May 5, 2026

Tenure Statement

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This statement covers §1 Research, §2 Teaching and §3 Service.

1 Research

My research lies at the interface of mathematical neuroscience and dynamical systems, with a focus on how neural systems generate, regulate, and adapt rhythmic activity across multiple levels of organization. I investigate how complex biological systems achieve both robustness and flexibility under perturbations, from the dynamics of individual neurons and circuits to interactions between neural activity, sensory feedback, and biomechanics. These questions arise naturally in the motor control of rhythmic behaviors such as respiration and feeding where neural circuits must maintain life-sustaining rhythmic outputs while remaining adaptable to modulation by chemical and behavioral cues.

To address these problems, I develop and apply analytic techniques from dynamical systems and bifurcation theory, along with numerical computation and data-driven modeling to uncover the underlying mathematical principles. In addition, I have made contributions to advancing dynamical systems theory beyond standard settings, such as extending geometric singular perturbation theory to systems with more than two timescales, phase reduction techniques to systems with hard boundaries, and the development of theory for coupled dynamical systems on fully inhomogeneous networks.

I will discuss four main components of my research program, with details and connections to future directions provided in the corresponding subsections.

§1.1: *Multiple-Timescale Neural Dynamics.* Biological rhythms typically evolve across disparate timescales. My work extends standard fast-slow decomposition and geometric singular perturbation theory to systems with three or more timescales, uncovering mechanisms underlying complex activity patterns in multiple-timescale systems and identifying solution properties that intrinsically require three timescales.

§1.2: *Modulation of Neural Networks.* A central focus of my recent work is understanding how neurmodulators such as norepinephrine reconfigure respiratory circuits to enable robust yet flexible control of breathing under perturbations such as opioid exposure. Through close collaboration with experimental researchers, my work seeks to develop an integrated framework that combines dynamical systems modeling and analysis with data-driven approaches.

§1.3: *Closed-loop Neuromechanical Control.* At the system level, motor systems achieve remarkable robustness through closed-loop control, integrating nonlinear interactions among neural circuits, biomechanics and sensory feedback. My research dissects this robustness against perturbations, a fundamental challenge in motor control, and develops novel mathematical tools for analyzing rhythmic motor behaviors, with applications to feeding in *Aplysia*.

§1.4: *Networks and Coupled Oscillator Systems.* I seek to disentangle model-dependent and model-independent behaviors in networks of dynamical systems, with a focus on how network structure constraints generic dynamics and how specific modeling forms impose further restrictions. My work spans two complementary directions: 1) *fully inhomogeneous networks*

with distinct nodes and arrows, where I use singularity theory and combinatorial matrix theory to characterize how network structure influences dynamics independent of model equations; 2) synchrony and symmetry breaking in *homogeneous networks* with identical nodes and identical arrows.

Highlights of my research accomplishments:

- My scholarly work described in this statement appears across 24 research articles (17 peer-reviewed, 2 book chapters, 1 conference proceeding, and 4 preprints).
- I have secured external funding of $1,773,230 as single PI (NIH R01 DA057767), and continue to actively pursue external funding with 3 NSF and 1 NIH proposals submitted in 2025.
- As of May 1, 2026, I have given 49 invited talks in seminars, colloquia and conferences across the United States and internationally, including one plenary lecture.

1.1 Multiple-Timescale Neural Dynamics

Mixed bursting dynamics in three-timescale respiratory neuron models. Breathing is a critical rhythmic behavior that continues from birth up until death. It is generated by neural networks, including the pre-Bötzinger complex (pre-BötC) [64] which has been established to be the inspiratory central pattern generators (CPGs). Within this network, certain neurons have *pacemaker* properties and can drive a rhythmic network activity without receiving a patterned synaptic input from another source. While pre-BötC pacemaker dynamics are well understood using *geometric singular perturbation theory* (GSPT) [24, 62], this framework fails to capture more complex respiratory patterns, such as sigh breaths [39, 78], immature breathing rhythms (Fig. 1A) [10, 79], or neuromodulation-induced irregular activity prior to stabilization, as revealed in our recent work [70]. These patterns involve interactions across at least three timescales and require analysis beyond the standard fast-slow decomposition.

My doctoral research [50, 80] addressed this gap by extending GSPT, originally developed for two-timescale systems, to three-timescale problems of the form

$$\begin{aligned} \varepsilon\dot{x} &= f(x, y, z, \varepsilon, \delta) \\ \dot{y} &= g(x, y, z, \varepsilon, \delta) \\ \dot{z} &= \delta\, h(x, y, z, \varepsilon, \delta) \end{aligned}$$

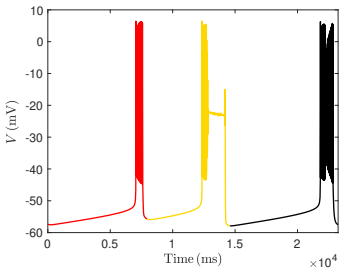

Figure 1: Mixed bursting in prenatal pre-BötC neurons [79].

where $0 < \varepsilon, \delta \ll 1$, and x, y, and z represent the fast, slow and superslow variables. I applied the extended GSPT to explain irregular bursting dynamics in biophysical respiratory neuron models [77, 78]. In particular, in [78], I investigated two competing hypothesis for sigh generation [39, 68] and identified ways to distinguish them in experimental studies. I have since extended this research program in the following three directions.

(a) Prenatal respiratory dynamics and developmental changes. While postnatal pre-BötC rhythmogenesis has been extensively studied, much less is known about the inspiratory network during embryonic development. In [79], I provided the first GSPT and bifurcation analysis of

mixed bursting dynamics in embryonic pre-BötC neurons (Fig. 1). This analysis yielded predictions for how developmental changes in the balance of two key burst-generating current conductances (g_{NaP} and g_{CAN}) shape intrinsic bursting activity. With my PhD student M. Brooke-deBock and collaborator J. Rubin (UPitt), we are now developing a network model of embryonic pre-BötC neurons to determine whether these changes arise at the single-cell or network level. Preliminary results suggest that they vary at the single-cell level, rather than through discrete switching, to account for the transient emergence of mixed bursting neurons prior to birth. Network-level analysis further indicates that additional intrinsic mechanisms must co-evolve over development to ensure both correct intrinsic dynamics and appropriate activity patterns when embedded within a heterogeneous network.

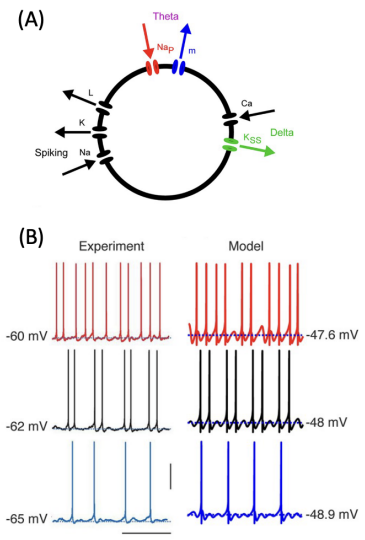

Figure 2: Diagram of single-cell theta oscillator and simulation results [59].

(b) Phase-locking in multiple-timescale neural oscillators. Speech is another multiple-timescale phenomenon. Cortical theta (∼3-9 Hz) oscillations are believed to play an essential role in syllabic segmentation by synchronizing neural rhythms to external acoustic features of the speech stream [28–30]. To support this function, these oscillators must flexibly phase-lock (i.e., synchronize the timing of their outputs) to rhythmic inputs spanning a wide range of timescales, including rhythms substantially slower than their intrinsic frequency. Using biophysical models, we found that phase-locking flexibility depends critically on the types of hyperpolarizing currents pacing the oscillators [59]. In particular, the greatest flexibility arose from a synergistic interaction between a slow m-current and a superslow calcium-activated potassium current (Fig. 2A), a synergy not replicated by synaptic inhibition at similar timescales.

A distinctive feature of our model is the emergence of mixed-mode oscillations (MMOs) in response to tonic input, characterized by alternating small- and large-amplitude oscillations, consistent with experimental recordings (Fig. 2B). Using dynamical systems tools, we show that interactions between slow and superslow inhibitory processes create a three-timescale structure in the full model that generates pronounced post-input recovery delays via a delayed Andronov-Hopf bifurcation (DHB), thereby substantially expanding the frequency range over which entrainment can occur [76]. Notably, the DHB plays little role in the unforced oscillatory dynamics, but is recruited to support phase locking under external strong forcing. Moreover, the intermediate-timescale m-current further enhances flexibility by prolonging delayed recovery and enabling entrainment to even slower inputs (lower than 1 Hz). Together, these results highlight the importance of intrinsic multiscale interactions for flexible phase locking in neural oscillators and motivate my further investigation of MMO-type dynamics in systems with more than two timescales.

(c) Mixed-mode bursting oscillations in three-timescale neural oscillators MMOs have been recognized in many branches of sciences including including physics, chemistry, and particularly life sciences (e.g., [2, 6, 38, 40, 42, 54, 72]). Compared with the extensively studied MMOs in two-timescale problems [13], which arises either from a canard mechanism associated with folded

node singularities or a delayed Hopf bifurcation (DHB) of the fast subsystem, the theory of MMOs in three-timescale settings remains less well developed, despite that many real-world systems have more than two timescales. With my former PhD student N.A. Phan (now postdoc in my group), we analyzed the geometric mechanisms underlying MMOs in three-timescale systems by considering a model of coupled Morris–Lecar neurons [56], with a particular focus on a novel singularity known as canard-delayed-Hopf (CDH) [44]. This singularity, unique to systems with more than two timescales, enables the canard and DHB mechanisms that remain separate in two-timescale systems to coexist and interact. Our analysis reveals that MMOs supported by CDH exhibit significantly greater robustness than those in its absence. We also demonstrated that the mere presence of CDH does not guarantee the occurrence of MMOs. These results yield important insights into the conditions under which canard dynamics and DHB interact to produce more robust MMOs in three-timescale systems.

MMO and bursting dynamics can combine to produce mixed-mode bursting oscillations (MMBOs) (Fig. 2B), which are analogous to MMOs but with the large-amplitude oscillations (LAOs) organized into bursts. Relatively few works have investigated MMBOs [14, 15, 31, 41, 63], most of which focus on simplified two-timescale models (e.g., polynomial vector field or firing-rate models). First described in [14], MMBOs were shown to be organized by folded-node canards. Similar MMBO-type solutions have been observed in a 4D conductance-based bursting model [6], where small-amplitude oscillations (SAOs) arise from a fast subsystem DHB. With postdoc N.A. Phan, we apply the extended GSPT framework [50, 56] to analyze MMBOs in an 8D three-timescale biophysical neuronal model [59] (Fig. 2). Combining GSPT with full-system bifurcation analysis, we identify the mechanisms underlying MMBOs and show that their bifurcation structure is organized in families of isolas (Fig. 3). Our results reveal additional complexity in MMBO isola structure and the underlying mechanisms due to the presence of three distinct timescales, compared to two-timescale systems [41]. In particular, the presence of a CDH singularity enables DHB and folded-node canards to jointly organize SAOs and spike-adding cascades during the transition from MMOs to MMBOs, either along a single isola or across different isolas. A manuscript on these results is in preparation [55].

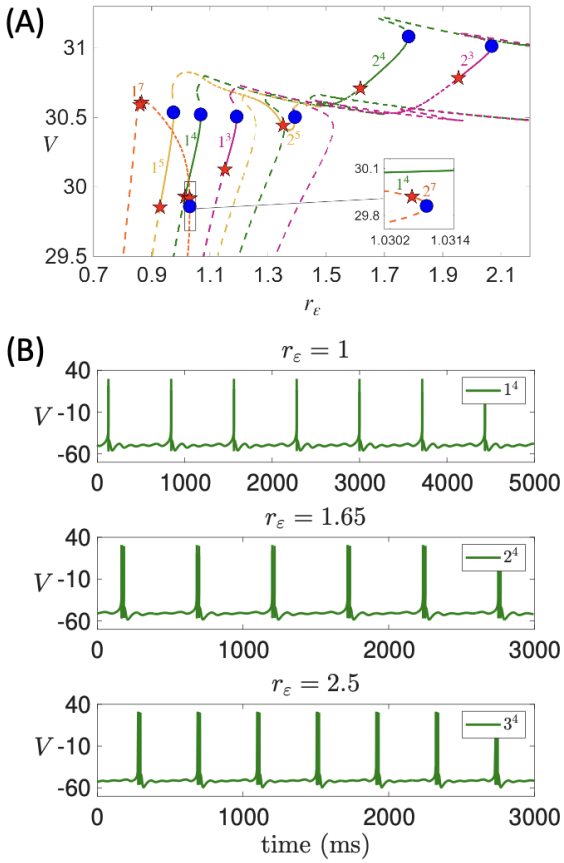

Figure 3: (A) Partial bifurcation diagram of the three-timescale theta model (Fig. 2), showing representative isolas of MMBO solutions. Increasing r_ε accelerates the fast variables. Each stable segment (solid) is bounded by a period doubling bifurcation (red star) and a saddle-node bifurcation of periodic orbits (blue circle), and denotes rhythms l^s with s SAOs and l LAOs. (B) Example MMBO periodic solutions undergoing spike-adding transitions along the green isola in (A), which is formed by orbits with 4 SAOs.

1.2 Modulation of Neural Networks

A fundamental challenge in the study of complex adaptive systems is understanding how biological networks maintain robustness despite internal noise or environmental perturbations, while preserving the flexibility to transition between distinct functional states. The pre-Bötzinger Complex (pre-BötC), a heterogeneous neural network driving the inspiratory rhythm, provides an ideal system to study this tradeoff, as it must maintain a life-sustaining rhythm while allowing for rapid, state-dependent modulation by neuromodulatory inputs.

The pre-BötC is constantly modulated by numerous neuromodulators through altering properties of neurons, synapses and networks [16, 17, 67, 71]. The role of neuromodulation depends on physiological context. For instance, norepinephrine (NE) can stabilize inspiratory activity in healthy networks [71], yet destabilize inspiratory activity under pathological conditions [84]. Furthermore, NE can act as a homeostatic buffer against inhibitory challenges such as opioid induced respiratory depression (OIRD) [5, 8, 12]. In collaboration with experimentalists A. Garcia and A. Tryba (University of Chicago), my research group integrates electrophysiological experiments and computational modeling across the levels of neurons and networks to uncover the mechanisms underlying effect of neuromodulation on respiratory control, with a focus on NE and opioids.

(a) Intrinsic mechanisms of noradrenergic modulation. Whether NE exerts stabilizing versus destabilizing effects on inspiratory rhythmogenesis likely depends on its heterogeneous actions on distinct neuronal subtypes within the preBötC [71]. With my PhD student S. Venkatakrishnan, we examined these differential effects by modeling NE modulation through two key parameters: the calcium-activated nonspecific cationic current conductance (g_{CAN}) and inositol-triphosphate (IP_3) [70]. Our model reproduces experimentally observed subtype-specific effects of NE modulation on pre-BötC neurons and reveals that this dual-modulation mechanism is essential for inducing conditional bursting in neurons that are otherwise tonic spiking.

(b) Intrinsic heterogeneity shapes modulatory flexibility. At the network level, my recent work with my PhD student M. Brooke-deBock shows that the structure of intrinsic heterogeneity critically determines a network's modulatory responses ([7], manuscript in preparation). By comparing *uniform* versus *bimodal* networks, two common representations in which intrinsic parameters are distributed either uniformly or across distinct subpopulations [9, 39, 57, 58], we find that modulatory flexibility requires a clear separation of roles between pacemakers (intrinsic bursting neurons) and followers (silent neurons).

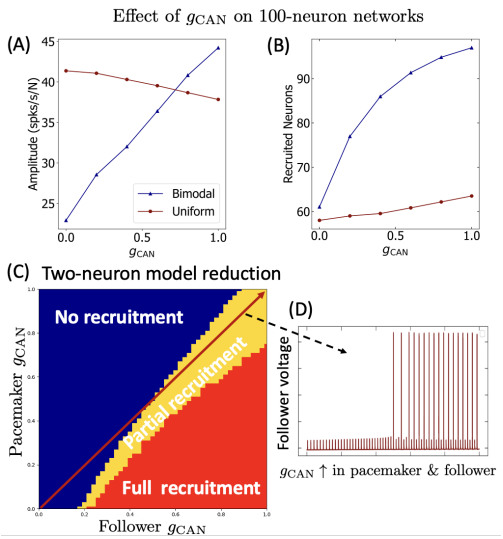

Figure 4: (A,B): The same g_{CAN} modulation produces opposite effects on network amplitude and follower recruitment, depending on the organization of intrinsic heterogeneity. (C) Phase diagram of a reduced two-neuron model showing follower recruitment as a function of g_{CAN} in pacemaker versus follower neuron. Increasing g_{CAN} along the red diagonal line moves the system from no recruitment to partial recruitment (voltage trace shown in (D)).

As illustrated in Fig. 4A & B, identical g_{CAN} modulation drives a strong increase in population amplitude and recruitment in bimodal networks, while uniform networks remains largely unresponsive. We show,

via a two-neuron motif reduction and bifurcation analysis, that this difference arises from opposing effects across subpopulations: increasing g_{CAN} suppresses recruitment in pacemakers but promotes it in followers (Fig. 4C). As a result, these competing effects cancel in uniform networks, whereas bimodal networks maintain a distinct pacemaker-follower structure that supports flexible modulation. These results identify key network features that governs the tradeoff between modulatory robustness and flexibility in the pre-BötC.

(c) NE induces a NMDA-dependent network state of respiratory rhythmogenesis. Using a combination of electrophysiological recordings and computational modeling [69], we also identified a previously undescribed synaptic mechanism by which noradrenergic modulation selectively recruits NMDA receptors (NMDARs) without altering other synaptic activity. I led the modeling and network analysis components of this work, showing that synaptic calcium entry via NDMARs plays a key role in maintaining network synchrony during NE-induced elevated intrinsic excitability, consistent with multielectrode array (MEA) recordings from my collaborator Dr. Garcia's lab.

With postdoc N. Sutton and Ph.D. student S. Venkatakrishnan, we are now integrating NE-induced cell-specific effects on intrinsic properties with NMDA receptor recruitment to understand how these mechanisms interact to shape network-level effects under both healthy and disease conditions. We are also investigating how NE interacts with opioid modulation to impact rhythmic network assembly and synchrony. These modeling efforts are closely guided and validated by experimental recordings from the preBötC [69]. Ultimately, we aim to understand how NE modulates inspiratory rhythmogenesis and mitigates opioid-induced respiratory depression, providing a theoretical framework for understanding and treating clinical respiratory instability.

In the longer term, my research on the modulation of neural networks will focus on three main directions:

- **Data-driven approaches to infer neuromodulatory mechanisms:** We recently developed a physics-informed neural network framework for the joint inference of hidden state variables and unknown parameters in multiple-timescale biophysical neuronal models [82], and are now extending it to infer biophysical parameters from experimental data. This approach represents a promising future direction for probing neuromodulatory mechanisms from membrane potential recordings, complementing our ongoing mechanistic modeling efforts.

- **Computational modeling of homeostatic regulation in opioid use disorder** (NSF CRCNS proposal under review; Lead PI, with PI A. Garcia at UChicago): Many biological systems maintain function despite persistent challenges, suggesting homeostatic mechanisms operating over longer timescales. Motivated by experimental evidence of cryptic states in respiratory networks [61], where network function appears normal despite parameter reconfiguration but responds differently to subsequent perturbations [1], we aim to investigate how the preBötC adapts to repeated opioid exposure and to characterize the mechanisms underlying the cryptic states. With postdoc N.A. Phan and undergraduate Z. Lin, we have adapted activity-dependent homeostatic regulation models [46, 53] to single respiratory neurons, capturing key features of recovery dynamics and tolerance under repeated challenges, as observed experimentally [52]. We will extend this framework to network-level dynamics, with the goal of uncovering principles governing the homeostatic stabilization of a survival-critical neuronal network.

- **Closed-loop respiratory control** (NITMB Focused Research Group Award): Building on my work on respiratory networks, I plan to study respiratory control as a closed-loop system coupling neural circuits, biomechanics and sensory feedback. As part of a recently funded collaborative NITMB Focused Research Group, we will build on existing respiratory rhythm generation and feedback models to develop a comprehensive respiratory control model and to understand how different key components interact together to produce stable, natural breathing that is robust to external perturbations.

1.3 Closed-loop neuromechanical control

To survive and reproduce, an animal must adjust to changes in its internal state and the external environment. Motor systems show remarkable robustness through *closed-loop* brain-body interactions among central neural circuitry, biomechanics, and sensory feedback [11]. Understanding the mechanisms underlying this robustness remains a fundamental challenge in motor control. We address this question using a closed-loop neuromechanical model of motor patterns for feeding in the *Aplysia californica*, which also motivates the development of new mathematical techniques.

(a) **Development of new mathematical techniques for studying motor robustness.** In many rhythmic systems, robustness involves response of both the shape and the timing of the periodic neuromechanical trajectory. While classical techniques such as the *infinitesimal phase response curve* (iPRC) and variational analysis have been widely used to study robustness, each has important limitations. To address these, in [74], we (1) characterized for the first time the iPRC for nonsmooth systems with hard limits; (2) developed the *infinitesimal shape response curve* to describe the shape changes under sustained perturbations; and (3) developed the *local timing response curve* to measure timing sensitivity of a limit cycle within specific regions. We demonstrated the utility of these tools in coupled stick-slip oscillators [74] and a biologically inspired robotic system [25].

(b) **Motor Robustness in *Aplysia*.** Our methods provide a general framework for understanding neural control of robust motor behaviors. As a concrete example, we focused on a closed-loop neuromechanical model of rhythmic feeding behaviors of the marine mollusk, *Aplysia californica*, incorporating central pattern generators, biomechanics and sensory feedback [47]. The periodic orbits corresponding to the swallowing behavior make and break contact with firing rate hard boundaries. In [81], we applied methods developed in [74] to dissect robustness of this neuromechanical model against external mechanical perturbations. Interestingly, we discovered that biomechanics played an essential role in robustness by producing an immediate resistance to overcome the perturbation, whereas the sensory feedback effect is significantly delayed by the hard boundary properties. These findings refine and expand a previous hypothesis that sensory feedback is the key mechanism underlying motor robustness by revealing a previously underappreciated role of biomechanics.

In the future, I plan to extend this theoretical framework [74, 81] to study how changes in neuronal activity and sensory feedback influence robustness and flexibility in feeding motor control, and to investigate other biological rhythms including breathing (as part of the NITMB FRG effort described in §1.2), and, more broadly, circadian rhythms and the cell cycle.

(c) **Energetic efficiency and behavioral adaptation to environmental variability.** In [83], we incorporated muscle energetics and neuromodulation into the neuromechanical model

of *Aplysia californica* feeding. We showed that an optimal intermediate level of neuromodulation emerged from physiological constraints on serotonergic modulation trajectories, highlighting a trade-off between performance and energy cost and suggesting experimentally testable hypotheses on neuromodulatory control. Building on this, we are developing a framework that combines a behavioral model of *Aplysia californica*, based on Markov decision processes and reinforcement learning [60, 65, 66], with controlled behavioral experiments to study how *Aplysia* allocate energy across competing behaviors such as feeding, locomotion, and rest under changing environmental conditions. The goal is to understand how animals adapt behavior under environmental uncertainty. We are preparing an NSF IOS Behavioral Systems proposal in collaboration with H. Chiel (CWRU Biology), A. Susswein (Bar-Ilan Biology), P. Thomas (CWRU Math) and Z. Yu (CMU Math).

1.4 Networks and Coupled Oscillator Systems

Networks of coupled dynamical systems arise in many branches of science, where a central question is how network structure influences the generic network dynamics and bifurcations. At the same time, the form of a chosen model (e.g., symmetry, types of coupling) can impose further restrictions. My research addresses both aspects.

First, I study *fully inhomogeneous networks*, where all nodes and arrows have distinct types, with an emphasis on identifying the role of network architecture in shaping generic dynamics. Much of the theoretical work in this area focuses on networks with additional structure or assumptions (e.g., feedforward architecture, homogeneity, symmetry) or on networks of relatively small size. My research instead develops results for arbitrarily large fully inhomogeneous networks. Second, I study symmetric networks of coupled identical oscillators, focusing on *strong symmetry-breaking*, a new type of symmetry breaking occurring far away from any symmetric state, and how symmetry-predicted patterns deform when systems deviate from ideal symmetry.

1.4.1 Fully inhomogeneous networks

(a) **Role of network structure on bifurcations.** Using center manifold reduction and singularity theory, my collaborators and I have analyzed and classified bifurcations in fully inhomogeneous networks [26]. We proved that generic codimension-1 bifurcations are saddle-node and standard Hopf, e.g.,

Theorem ([26]). *Generically, codimension-1 Hopf bifurcation on a fully inhomogeneous network is nondegenerate. It yields periodic motion in nodes in the path component associated with critical eigenvalues and all nodes downstream. The amplitude of this periodic motion has a growth rate* $\sqrt{|\lambda|}$*, while all other nodes remain constant and experience at most* $|\lambda|$ *growth.*

In contrast, we discovered that generic codimension-2 bifurcations with network structure differ significantly from those in a general dynamical system. Our results make it possible to predict what patterns of dynamic behaviors can arise based solely on network architecture, even in arbitrarily large networks.

(b) **Role of network structure on homeostasis.** Homeostasis is an important biological phenomenon whereby the output of a system (say, body temperature) is approximately constant despite changes of an input (say, ambient temperature). Adaptation is a closely related notion

that has been widely used in synthetic biology and control engineering [48]. A particularly fruitful approach to understand the mechanisms leading to homeostasis is to regard homeostasis as a network concept. Using singularity theory coupled with graph-theoretic ideas from comminatory matrix theory, we developed a theoretical framework for classifying *homeostasis types* in arbitrarily large input-output networks with a single input node and a single output node [32, 35, 75]. One striking outcome of [75] is that, without specifying any homeostasis-generating mechanisms at the outset, we find a posteriori that there are essentially only two types:

Theorem ([75]). *In an input-output network with a distinguished input node and a distinguished output node, the homeostasis mechanism is either* structural *(generalized feedforward) or* appendage *(generalized feedback).*

To make this framework accessible without requiring expertise in graph-theoretic concepts, I worked with undergraduate student X. Lin to develop a Python-based algorithm [45] that identifies homeostasis motifs and conditions directly from network topology. By introducing an augmented representation that maps multiple inputs to an equivalent single-input form, I extended the theory to multi-input networks. This representation has inspired several extensions, including networks with multiple outputs and systems with conservation laws. I am also leading an effort to develop algorithms for enumerating *homeostasis patterns* [21] - a set of nodes that are simultaneously infinitesimally homeostatic. Our framework links homeostasis mechanisms directly to network structure and lays the groundwork for using singularity theory to study higher codimension homeostasis singularities.

1.4.2 Symmetry breaking in networks of coupled identical oscillators

Symmetry breaking occurs when a system has certain symmetries, but its solutions do not necessarily share them. Recently, a new type of symmetry-breaking, called *strong symmetry-breaking* (SSB), was discovered in minimal chimera systems [2–4, 23], where the coupled identical oscillators exhibit substantially different amplitudes, frequencies, or other properties. Below I describe my recent work on symmetry breaking in networks of coupled identical oscillators.

(a) Inspired by efforts to understand the latching-gate mechanism that governs the strict "one-way" transitions between G1 (unreplicated chromosomes) and S-G2-M (replication and partitioning of chromosomes) phases in the eukaryotic cell cycle [18, 51], P. Gandhi (VCU) and I recently developed a simplified conceptual model in which normal cell cycling is modeled by alternating, large-amplitude oscillations of the two identical oscillators which is induced by inhibitory coupling [27]. We identified a strong symmetry-breaking (SSB) homoclinic bifurcation underlying the transition from normal cell cycle oscillations to endocycles [22, 49], in which one oscillator exhibits large-amplitude oscillations while the other exhibits small-amplitude oscillations.

(b) With T. Vo (Monash University, Australia), we discovered a distinct mechanism for SSB in symmetrically coupled identical slow/fast oscillator networks ([73], manuscript in preparation). We showed that the SSB, surprisingly, originates from the canard dynamics of a folded node that lies on the axis of symmetry. By applying GSPT and the blow-up technique [20, 43]

to a normal form, we determined the geometric mechanisms by which the *symmetric folded node* induces symmetry breaking. We further demonstrate our results in a biological model of the eukaryotic cell cycle [19].

(c) SSB has often been associated with folded nodes located off the symmetry axis [3]. In contrast, our work identifies two distinct mechanisms arising, respectively, from the symmetric equilibrium [27] and symmetric folded nodes [73]. Moving forward, I aim to understand which features of the underlying model equations give rise to these different mechanisms despite sharing identical network structure, and to investigate whether a higher-order organizing singularity may underlie them. From a biological perspective, it is important to understand how symmetry-breaking mechanisms persist when oscillators or their couplings are no longer identical. I will investigate the robustness of the SSB mechanisms, with cell cycle biology as an application system. This direction also connects to the following project.

(d) **Symmetry-breaking control of network dynamics** (NSF proposal under review; Co-PI, with PI J. Touboul (Brandeis Math) and Co-PI S. Fraden (Brandeis Physics)) Symmetry provides a powerful organizing principle for understanding pattern formation in coupled oscillator networks, as equivariant dynamical systems theory enables theoretical predictions of spatiotemporal phase patterns [33, 34, 36, 37]. Yet, natural systems are never perfectly symmetric. J. Touboul and I lead the theoretical component of this project, focusing on how symmetry breaking via heterogeneity or noise alters symmetry-predicted patterns and how asymmetries can be used to control network dynamics. Our analysis reveals a hierarchy in the robustness of these patterns to heterogeneity and shows how structured asymmetry selectively destabilizes some patterns while preserving others.

2 Teaching

At the undergraduate level, I have taught Math 37A: Differential Equations, Math 40A: Topics in Applied Mathematics, and Math 123A: Principles of Mathematical Modeling. My overall instructor ratings for these courses were: 4.51/5 for Math 37A; 4.71/5 and 4.08/5 for Math 40A; and 4.83/5 and 4.4/5 for Math 123A. At the graduate level, I have taught Math 238A: Topics in Applied Mathematics. Due to the small class size, course evaluations were not generated. Prior to joining Brandeis, I taught graduate-level courses Math 5600: Dynamical Systems and Math 5750: Mathematical Biology at the University of Iowa, with instructor ratings of 5.55/6, 5.5/6, and 5.9/6. A full list of courses I have taught is provided in my CV.

In my teaching, I strive to present complex concepts in a clear and accessible way, helping students see how mathematical ideas connect and build on one another. Recognizing that students do not always learn effectively by watching an instructor solve problems, I incorporate in-class problem solving, where students work through examples in small groups and present solutions at the board. I also regularly pause to check for understanding and encourage questions, fostering a classroom environment in which students become increasingly comfortable participating in discussions. Together, these approaches have improved both participation and learning. Students say:

- *This class was so amazing and I feel like I learned a lot. Professor Wang makes what seems confusing a lot easier to understand!*

- *She's so organized and her lectures don't ramble at all. Each section really builds on the previous one which helps you learn the logic/process.*
- *As the semester went on, I felt that it became much easier for any of the students to ask questions and I thought you did a great job of clarifying when questions were brought up.*

I truly believe that connecting mathematics to real-world applications is a great way to motivate student learning and interest, especially in applied math courses. To support this, I often replace traditional exams with in-class presentations or final projects to give students opportunities to apply what they have learned to real-world problems. In a math modeling class (Math 123A), I also encourage students to replace the final project with participation in the Mathematical Contest in Modeling (MCM) and Interdisciplinary Contest in Modeling (ICM), where they tackle an open-ended, real-world problem within 48 hours. One team earned both a Finalist award and the Mathematical Association of America (MAA) award.

My role as a teacher extends beyond the classroom: I dedicate office hours, emails, and online communications to resolving students' questions patiently and promptly. I greatly value student feedback and actively adapt my teaching in response. For example, after receiving critical feedback on course evaluations about confusion surrounding a newly added participation grade, I clarified course expectations by consistently reinforcing policies in subsequent classes. Furthermore, I conduct midterm surveys and follow up with students after class or during office hours to gather feedback beyond the final course evaluation. I then make adjustments based on these feedback. Students have noted:

- *I really appreciate how Dr. Wang was open to our suggestions throughout the semester and made adjustments based on our requests.*
- *I feel that Dr. Wang did a great job presenting the course material in a very logical and clear manner. I greatly appreciated that she did so many examples during her lectures and was very willing to answer any questions we had. She is a wonderful professor!*
- *YangYang is a great professor–always prepared, enthusiastic, and helpful outside of class. She is helpful with tips of what to study before quizzes and exams, and she seems to really care about her students.*

In addition to teaching, I also contribute to curriculum innovation. In response to the lack of mathematics courses fulfilling the university's oral communication (OC) requirement, I revised the *Math 123a* syllabus to meet OC standards, and the course was subsequently approved for this designation. I have developed a graduate-level topic course on network science and graph theory, designed to attract students across pure and applied math, as well as other science disciplines. This course brought together students and postdocs from mathematics, physics and neuroscience. Previously, at the University of Iowa, I co-developed new courses in mathematical biology at both undergraduate and graduate levels, and I look forward to continuing to expand the applied mathematics curriculum at Brandeis.

2.1 Mentoring

Highlights in mentoring:

- Undergraduate trainee X. Lin received the Brandeis Science Summer Undergraduate Research Fellowship (SURF); this project resulted in a manuscript with undergraduate first author [45].
- PhD student Ngoc Anh Phan received the Bor-Luh Lin Award for Outstanding Thesis in Mathematics.
- Ph.D. students received 8 external travel awards for attending conferences, including 4 SIAM Student Travel Awards.
- Letters of recommendation written for 15 Brandeis undergraduates.

A full list of my undergraduate, graduate and postdoctoral trainees, and their research topics, is provided in my CV.

Undergraduate Mentoring I have mentored 9 undergraduate students from diverse academic backgrounds, including Computer Science, Economics, Neuroscience and Mathematics. One undergraduate trainee was awarded the Summer Undergraduate Research Fellowship (SURF) for work on homeostasis structure in input-output networks [45]. Besides advising undergraduate research, I also support students in pursuing their goals through letters of recommendations. Since arriving at Brandeis, I have written letters for 15 undergraduate students.

Ph.D. students and Postdocs I have advised several Ph.D. students and postdoctoral scholars, and am deeply committed to their professional development. I have developed a mentoring framework that emphasizes scientific training, communication, leadership, and career planning. I meet with trainees regularly (typically weekly), and organize joint lab meetings with experimental collaborators.

Previously, at The University of Iowa, I advised postdoctoral scholar H. Mofidi (now assistant professor at BIMSA, Tsinghua University) and Ph.D. student N.A. Phan (Ph.D., May 2024), who received the Bor-Luh Lin Award for Outstanding Thesis in Mathematics. She is currently a research associate in my group. In addition, I supervise two Ph.D. students at Brandeis (S. Venkatakrishnan in Mathematics, M. Brooke-deBock in Physics), co-advise one Ph.D. student at UIowa (V. Prudencio, with L. Wang), and mentor a postdoctoral scholar N. Sutton. This year, I sponsored Nate's NIH Postdoctoral Individual National Research Service Award (F32) application to support his transition to research independence. This application is currently under review at the National Heart, Lung, and Blood Institute.

Beyond research supervision, I actively support trainees in presenting their work at conferences and in applying for external funding. My Ph.D. students have received 8 external travel awards to attend conferences, including 4 SIAM Student Travel Awards. This summer, my third-year PhD student M. Brooke-deBock will give two invited minisymposium talks at the International Conference on Mathematical Neuroscience and the SIAM Conference on the Life Sciences. To further support communication and leadership development, I encourage vertical mentoring in my group, with postdoctoral scholars co-mentoring graduate students and graduate students co-mentoring undergraduate researchers. I also support trainees in co-organizing seminars, through which they not only contribute to the research community but also gain valuable experience in inviting speakers and hosting visiting researchers. For example, this year, my Ph.D. student S. Venkatakrishnan and postdoc N. Sutton took a leading role in organizing the Brandeis Mathematical Biology Seminar.

3 Service

Since my arrival at Brandeis, I have contributed actively to services at both the departmental and university levels, while continuing to expand my contributions to the broader profession. In particular, my professional service has increasingly taken on leadership roles. I was elected to serve as Chair of the Society for Mathematical Biology Mathematical Neuroscience Subgroup (2023-2025) and recently elected as the Program Director of the Society for Industrial and Applied Mathematics (SIAM) Activity Group on Dynamical Systems (2026-2027). I will co-chair the next SIAM Conference on Applications of Dynamical Systems (DS27).

To the Department

(a) Undergraduate Committee Member, Fall 2025 - present

(b) Postdoc Search Committee Chair, Spring 2025

(c) Interim Advisor for Study Abroad Liaison and Transfer Credit, Spring 2025

(d) Co-organizer, Mathematical Biology Seminar, Fall 2023 - present

To the University

(a) Committee Member, School of Science, Engineering and Technology (SET) Curriculum Committee, Fall 2025-present

(b) Committee Member, Board of Pre-Health Advisors, 2023-2025

- In this role, I worked closely with the Pre-Health Advising team in Academic Services, interviewing and evaluating pre-health students applying to medical, dental and optometry schools, and providing feedback based on their dossiers and interviews to assess their readiness and identify areas for improvement for their chosen profession. applicants to medical school and dental school.

To the Profession

(a) Conference Co-Chair, SIAM Conference on Applications of Dynamical Systems, 2026-2027

(b) Program Director, SIAM Activity Group on Dynamical Systems, 2026-2027

(c) Organizing Committee Member, SIAM Conference on the Life Sciences, 2025-2026

(d) Advisory Member, Society for Mathematical Biology Mathematical Neuroscience Subgroup, 2025-2027

(e) Chair, Society for Mathematical Biology Mathematical Neuroscience Subgroup, 2023-2025

(f) Co-organizer of three workshops:

- SMB MathNeuro Mini-Conference (Virtual), June 12-13, 2025
  - Organized as part of my role as the SMB mathematical neuroscience subgroup chair

- Attracted over 200 participants from multiple continents
- Mathematical Biosciences Workshop, Penn State University, August 8-9, 2024
- Dynamical Systems in the Life Sciences Workshop, The Ohio State University, July 13-15, 2023
  - I helped secure NSF-supported travel funding for junior participants
  - Led to a special issue in *Mathematical Biosciences* (24 papers), where I served as guest editor

(g) Co-organizer of 11 conference minisymposia or equivalent

(h) Poster Judge

- Red Sock Award at the SIAM Conference on Applications of Dynamical Systems Poster Session, 2025
- Poster Award at Society for Mathematical Biology Annual Meeting, 2024 & 2025

(i) Served twice as a grant review panelist and once as an ad hoc reviewer for NSF/NIH Collaborative Research in Computational Neuroscience Program, 2023-2025

(j) Referee: *PLOS Computational Biology, SIAM Journal on Applied Dynamical Systems, SIAM Journal on Applied Mathematics, Journal of Computational Neuroscience, Frontiers, Chaos, Mathematical Biosciences and Engineering, Applied Mathematical Modeling, Nonlinearity, Biological Cybernetics, Neural Computation, Nonlinear Dynamics, Mathematical Biosciences Journal*

References

[1] Leandro M Alonso, Mara CP Rue, and Eve Marder. Gating of homeostatic regulation of intrinsic excitability produces cryptic long-term storage of prior perturbations. *Proceedings of the National Academy of Sciences*, 120(26):e2222016120, 2023.

[2] N. M. Awal, I. R. Epstein, T. J. Kaper, and T. Vo. Symmetry-breaking rhythms in coupled, identical fast–slow oscillators. *Chaos*, 33(1):011102, 2023.

[3] Naziru M Awal, Irving R Epstein, Tasso J Kaper, and Theodore Vo. Strong symmetry breaking in coupled, identical lengyel–epstein oscillators via folded singularities. *Journal of nonlinear science*, 34(3):53, 2024.

[4] N.M. Awal, D. Bullara, and I.R. Epstein. The smallest chimera: Periodicity and chaos in a pair of coupled chemical oscillators. *Chaos*, 29(1):013131, 2019.

[5] Nathan A Baertsch, Nicholas E Bush, Nicholas J Burgraff, and Jan-Marino Ramirez. Dual mechanisms of opioid-induced respiratory depression in the inspiratory rhythm-generating network. *Elife*, 10:e67523, 2021.

[6] R. Bertram, J. Rhoads, and W. P. Cimbora. A phantom bursting mechanism for episodic bursting. *Bulletin of Mathematical Biology*, 70:1979–1993, 2008.

[7] M. Brooke-deBock, A.K. Tryba, A.J. Garcia, and Y. Wang. Intrinsic cellular organization shapes rhythmogenesis and modulatory responses in respiratory networks. *In Preparation*, 2026.

[8] Nicholas J Burgraff, Nicholas E Bush, Jan M Ramirez, and Nathan A Baertsch. Dynamic rhythmogenic network states drive differential opioid responses in the in vitro respiratory network. *Journal of Neuroscience*, 41(48):9919–9931, 2021.

[9] Robert J Butera, John Rinzel, and Jeffrey C Smith. Models of respiratory rhythm generation in the pre-botzinger complex. ii. populations of coupled pacemaker neurons. *Journal of neurophysiology*, 82(1):398–415, 1999.

[10] M. Chevalier, N. Toporikova, J. Simmers, and M. Thoby-Brisson. Development of pacemaker properties and rhythmogenic mechanisms in the mouse embryonic respiratory network. *Elife*, 5:e16125, 2016.

[11] Hillel J Chiel and Randall D Beer. The brain has a body: adaptive behavior emerges from interactions of nervous system, body and environment. *Trends in neurosciences*, 20(12):553–557, 1997.

[12] Grant M Chou, Nicholas E Bush, Ryan S Phillips, Nathan A Baertsch, and Kameron Decker Harris. Modeling effects of variable prebötzinger complex network topology and cellular properties on opioid-induced respiratory depression and recovery. *eneuro*, 11(3), 2024.

[13] M. Desroches, J. Guckenheimer, B. Krauskopf, C. Kuehn, H.M. Osinga, and M. Wechselberger. Mixed-mode oscillations with multiple time scales. *SIAM Review*, 54(2):211–288, 2012.

[14] M. Desroches, T. J. Kaper, and M. Krupa. Mixed-mode bursting oscillations: Dynamics created by a slow passage through spike-adding canard explosion in a square-wave burster. *Chaos*, 23(4), 2013.

[15] M. Desroches, J. Rinzel, and S. Rodrigues. Classification of bursting patterns: A tale of two ducks. *PLoS Comput. Biol.*, 18(2), 2022.

[16] Rishi R Dhingra, Peter M MacFarlane, Peter J Thomas, Julian FR Paton, and Mathias Dutschmann. Asymmetric neuromodulation in the respiratory network contributes to rhythm and pattern generation. *bioRxiv*, pages 2024–11, 2024.

[17] Atsushi Doi and Jan-Marino Ramirez. Neuromodulation and the orchestration of the respiratory rhythm. *Respiratory physiology & neurobiology*, 164(1-2):96–104, 2008.

[18] C.-M. Dragoi, E. Kaur, A. R. B., J. J Tyson, and B. Novak. The oscillation of mitotic kinase governs cell cycle latches in mammalian cells. *J. Cell Sci.*, 137(3):jcs261364, 2024.

[19] C.-M. Dragoi, J. J. Tyson, and B. Novak. Newton's cradle: Cell cycle regulation by two mutually inhibitory oscillators. *Mathematical Biosciences*, 377:109291, 2024.

[20] Freddy Dumortier and Robert H Roussarie. *Canard cycles and center manifolds*, volume 577. American Mathematical Soc., 1996.

[21] William Duncan, Fernando Antoneli, Janet Best, Martin Golubitsky, Jiaxin Jin, H Frederik Nijhout, Mike Reed, and Ian Stewart. Homeostasis patterns. *SIAM Journal on Applied Dynamical Systems*, 23(3):2262–2292, 2024.

[22] B. A. Edgar and T. L. Orr-Weaver. Endoreplication cell cycles: more for less. *Cell*, 105:297–306, 2001.

[23] Irving R Epstein, Naziru M Awal, Tasso J Kaper, and Theodore Vo. Strong symmetry breaking rhythms created by folded nodes in a pair of symmetrically coupled, identical koper oscillators. *Chaos: An Interdisciplinary Journal of Nonlinear Science*, 34(5), 2024.

[24] Neil Fenichel. Geometric singular perturbation theory for ordinary differential equations. *Journal of differential equations*, 31(1):53–98, 1979.

[25] M. Fitzpatrick, Y. Wang, P.J. Thomas, R.D. Quinn, and N.S. Szczecinski. Robotics application of a method for analytically computing infinitesimal phase response curves, conference on biomimetic and biohybrid systems. *Springer*, pages 104–115, 2020.

[26] P. Gandhi, M. Golubitsky, C. Postlethwaite, I. Stewart, and Y. Wang. Bifurcations on fully inhomogeneous networks. *SIAM J. Appl. Dyn. Syst.*, 19(1):366–411, 2020.

[27] Punit Gandhi and Yangyang Wang. A conceptual framework for modeling a latching mechanism for cell cycle regulation. *Mathematical biosciences*, 382:109396, 2025.

[28] O. Ghitza. On the role of theta-driven syllabic parsing in decoding speech: intelligibility of speech with a manipulated modulation spectrum. *Frontiers in psychology*, 3(238), 2012.

[29] O. Ghitza. Behavioral evidence for the role of cortical theta oscillations in determining auditory channel capacity for speech. *Frontiers in psychology*, 5(652), 2014.

[30] O. Ghitza and S. Greenberg. On the possible role of brain rhythms in speech perception: intelligibility of time-compressed speech with periodic and aperiodic insertions of silence. *Phonetica*, 66(12):113–126, 2009.

[31] S. Ghosh, A. Mondal, P. Ji, A. Mishra, S. K. Dana, C. G. Antonopoulos, and C. Hens. Emergence of mixed mode oscillations in random networks of diverse excitable neurons: the role of neighbors and electrical coupling. *Front. comput. neurosci.*, 14:49, 2020.

[32] M. Golubitsky and Y. Wang. Infinitesimal homeostasis in three-node input–output networks. *J. Math. Biol.*, 80(4):1163–1185, 2020.

[33] Martin Golubitsky and Ian Stewart. *The symmetry perspective: from equilibrium to chaos in phase space and physical space*, volume 200. Springer Science & Business Media, 2003.

[34] Martin Golubitsky and Ian Stewart. *Dynamics and bifurcation in networks: theory and applications of coupled differential equations*. SIAM, 2023.

[35] Martin Golubitsky, Ian Stewart, Fernando Antoneli, Zhengyuan Huang, and Yangyang Wang. Input-output networks, singularity theory, and homeostasis. In *Advances in Dynamics, Optimization and Computation: A volume dedicated to Michael Dellnitz on the occasion of his 60th birthday*, pages 31–65. Springer, 2020.

[36] Martin Golubitsky, Ian Stewart, Pietro-Luciano Buono, and JJ Collins. Symmetry in locomotor central pattern generators and animal gaits. *Nature*, 401(6754):693–695, 1999.

[37] Martin Golubitsky, Ian Stewart, and David G Schaeffer. *Singularities and groups in bifurcation theory: volume II*, volume 69. Springer Science & Business Media, 2012.

[38] J. L. Hudson, M. Hart, and D. Marinko. An experimental study of multiple peak periodic and nonperiodic oscillations in the Belousov–Zhabotinskii reaction. *J. Chem. Phys.*, 71(4):1601–1606, 1979.

[39] P.E. Jasinski, Y.I. Molkov, N.A. Shevtsova, J.C. Smith, and I.A. Rybak. Sodium and calcium mechanisms of rhythmic bursting in excitatory neural networks of the pre-botzinger complex: a computational modelling study. *Eur. J. Neurosci.*, 37:212—-230, 2013.

[40] J. Kimrey, T. Vo, and R. Bertram. Big ducks in the heart: canard analysis can explain large early afterdepolarizations in cardiomyocytes. *SIAM J. Appl. Dyn. Syst.*, 19(3):1701–1735, 2020.

[41] Elif Köksal Ersöz, Mathieu Desroches, Antoni Guillamon, John Rinzel, and Joel Tabak. Canard-induced complex oscillations in an excitatory network. *Journal of mathematical biology*, 80(7):2075–2107, 2020.

[42] M. Krupa, N. Popović, N. Kopell, and H. G. Rotstein. Mixed-mode oscillations in a three time-scale model for the dopaminergic neuron. *Chaos: An Interdisciplinary Journal of Nonlinear Science*, 18(1):015106, 2008.

[43] Martin Krupa and Peter Szmolyan. Extending geometric singular perturbation theory to non-hyperbolic points—fold and canard points in two dimensions. *SIAM journal on mathematical analysis*, 33(2):286–314, 2001.

[44] B. Letson, J.E. Rubin, and T. Vo. Analysis of interacting local oscillation mechanisms in three-timescale systems. *SIAM J. Appl. Dyn. Syst.*, 77(3):1020–1046, 2017.

[45] Xinni Lin, Fernando Antoneli, and Yangyang Wang. Automated classification of homeostasis structure in input-output networks. *arXiv preprint arXiv:2603.08882*, 2026.

[46] Zheng Liu, Jorge Golowasch, Eve Marder, and LF Abbott. A model neuron with activity-dependent conductances regulated by multiple calcium sensors. *Journal of Neuroscience*, 18(7):2309–2320, 1998.

[47] D.N. Lyttle, J.P. Gill, K.M. Shaw, P.J. Thomas, and H.J. Chiel. Robustness, flexibility, and sensitivity in a multifunctional motor control model. *Biol. Cybern.*, 2017.

[48] W. Ma, A. Trusina, H. El-Samad, W.A. Lim, and C. Tang. Defining network topologies that can achieve biochemical adaptation. *Cell*, 138:760–773, 2009.

[49] R. Manzoni, F. Montani, C. Visinitin, F. Caudron, A. Ciliberto, and R. Visintin. Oscillations in cdc14 release and sequestration reveal a circuit underlying mitotic exit. *J. Cell Biol.*, 190:209–222, 2010.

[50] P. Nan, Y. Wang, V. Kirk, and J.E. Rubin. Understanding and distinguishing three-time-scale oscillations: case study in a coupled morris-lecar system. *SIAM J. Appl. Dyn. Syst.*, 14:1518–1557, 2015.

[51] Bela Novak and John J Tyson. Mitotic kinase oscillation governs the latching of cell cycle switches. *Current Biology*, 32(12):2780–2785, 2022.

[52] Chinwendu Nwakudu, Caroline Szujewski, Ellen Ma, and Alfredo J Garcia III. A model of repeated opioid use producing tolerance to opioid induced respiratory depression. *The FASEB Journal*, 36, 2022.

[53] Timothy O'Leary, Alex H Williams, Alessio Franci, and Eve Marder. Cell types, network homeostasis, and pathological compensation from a biologically plausible ion channel expression model. *Neuron*, 82(4):809–821, 2014.

[54] E. Pavlidis, F. Campillo, A. Goldbeter, and M. Desroches. Multiple-timescale dynamics, mixed mode oscillations and mixed affective states in a model of bipolar disorder. *Cognitive Neurodynamics*, 2022.

[55] N.A. Phan and Y. Wang. Mixed-mode bursting oscillations in a three-timescale biophysical neural oscillator model. *In Preparation*, 2026.

[56] Ngoc Anh Phan and Yangyang Wang. Mixed-mode oscillations in a three-timescale coupled morris–lecar system. *Chaos: An Interdisciplinary Journal of Nonlinear Science*, 34(5), 2024.

[57] Ryan S Phillips, Tibin T John, Hidehiko Koizumi, Yaroslav I Molkov, and Jeffrey C Smith. Biophysical mechanisms in the mammalian respiratory oscillator re-examined with a new data-driven computational model. *Elife*, 8:e41555, 2019.

[58] Ryan S Phillips and Jonathan E Rubin. Putting the theory into 'burstlet theory'with a biophysical model of burstlets and bursts in the respiratory prebötzinger complex. *Elife*, 11:e75713, 2022.

[59] B. Pittman-Polletta, Y. Wang, D. Stanley, C. Schroeder, M. Whittington, and N. Kopell. Differential contributions of synaptic and intrinsic inhibitory currents to parsing via flexible phase-locking in neural oscillators. *PLoS Computational Biology*, 17(4):e1008783, 2021.

[60] Martin L Puterman. *Markov decision processes: discrete stochastic dynamic programming*. John Wiley & Sons, 2014.

[61] Karan G Rai, Chinwendu U Nwakudu, Caroline C Szujewski, Brigitte M Browe, Gia E Fisher, Willard W Sharp, Andrew K Tryba, and Alfredo J Garcia 3rd. Divergent ventilatory responses during opioid-induced respiratory depression in response to repeated fentanyl use. *American Journal of Physiology-Lung Cellular and Molecular Physiology*, 329(1):L161–L171, 2025.

[62] John Rinzel. A formal classification of bursting mechanisms in excitable systems. In *Mathematical Topics in Population Biology, Morphogenesis and Neurosciences: Proceedings of an International Symposium held in Kyoto, November 10–15, 1985*, pages 267–281. Springer, 1987.

[63] J. E. Rubin, J. Signerska-Rynkowska, J. D. Touboul, and A. Vidal. Wild oscillations in a nonlinear neuron model with resets: (ii) mixed-mode oscillations. *Discrete Contin. Dyn. Syst.*, 22(10):4003–4039, 2017.

[64] J. Smith, H. Ellenberger, K. Ballanyi, D. Richter, and J. Feldman. Pre-bötzinger complex: a brainstem region that may generate respiratory rhythm in mammals. *Science*, 254(5032):726–729, 1991.

[65] Richard S Sutton, Andrew G Barto, et al. *Reinforcement learning: An introduction*, volume 1. MIT press Cambridge, 1998.

[66] Richard S Sutton, Doina Precup, and Satinder Singh. Between mdps and semi-mdps: A framework for temporal abstraction in reinforcement learning. *Artificial intelligence*, 112(1-2):181–211, 1999.

[67] N. Toporikova and R.J. Butera. Two types of independent bursting mechanisms in inspiratory neurons: an integrative model. *J. Comp. Neurosci.*, 30(3):515–528, 2011.

[68] N. Toporikova, M. Chevalier, and M. Thoby-Brisson. Sigh and eupnea rhythmogenesis involve distinct interconnected subpopulations: A combined computational and experimental study. *eNeuro*, 2(2):ENEURO–0074, 2015.

[69] Andrew Kieran Tryba, Jean-Charles Viemari, Yangyang Wang, and Alfredo J Garcia 3rd. Noradrenergic neuromodulation produces a nmdar-dependent network state of respiratory rhythmogenesis in the prebötzinger complex. *bioRxiv*, pages 2026–02, 2026.

[70] Sreshta Venkatakrishnan, Andrew K Tryba, Alfredo J Garcia III, and Yangyang Wang. Dual mechanisms for heterogeneous responses of inspiratory neurons to noradrenergic modulation. *SIAM Journal on Life Sciences*, 1(1):58–98, 2026.

[71] Jean-Charles Viemari and Jan-Marino Ramirez. Norepinephrine differentially modulates different types of respiratory pacemaker and nonpacemaker neurons. *Journal of neurophysiology*, 95(4):2070–2082, 2006.

[72] T. Vo, J. Tabak, R. Bertram, and M. Wechselberger. A geometric understanding of how fast activating potassium channels promote bursting in pituitary cells. *J. Comput. Neurosci.*, 36:259–278, 2014.

[73] T. Vo and Y. Wang. A symmetric mechanism for strong symmetry-breaking in oscillator networks with nonlinear coupling. *In Preparation*, 2026.

[74] Y. Wang, J.P. Gill, H.J. Gill, and P.J. Thomas. Shape versus timing: linear responses of a limit cycle with hard boundaries under instantaneous and static perturbation. *SIAM J. Appl. Dyn. Syst.*, 20(2):701–744, 2021.

[75] Y. Wang, Z. Huang, F. Antoneli, and M. Golubitsky. The structure of infinitesimal homeostasis in input-output networks. *J. Math. Biol.*, 82(7):1–43, 2021.

[76] Y. Wang and B.R. Pittman-Polletta. Dynamical mechanisms of flexible phase-locking in cortical theta oscillators. *Submitted*, 2026.

[77] Y. Wang and J.E. Rubin. Multiple time scale mixed bursting dynamics in a respiratory neuron model. *J. Comp. Neurosci.*, 41(3):245–268, 2016.

[78] Y. Wang and J.E. Rubin. Timescales and mechanisms of sigh-like bursting and spiking in models of rhythmic respiratory neurons. *J. Math. Neurosci.*, 7(1):1–39, 2017.

[79] Y. Wang and J.E. Rubin. Complex bursting dynamics in an embryonic respiratory neuron model. *Chaos: an interdisciplinary journal of nonlinear science*, 30(4):043127, 2020.

[80] Yangyang Wang. *Analysis of Complex Bursting Patterns in Multiple Timescale Respiratory Neuron Models*. PhD thesis, University of Pittsburgh, 2016.

[81] Yangyang Wang, Jeffrey P Gill, Hillel J Chiel, and Peter J Thomas. Variational and phase response analysis for limit cycles with hard boundaries, with applications to neuromechanical control problems. *Biological cybernetics*, 116(5):687–710, 2022.

[82] Changliang Wei, Yangyang Wang, and Xueyu Zhu. Robust parameter and state estimation in multiscale neuronal systems using physics-informed neural networks. *arXiv preprint arXiv:2603.08742*, 2026.

[83] Zhuojun Yu, Yangyang Wang, Peter J Thomas, and Hillel J Chiel. Tradeoffs in the energetic value of neuromodulation in a closed-loop neuromechanical system. *Journal of theoretical biology*, 604:112050, 2025.

[84] Sébastien Zanella, Atsushi Doi, Alfredo J Garcia, Frank Elsen, Sarah Kirsch, Aguan D Wei, and Jan-Marino Ramirez. When norepinephrine becomes a driver of breathing irregularities: how intermittent hypoxia fundamentally alters the modulatory response of the respiratory network. *Journal of Neuroscience*, 34(1):36–50, 2014.