robustness of action potential propagation. By providing efficient access to both stable and unstable traveling wave solutions, TWIST opens new avenues for studying complex spatiotemporal dynamics in cardiac systems.

At present, TWIST's computations are limited to traveling waves of fixed spatial profiles and the creation of dispersion curves in one parameter. Notably, the investigation of time-dependent phenomena, such as the modulated waves in Fig. 4, is currently restricted to the use of TWIST's time integration capabilities and is, thus, an unreliable methodology for probing unstable spatiotemporal behaviors. However, such periodic in time behaviors can often be similarly framed as equilibria in an appropriate spatiotemporal frame, allowing for their robust computation.⁷¹ We aim to extend the current functionalities to include the computation of modulated waves as well as two-parameter bifurcation diagrams.

In summary, this work demonstrates that while minimal models capture essential features of wave propagation, biophysically detailed models can exhibit substantially richer dynamical structure due to interactions among multiple ionic processes. By enabling systematic computation and comparison of dispersion relations across model hierarchies, TWIST provides a framework for bridging the gap between simplified theoretical models and physiologically detailed descriptions, and for identifying the mechanisms underlying complex wave dynamics in cardiac tissue.

SUPPLEMENTARY MATERIAL

The [supplementary material](#) includes three movies illustrating the parameter-dependent evolution of traveling wave solutions and their associated stability spectra along the dispersion relations for each model considered. Supplementary Movie 1 shows the FitzHugh–Nagumo model, Supplementary Movie 2 shows the Beeler–Reuter model, and Supplementary Movie 3 shows the ten Tusscher–Panfilov (TP06) model. In each case, the movies display the traveling wave profile, the corresponding linear stability spectrum, and the location of the solution on the dispersion curve, providing a dynamic visualization of bifurcations and waveform transitions described in the main text. Detailed captions for each movie are provided in the [supplementary material](#).

AUTHOR DECLARATIONS

Conflict of Interest

The authors have no conflicts to disclose.

Author Contributions

Vincent Lovero: Data curation (equal); Formal analysis (equal); Investigation (equal); Methodology (equal); Software (equal); Validation (equal); Visualization (equal); Writing – original draft (equal); Writing – review & editing (equal). **Stephanie Dodson:** Conceptualization (equal); Formal analysis (equal); Investigation (equal); Methodology (equal); Supervision (equal); Validation (equal); Writing – original draft (equal); Writing – review & editing (equal). **Timothy J. Lewis:** Conceptualization (lead); Formal analysis (equal); Investigation (equal); Methodology (equal); Project administration (lead); Supervision (lead); Validation (equal); Writing – original draft (equal); Writing – review & editing (equal).

DATA AVAILABILITY

The data that support the findings of this study are available from the corresponding author upon reasonable request.

APPENDIX: VALIDATION AND PERFORMANCE BENCHMARKS

We verify the accuracy and efficiency of TWIST by comparing it against AUTO using three models of varying complexity and computational demand: FitzHugh–Nagumo (FHN), Hodgkin–Huxley (HH), and the nine-variable ten Tusscher–Panfilov (TP06) model. To assess computational efficiency, TWIST was benchmarked against AUTO using the three systems (Table I).

1. Benchmark methodology and hardware

The benchmarks presented in Table I were conducted on an Intel® Core™ i5-5257U processor with 8 GB of RAM. To assess consistency across hardware platforms, the benchmarks were also performed on an Intel® Xeon® CPU E5-2680 v3 with 128 GB of RAM and an Apple M5 CPU with 16 GB of RAM. Comparable performance speedups were observed across all systems.

TABLE I. Performance benchmarks comparing the computation of traveling waves and their stability in TWIST and AUTO. For the stability analysis benchmark, which is not natively supported by AUTO, the AUTO computation uses a finite-difference discretization and OpenBLAS, as described in the text.

Benchmark	TWIST(s)	AUTO(s)	Speedup
<i>Standard continuation</i>			
FHN (coarse)	1.0	1.3	1.3
TP06 (coarse)	3.3	12.2	3.7
TP06 (fine)	21.8	56.6	2.6
<i>Stability analysis (8-digit accuracy)</i>			
FHN	0.019	0.393	20.7
HH	0.136	Failed	∞
TP06	4.01	Failed	∞
<i>Stability analysis (10-digit accuracy)</i>			
FHN	0.039	0.490	12.6

2. Continuation and mesh specifications

For the continuation benchmarks, 1000 solutions were computed using six collocation points. The spatial domain was discretized using two different mesh resolutions: a coarse mesh with 35 grid points and a fine mesh with 230 grid points. The accuracy of TWIST is validated by comparing its dispersion curves against those generated by AUTO for the FHN and HH equations. The results were visually indistinguishable (Fig. 9) with relative differences of the order of 10^{-8} , which arise from the different strategies for mesh refinement, specifically mesh distribution and grid size, and continuation stepping. Note that stability is not included in Fig. 9. While both tools exhibit comparable performance for standard continuation (generation of the dispersion curve without stability) in low-dimensional systems, TWIST demonstrates a clear advantage for stiff biophysical models like the TP06 model, where it achieved speedups of approximately 2.6–3.7 times faster than AUTO (Table I).

3. Stability analysis differences

A key functional difference between TWIST and AUTO is that TWIST natively supports linear stability analysis of traveling

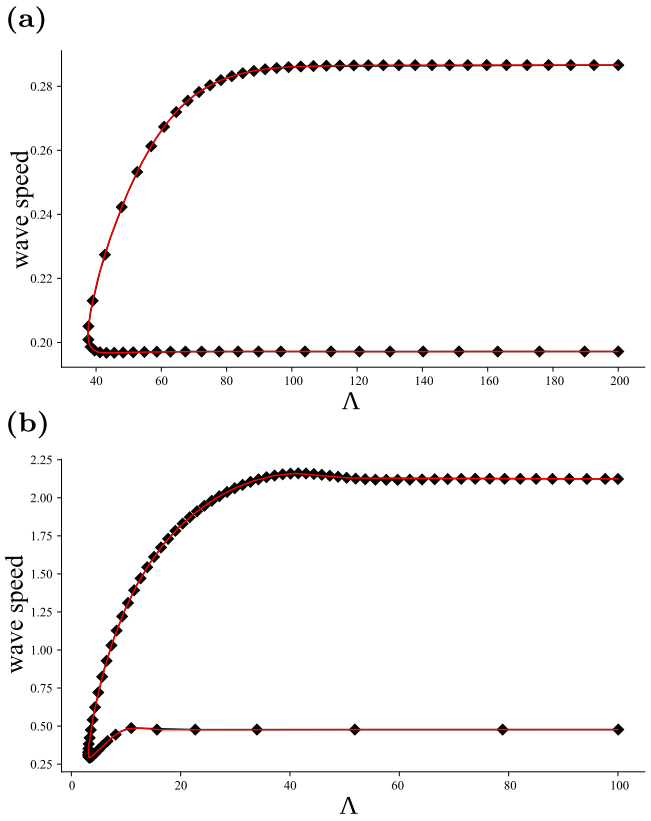


FIG. 9. Validation of TWIST against AUTO. Dispersion curves computed for the (a) FitzHugh–Nagumo equations and (b) Hodgkin–Huxley equations. Black diamonds correspond to points computed using TWIST. The red curve corresponds to points computed using AUTO. For both (a) and (b), spatial period is the continuation parameter.

waves, whereas AUTO does not. Consequently, for the stability analysis benchmarks in AUTO, it was necessary to approximate spatial derivatives using finite differences and use the eigenvalue routines provided by OpenBLAS on the resulting discrete operators. This is the standard approach for performing linear stability analysis in such a setting. Table I shows that achieving approximately eight digits of accuracy for the spectrum of a single traveling wave solution in the HH equations using finite difference matrices was either not possible (due to RAM limitations) or took about an entire day to run the necessary computations for one point along the dispersion curve in Fig. 9(b), of which there are many. However, when determining the stability to generate a complete dispersion curve with stability, the performance gap is more pronounced and TWIST is consistently orders of magnitude faster. Unlike the standard AUTO workflow, which requires the use of external tools and linear algebra libraries for stability analysis, TWIST computes full spectra with high accuracy in seconds for detailed ionic models where standard approaches often prove to be significantly slower or fail to converge.

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