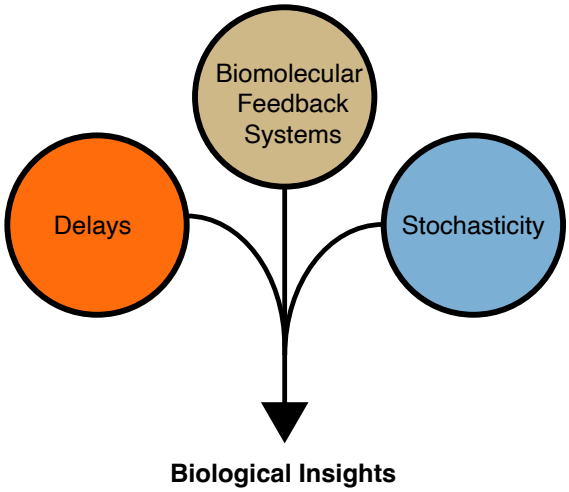
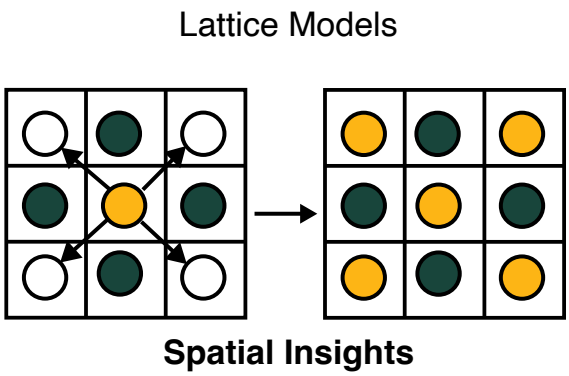
Introduction. My interests lie in the interdisciplinary field of Biomathematics. Broadly speaking, my current research focuses on stochastic processes, dynamical systems, nonequilibrium statistical physics, and control theory, with particular applications to biomolecular feedback systems, spatiotemporal ordering in biological systems, and social decision-making. I am specifically interested in developing mathematical models of biological and decision-making processes and analyzing them with mathematical techniques to unveil fundamental principles underlying them. Over the past five years, I have worked extensively in three primary areas:

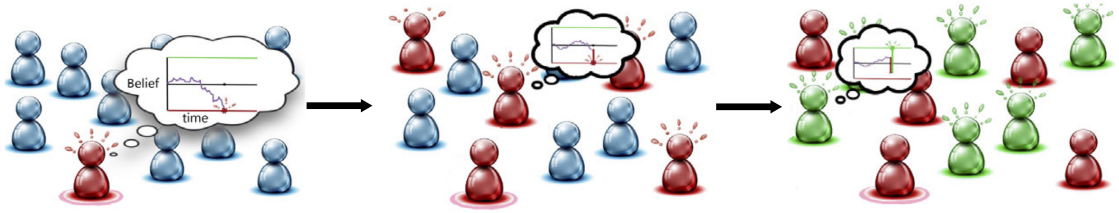
(1) **Biomolecular Feedback Systems** Biomolecular feedback systems (BFS) are a hallmark of biological modeling, and specifically have been used to describe gene network motifs and how they affect macroscopic protein concentration dynamics. However, most existing models of BFS are comprised of ODEs. While such models can be insightful, they do not grasp key details that could affect protein output. **My research into BFS involves investigation of the roles temporal delay and stochasticity, together or separately, affect dynamics of biomolecular feedback systems.** The effect of delays is important because any biophysical action requires nonzero time to occur. Stochasticity is important to consider because gene networks exist in the subcellular environment, which is a highly fluctuating, heterogeneous environment.



(2) **Lattice Models of Spatial Systems** Spatial patterns are a widely studied phenomenon across disciplines. Theoretical formulations of spatial patterns often involve PDEs or agent-based modeling. PDEs offer analytic tractability in many cases but are too coarse-grained to bring to light how individuals may affect macroscopic patterns. Agent-based models incorporate detailed physics of the system in question, but they are often too complicated to analyze and computationally expensive. Lattice models provide a middle ground and can link how individual interactions affect global spatial structure. They are computationally efficient and allow for derivation of mean field equations that can glean insights into biological systems. **Lattice models I developed have been applied to molecular motor dynamics, spatiotemporal ordering in synthetic bacteria, and foraging ants.**



(3) **Social Decision-Making** Most normative models of decision-making apply to individuals in isolation. While they successfully describe decision-making in a laboratory setting, they fail to apply to the real world, where decisions are made by incorporating other individuals' behaviors. I have generalized well-established Bayesian models of individual decision-making to describe social decision-making. **I have developed models that reveal the effect the first decision**



in a group affects overall group decision accuracy, how correlated information affects group accuracy, and how biases affect group accuracy in a group of rational, optimal observers.

Though my research interests are quite diverse in scope, they are unified by the main research questions that underlie them: how do actions and interactions at the individual level affect macroscopic behavior? What effect does noise impart upon the system?

In the following I provide details of my most substantive projects pertaining to each topic described above.

1. Biomolecular Feedback Systems

Background. Biomolecular feedback systems are biological networks consisting of positive or negative feedback [23]. Positive feedback describes a process or reaction whose output amplifies the process. Negative feedback describes a process where the output inhibits the process [23,33,59]. Such feedback loops are prominent across scales, ranging from interactions at the molecular and genetic level through the tissue and organ level [13, 37, 42, 43]. A formal elucidation of several types of biomolecular feedback was first presented by John Tyson in the early 2000s [64]. Since then, feedback systems have become a hallmark of several theoretical studies of various biological systems.

There is a rich literature surrounding theory of bimolecular feedback systems, but most models studied are comprised of ODEs. While significant, they do not take into consideration inherent delays or noise. Modeling biomolecular feedback loops with DDEs or stochasticity renders more biologically plausible outputs from models. In the following, I describe some recent projects in this capacity.

1.1. Stochastic Delays in Delayed Negative Feedback.

Delayed negative feedback is a paradigmatic BFS whose signature temporal dynamic is oscillations [33, 41, 43, 59, 64, 70]. It describes a process wherein the output of a reaction inhibits the reaction following a temporal delay. A simple model of delayed negative feedback is as follows. Let $y(t)$ be a scalar field evolving according to

$$\frac{dy}{dt} = I - \gamma y - w\frac{y(t-\tau)^n}{K^n + y(t-\tau)^n}. \tag{1}$$

Here, $y(t)$ could represent the concentration of a protein that is constitutively produced at a rate I and inhibits its own production. The first order rate constant γ describes the natural degradation rate of the substance y. The weight w to describe the strength of the autoinhibition based on Michaelis-Menten kinetics. For sufficiently small τ, Eq. (1) exhibits a stable equilibrium. However, for delay values past some critical value, τ_c, the system admits a limit cycle, having undergone a supercritical Hopf bifurcation at $\tau = \tau_c$ (see Fig. 1).

This feature of delayed negative feedback systems is so ubiquitous that experimentalists often immediately attribute any observed oscillations in the lab to it. However, intracellular protein dynamics are subject to noisy, heterogeneous environments, so investigating how noise affects such oscillatory dynamics is necessary [16].

How to incorporate noise to describe intracellular processes is an open question [16, 36, 63]. Here, we take τ to be time-dependent and to evolve according to a continuous time Markov chain on a finite discrete set, $\tau \in \{\tau_1, \tau_2, ..., \tau_N\}$ with $N \in \mathbb{N}$. The model therefore couples Eq. (1) with a master equation for $Q(\tau_i, t)$, the probability that $\tau = \tau_i$ at time t:

$$\frac{dQ}{dt}(\tau_i, t) = \sum_{j=1}^{N} \mathcal{W}_{ji} Q(\tau_j, t) - \mathcal{W}_{ij} Q(\tau_i, t), \tag{2}$$

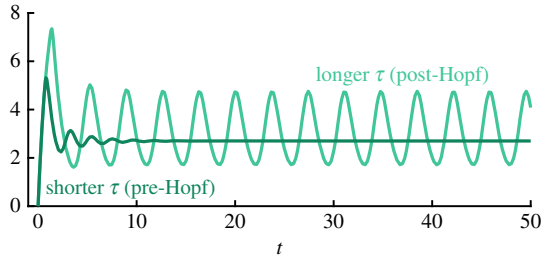


Figure 1. τ-dependent supercritical Hopf bifurcation in Eq. (1).

where $\mathcal{W}_{ij}$ denotes the propensity of the transition $\tau_i \to \tau_j$ and $\mathcal{W}_{jj} = 0$. Equations (1) and (2) together form a so-called *stochastic hybrid system*—a system wherein the state of the system evolves stochastically, but within each state the system evolves deterministically [15].

We found that if the transitions between states occurred fast enough, oscillations disappeared from the dynamics of Eq. (1), even if each $\tau_i > \tau_c$. To understand why, we invoked the van Kampen [65] and quasi steady state [16] approximations upon Eqs. (1) and (2) to compute the effective equation governing the dynamics. In the case $N = 2$, we found that effective equation was

$$\frac{dy_{\text{eff}}}{dt} = I - \gamma y_{\text{eff}} - \frac{\beta}{\beta+\alpha} \frac{w y_{\text{eff}}(t-\tau_1)^n}{K^n + y_{\text{eff}}(t-\tau_1)^n} - \frac{\alpha}{\beta+\alpha} \frac{w y_{\text{eff}}(t-\tau_2)^n}{K^n + y_{\text{eff}}(t-\tau_2)^n}. \tag{3}$$

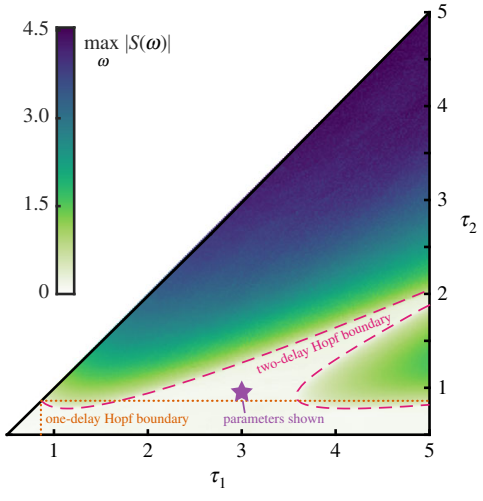


Figure 2. Bifurcation structure of Eq. (3).

Surprisingly, the effective equation did not include a single effective delay, but rather incorporated the effects of all the delays simultaneously via a linear combination of the feedback functions. Each feedback function was weighted by the stationary measure of the Markov chain.

The equilibrium behavior of Eq. (3) is substantially different than Eq. (1). In Figure 2, we show the bifurcation structure of Eq. (3) by computing the spectrogram of the solutions for sampled pairs of delays. We see a substantial region past τ_c in the two-delay case where oscillations are not observed.

The mathematical novelty of this work was the computation of the effective multi delay equation in the fast switching limit, providing further mechanistic justification for delay distributions commonly used in gene network models. Biologically, it is fascinating that noise undoes a common feature of delayed negative feedback–namely oscillations. How the effects of delays in the delay interact with other sources of noise to impact dynamics is an intriguing area of potential future work.

1.2. **Oscillations in Delayed Positive Feedback Systems.** Positive feedback is ubiquitous in biological systems and serves to produce switch-like responses to input. This feedback often results in bistability, in which there are two stable equilibrium states that the system can be in, depending on its starting conditions. Classic examples involving two agents are the toggle switch [29], in which one agent is activated and the other inactivated, and the one-way switch [8] in with either both agents are activated or both inactivated. In neither case are oscillations in activity levels expected. Indeed, the whole point of the positive feedback is to make the system regenerative, so input produces either a large response or no response at all.

We recently discovered that positive feedback systems can exhibit oscillatory behavior [55]. If explicit temporal delay is taken into consideration, oscillations may appear. Importantly, the oscillations are not an asymptotic state—that is, there is no limit cycle. Rather, the oscillations are a long-lasting transient, often persisting for several periods, before the dynamics contract to one of the bistable equilibria. We will show this with a simple model of the toggle switch, with explicit temporal delay included. Let $x(t)$ and $y(t)$ be scalar fields evolving according to

$$\frac{dx}{dt} = \frac{\alpha}{1+y(t-\tau)^n} - x \qquad \frac{dy}{dt} = \frac{\alpha}{1+x(t-\tau)^n} - y, \tag{4}$$

where α represents the maximal production rate of x and y and $n > 0$ is the Hill coefficient characterizing the interaction. Here, x and y could represent the concentrations of enzymes that inhibit each other, such as Cdc2-Cyclin B and Wee1 in the cell cycle [5, 64]. Though Eq. (4) describes two species inhibiting one another, each species provides positive feedback onto itself by deactivating its repressor. Thus the net effect of x on x and y on y is positive feedback.

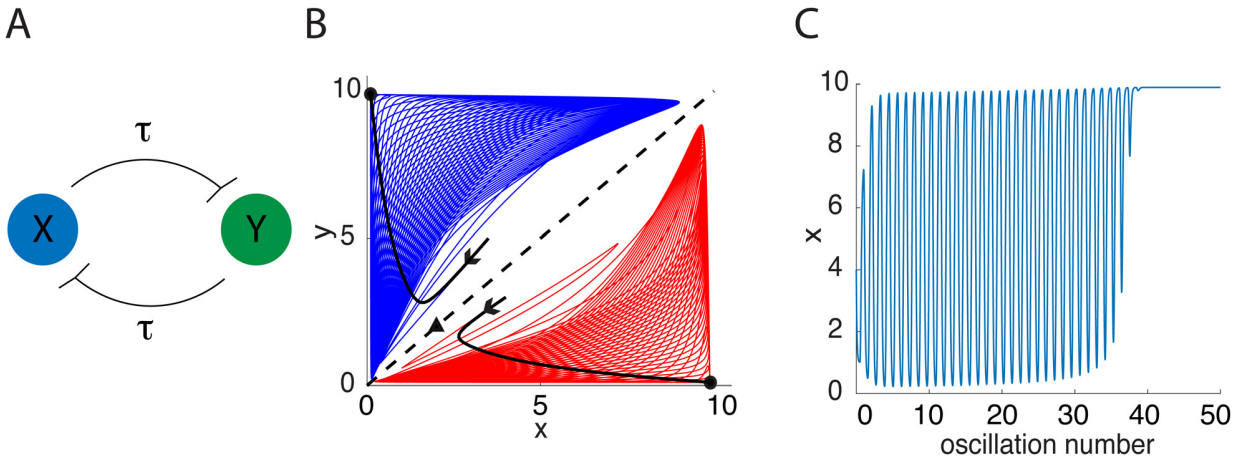


Figure 3. The delayed toggle switch. (A) Schematic. (B) Projection of phase space onto two dimensions. Blue and red curves are solutions to Eq. (4) demonstrating oscillatory dynamics on either side of the seperatrix. Black curves are solutions to the corresponding ODE system, showing how much information is missed by it. Dashed line is the seperatrix, and for this system is the line $y = x$.

For $n \geq 2$, Eq. (4) is bistable [2] (see Fig. 3B), with the seperatrix being the stable manifold of the saddle point between the two stable equilibria. In general, determining the stable manifold of an infinite-dimensional system is difficult, but for Eq. (4), the projection of the stable manifold onto the xy–plane is the line $y = x$ [9] (Fig. 3B dashed line). Along this invariant manifold, Eq. (4) reduces to the single equation

$$\frac{dx}{dt} = \frac{\alpha}{1+x(t-\tau)^n} - x, \tag{5}$$

which is a model of delayed *negative feedback*, meaning for sufficiently large τ values, the system admits a persistent oscillation via a supercritical Hopf bifurcation. Importantly, only along the stable manifold does the persistent oscillation exist. Moving slightly off the stable manifold immediately renders the oscillation a transient. However, they last for many cycles (see Fig. 3C) and they resemble the oscillation along the stable manifold. As such, we call these long-lasting transients remnants of the delayed negative feedback oscillator.

The duration of the transient depends on the value of τ and the distance, Δ, from the stable manifold. Notably, the number of oscillations increases superlinearly as τ is increased and decreases as the distance of the initial data from the stable manifold increases.

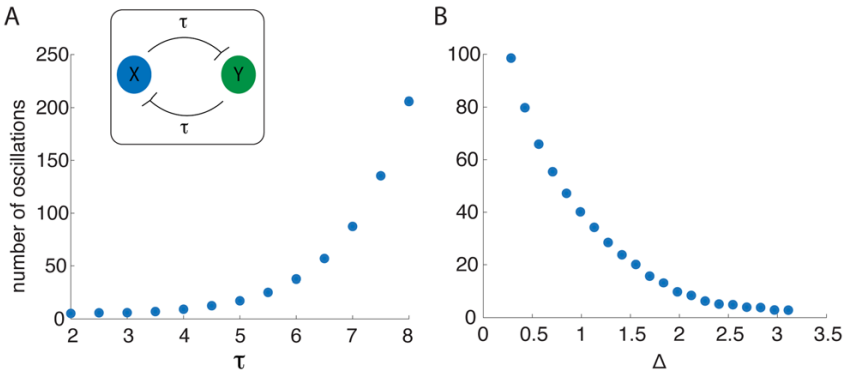


Figure 4. Duration of transient oscillations with respect to τ and Δ.

We examined various delayed positive feedback systems, and the properties that hold for the toggle switch appear to hold in general. That is, long-lasting oscillations exist in the phase space of delayed positive feedback systems. They are driven by a persistent oscillation that exists along an invariant manifold of the phase space.

Though the oscillations are transient, they could be sufficiently long-lasting so as to outlive the species or cell within which they occur. This result **fundamentally challenges current understanding of mechanisms driving biological oscillations.** Positive feedback loops are designed to resist oscillating. Protein oscillations are abundant in biological systems [17,35,49]. It is unlikely that all of them are driven by delayed negative feedback. The implication of our work is that oscillations observed in the lab may be driven by positive feedback, which is unprecedented. Oscillations observed in the experimental lab are often attributed to delayed negative feedback. Our results here argue that that assumption could prevent investigation into the true mechanism that may underlie biological oscillations in specific cases.

1.3. **Further Reading.**

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(2) C. J. Ryzowicz, R. Bertram, **B. R. Karamched**. Oscillations in Delayed Positive Feedback Systems. Physical Chemistry Chemical Physics. 26, 24861-24869. (2024) (summarized above)
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(4) F. Bai, R. Bertram, and **B. R. Karamched**. A Mathematical Study of the Efficacy of Possible Negative Feedback Pathways Involved in Neuronal Polarization. Journal of Theoretical Biology. 111561. (2023)

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2. Lattice Models of Spatial Systems

Background. Lattice models have a rich history in biological modeling. They provide a valuable framework for modeling complex spatiotemporal dynamics in biological tissues. They are an alternative to agent-based models and PDEs for modeling emergent spatiotemporal patterning. Agent-based models provide realistic models of biological tissues and populations, but are often intractable and computationally expensive. Partial differential equation models offer scope for analysis, but they often coarse grain dynamics so that an individual's impact on the population is unclear. Lattice models capture individual properties at a high level but nevertheless sacrifice some fidelity to reality for the sake of analytical tractability. Often times, one can invoke a mean field approximation to derive an effective equation to describe overall dynamics of the microscopic configurations of the lattice.

Lattice models have been used to model protein folding [58], cancer initiation and progression [45, 46], and motor protein transport through a cell [12, 14, 47], amongst numerous other applications [10, 21]. Here I will describe in detail lattice models I developed for understanding (1) emergent spatiotemporal ordering in synthetic microbial consortia and (2) trail formation in foraging ants.

2.1. Cell Alignment in Extended Microfluidic Traps.

A central goal of synthetic biology is the construction of practical, engineered genetic circuits for medical and industrial applications [8, 19]. Critical to this goal is the elucidation of the fundamental mechanisms that govern gene regulation at all levels.

Populations of *E. coli* cells trapped in microfluidic devices can be used to study genetic signaling networks and understand how information is communicated between genetic modules distributed across two or more strains of bacteria. In extended microfluidic traps, populations are not well- mixed; therefore, spatiotemporal patterning of distinct bacterial strains plays an important role in inter-strain communication [1]. For example, multi-strain consortia of *E. coli* in open, rectangular microfluidic traps

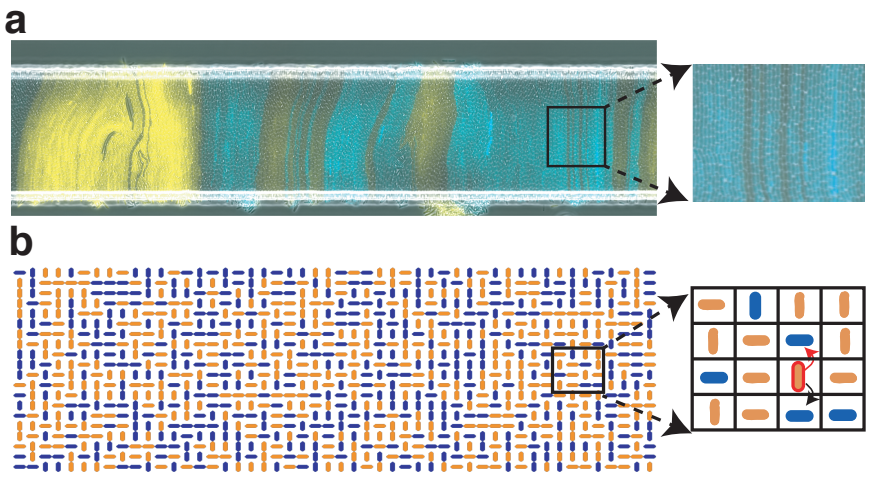

Figure 5. (a) A monolayer of *E. Coli* in an open microfluidic trap with cells aligned orthogonally to the trap's long side. Colors represent distinct strains. (b) In our spatial Moran model cell growth is directional and location dependent: The outlined vertical cell can grow only upward or downward at a location-dependent rate. The red arrow indicates growth direction, so the cell above will be replaced by a descendant of the outlined cell. We model single strain populations, but use the same color for mother and daughter cells for visualization.

form single-strain bands
orthogonal to the long side of the trap (Fig. 5) [1]. The distribution of such bands can affect the efficiency of communication between distinct strains of bacteria due to the limited diffusivity of quorum sensing molecules. Understanding the mechanisms underlying this emergent order is therefore important for engineering synthetic gene circuits with desired properties.

We developed an analytically tractable spatial Moran model (SMM) that captures essential features of the dynamics of growing populations of *E. coli* cells and provides insight into the emergence of these single-strain bands [38]. These capsule-shaped bacterial cells tend to grow more slowly in crowded environments; that is, cells in the center of a trap grow slower than cells along the boundary [20,68]. We model the microfluidic trap as an $M \times N$ lattice and the cells as oriented particles on the lattice (Fig. 5). The cells are in one of two orientations: horizontal or vertical. These cells grow along the major axis of their bodies asymmetrically. We assume a cell's growth rate in a given direction is a monotonically decreasing function of the distance the cell is from the boundary in that direction. The SMM shows that provided this growth-rate dampening due to crowding, denoted by the parameter κ, is sufficiently strong, cells align orthogonally to the long side of the trap, as seen in experiments. However, if κ decreases below a critical value κ^*, a phase transition occurs and cells align *parallel* to the long side of the trap.

The transitions between various microscopic configurations of the lattice are described by a master equation [16, 67] for the probability $p_{ij}(t)$ that the ijth site of the lattice is occupied by a vertical cell. Invoking a mean field approximation and averaging the resulting equation over all of space [62] shows that the fraction of cells vertical at time t in the lattice, $n(t)$, obeys a logistic equation

$$\frac{dn}{dt} = \mu(\kappa, M, N)n(1-n) \qquad \mu(\kappa, M, N) \equiv 2\left(\bar{v}_\kappa(1-\frac{1}{M}) - \bar{h}_\kappa(1-\frac{1}{N})\right). \tag{6}$$

Here, $\bar{v}_\kappa$ and $\bar{h}_\kappa$ are effective growth rates in the vertical and horizontal directions, respectively, and emerge from the mean field. The directional growth rates decrease exponentially as a cell's distance to the boundary in that direction grows. The logistic growth rate μ conveys that whether all cells are vertical or horizontal at equilibrium is determined by a competition between κ and the boundaries of the domain.

Setting $\mu = 0$ allows for the calculation of κ^* for any lattice with dimensions $sM \times sN$:

$$\kappa^* = \frac{2}{MNs^2}$$

We computed κ^* for several different growth functions (Fig. 6a) and several different lattice dimensions (Fig. 6b), and the match is remarkable. The lattice model asserts that cell orientation occurs via a competition between how strongly a cell feels its surrounding neighbors versus how far it is from the boundary that will eject it from the microfluidic trap.

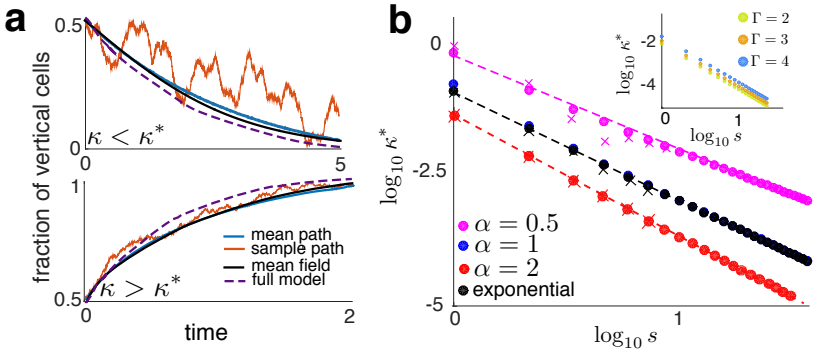

Figure 6. How the mean field compares with stochastic simulations of the full model.

We further used the same Moran model to demonstrate that in multi-strain microbial populations, rounder cells will flush more rod-like cells out of the microfluidic trap [69]. We also demonstrated by incorporating quorum sensing dynamics that experimentalists can dynamically

alter the strain ratio in a microfluidic trap. This is significant for synthetic biologists as they try to engineer microbial systems that can coordinate activity across large spatial domains.

2.2. **Trail Formation in Foraging Ants.** Foraging for resources is an essential process for the daily life of an ant colony. What makes this process so fascinating is the self-organization of ants into trails using chemical pheromone in the absence of direct communication. In the absence of an external motivation or central control center, ants can perform complex sets of tasks [25, 26] and exhibit macroscopic emergent behavior [22, 24]. This makes them an ideal model organism for studying the physical origins of self-organizing behavior, and understanding organization in ants can unveil general principles of self-organization.

In order to find food for survival, ant colonies send foragers away from the nest executing a random search process. All foragers effectively perform Brownian motion until food is found [18,52]. Once an ant finds food, it collects a morsel and makes a beeline to the nest, secreting pheromone along the way [22, 27, 28, 30, 31, 51, 54, 61]. The pheromone signals to the other ants of where the food is, and trail formation begins. We developed a lattice model to describe this phenomenon.

We model the general terrain as an $M \times N \subset \mathbb{N}^2$ lattice and the ants as $n \in \mathbb{N}$ particles hopping along the lattice nodes [34]. We assume that the timescale of trail formation is small enough (approximately 4-8 hours from biological observation of army ants *E. burchellii* [56, 57]) that we ignore births and death in the colony. There is no direct communication between individuals, but rather a response to a chemical pheromone gradient if present represents the only means of (indirect) communication. We designate a single site as the nest, $\mathbf{x}_0$. Initially, all n ants occupy that designated site. To understand how the location of food sources relative to the ant nest affects spatiotemporal structure of ant motion, we also randomly designate $\mathcal{N} \in \mathbb{N}$ sites as food sources. We assume the colony of ants are particles represented by a set of points $\{\mathbf{x}_i\}$, $i = 1, ..., n$, where $\mathbf{x}_i \in [1, M] \times [1, N]$. Each point can be thought of as the location of the center of mass for an individual ant. We assume the boundaries are reflecting for ants to maintain a fixed population.

In the absence of pheromone, foragers perform a random walk on the lattice, moving with equal probability to any site in its Moore neighborhood on a given time step (see Fig. 7. Once an ant finds food, it makes a beeline to the nest and secretes pheromone along the way. Pheromone deposition is modeled by a two-dimensional reaction-diffusion process for the chemical concentration $c(\mathbf{x}, t)$ first introduced in [54]:

$$\frac{\partial c}{\partial t} - D\Delta_{ij}c + \gamma c = \sum_{k=1}^{K}\sum_{j=1}^{J} Ae^{-\left(||\mathbf{x}_j(t)-\mathbf{x}_f^{(k)}||\right)^2}\delta(\mathbf{x}-\mathbf{x}_j(t)). \tag{7}$$

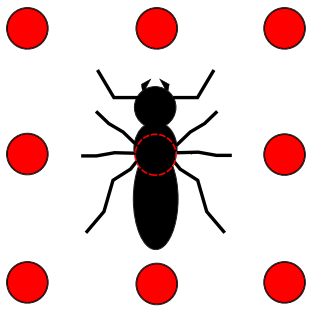

Figure 7. Foraging ant and its Moore neighborhood.

Here $\mathbf{x}_f^{(k)}$ is the location of the K food source(s), J is the number of food-carrying ants, Δ_{ij} is the discrete Laplacian, D is the diffusion coefficient controlling the rate at which the pheromone spreads, and γ is the evaporation coefficient that ensures an exponential decay of the pheromone in time. The coefficient $Ae^{-\left(||\mathbf{x}_j(t)-\mathbf{x}_f^{(k)}||\right)^2}$ represents the amount of pheromone deposited at time t and decays as a food-carrying ant moves away from the food source. This decrease is needed to ensure that the proper gradient forms due to the competition with diffusion. Eq. (7) is coupled with homogeneous Fourier-type boundary conditions.

With pheromone present, foraging ants perform a biased random walk, where they will preferentially move to the site in their Moore neighborhood yielding the greatest increase in local pheromone concentration. They maintain a non-zero probability of going into any other site. Simulations show that in environments with a single

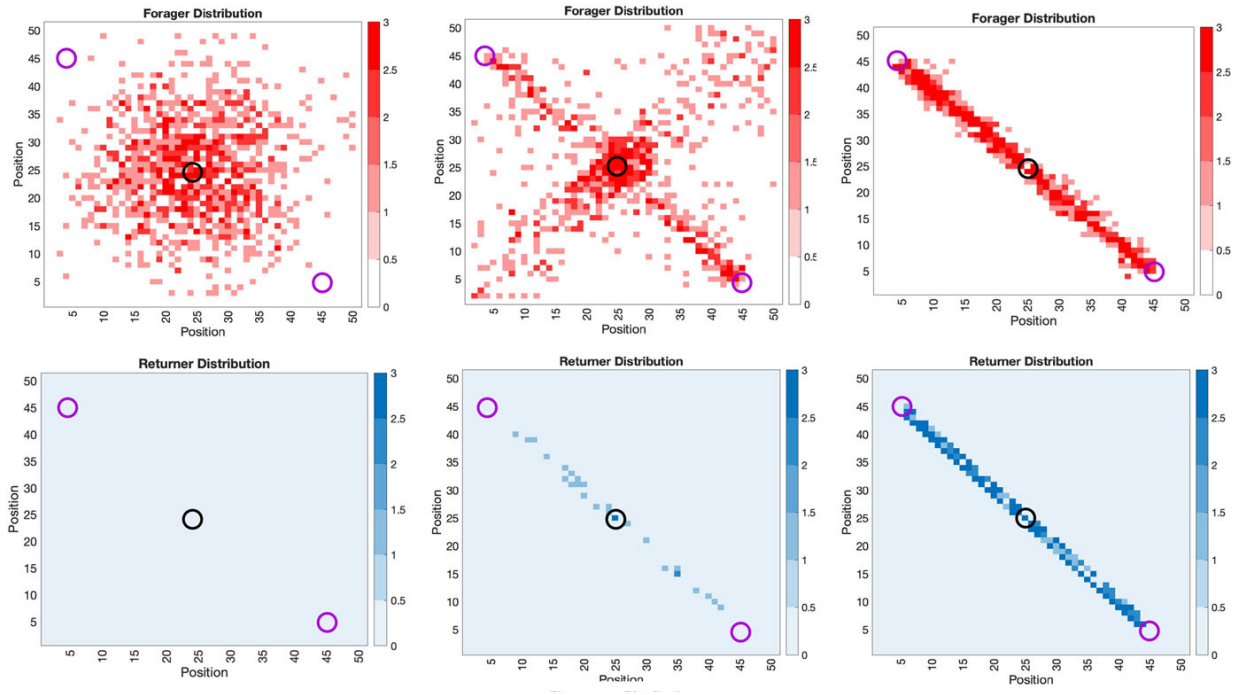

Figure 8. Snapshots showing emergence of trails in an environment with two food sources. Black circles are the nest and purple circles are food sources.

food source, ants will establish trails connecting the nest to the food source. There are several models that do this. **The main contribution of our model is that it allows for simultaneous trail formation to multiple food sources in environments with multiple food sources.** (see Fig. 8) Other trail formation models have failed to do this consistently, which is problematic because simultaneous trails are established by ant colonies [3,4,22,53]. The reason our model succeeded where others failed was we allowed pheromone-sensing foragers to have a non-zero probability of moving away from the pheromone. This sufficed to allow for robust multiple trail formation.

Our model predicts that if food sources are equidistant from the nest, ants will form longstanding trails to both food sources. That is, simultaneous trails will be the stable equilibrium of our model. If food sources are anisometric relative to the nest, simultaneous trails will form, but only transiently. All ants eventually converge to the trail connecting to the closer food source.

We were able to write down a master equation for the transitions between the microscopic configurations of the lattice and invoke a mean-field approximation and a continuum limit to derive a set of PDEs for foragers. One key contribution stemming from this derivation was that we performed a linear stability analysis and computed dispersion curves [50] to show that the presence of food was the cause of trail formation and that its existence was necessary but not sufficient. Parameters had to be tuned appropriately to ensure trail formation.

2.3. **Further Reading.**

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3. Social Decision-Making

Background. Evidence accumulation models describe how different organisms integrate information to make choices. The problem of a single agent integrating evidence to decide between two options—with only one correct—has been thoroughly studied in this capacity. In the simplest setting, a Bayesian observer makes a sequence of conditionally independent noisy observations of an environment and computes the probability that the environment is in one of two states. Once their belief crosses one of two thresholds, signifying they accumulated sufficient evidence, they make the analogous decision. The evidence accumulation and decision-making processes can be formulated as a drift-diffusion SDE on a bounded, symmetric domain with absorbing boundaries [11].

The principle issue with current models of evidence accumulation and decision-making is that they describe an observer in isolation, whereas decisions are often made in groups. Stock traders, while not privy to all of their competitor's information, can still observe each other's decisions. It is thus natural to ask how an observer should combine private measurements with social information optimally to make decisions. In the following I provide details of some of my most substantive recent work in this field.

3.1. Heterogeneity Improves Speed and Accuracy of Social Networks.

Consider an all-to-all network, or *clique*, of agents, each deciding between two options. Like day-traders, or strangers in a market, agents make private observations and gather social evidence by observing the choices of all other agents. They do not share private information but know the statistics of the observations each agent makes. A decision cannot be undone.

We assume N agents accumulate noisy private observations and optimally combine them with information obtained from observing the decisions of their neighbors to choose between two hypotheses, H^+ or H^- [39,40]. Either hypothesis is *a priori* equally likely to be correct. Each agent, i, makes decisions based on their *belief*, $y_i(t)$, which equals the log-likelihood ratio (LLR) between the hypotheses given all available evidence [1]. After a sequence of private observations, $\xi_{1:t}^{(i)}$, the belief is $y_i(t) = \log[\mathbb{P}(H^+|\xi_{1:t}^{(i)})/\mathbb{P}(H^-|\xi_{1:t}^{(i)})]$. If private observations are rapid and *conditionally uncorrelated* in time and between agents, beliefs evolve according to

$$dy_i = \pm\alpha dt + \sqrt{2\alpha}dW_i, \tag{8}$$

where the sign of the drift equals that of the correct hypotheses, and $W_i(t)$ are independent, standard Wiener processes [11,66]. Each observer starts with no evidence, so $y_i(0) = 0$. We further assume that H^+ is correct and $\alpha = 1$.

Each agent, i, sets a threshold, θ_i, and chooses H^+ (H^-) at time T_i if $y_i(T_i) \geq \theta_i$ ($y_i(T_i) \leq -\theta_i$), and $y_i(t) \in (-\theta_i, \theta_i)$ for $0 \leq t < T_i$. All other agents observe a decider's choice, but may not know their threshold. We consider *omniscient* agents who know each other's thresholds.

Without loss of generality, we assume the belief of agent $i = 1$ is the first to reach threshold at time $t = T$. Until this decision, beliefs of all agents, $y_i(t)$ with $i = 2, ..., N$, evolve independently according to Eq. (8). Upon observing the first decision, *omniscient* agents update their belief by

[1] Often the belief refers to the posterior probability of the state. The two definitions are closely related as $p(H_-|\xi) = 1/(1 + \exp(y))$.

the evidence independently accumulated by the first decider, $y_i(T) \to y_i(T) \pm \theta_1$ [**?**]. Observing a positive (H^+) first decision causes any belief that satisfies $y_i(T^-) \in [\theta_i - \theta_1, \theta_i)$, to cross the positive threshold, θ_i, evoking a positive decision by agent i. Thus, a wave of a_1 *agreeing* agents follows the first choice. Each of the remaining $N - a_1 - 1$ undecided agents then obtains information by observing who followed the first decision and who remained undecided. How do the undecided agents make use of this newly revealed information?

For homogeneous populations ($\theta_i = \theta$) and sufficiently large N, we find that

$$\mathbb{E}[a_1 | y_1(T) = \pm\theta] \approx \frac{N-1}{2}\Big(1 \pm \frac{\theta}{\sqrt{4\pi \ln N}}\Big). \tag{9}$$

Thus, if the first decider chooses correctly (incorrectly) slightly more (less) than half the population will agree with the first decision. The remaining undecided agents then reveal that their respective private information had them leaning the opposite way before observing the first decision, and this information causes all remaining undecided agents to update their belief by $\frac{\theta^2 N}{2\pi \ln N}$, which is greater than 2θ for sufficiently large N. Thus, all agents will make a decision upon observing the first decision. If the first decision is correct, everyone agrees with them and everyone chooses correctly. If the first decision is incorrect, then about half the clique chooses correctly.

We model heterogeneous populations with a dichotomous population. That is, we take $\theta_i \in \{\theta_{\min}, \theta_{\max}\}$ with $\theta_{\min} < \theta_{\max}$. The parameter γ represents the fraction of agents with threshold $\theta_{\min}$. Surprisingly, we find that such dichotomous populations perform better than homogeneous populations provided γ is tuned optimally. The basic idea is as follows: Agents with $\theta_{\min}$ as their threshold decide hastily leading only other hasty agents to make a potentially ill-advised decision, with $\mathbb{E}[a_1] \approx \frac{\gamma N - 1}{2}\Big(1 \pm \frac{\theta_{\min}}{\sqrt{4\pi \ln \gamma N}}\Big)$. More deliberate agents can weigh this information correctly and thereby make a correct decision.

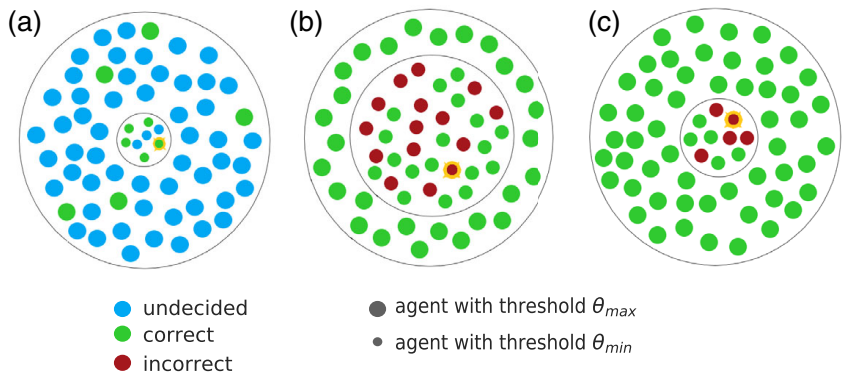

Figure 9. Graphical description of optimal γ value.

A dichotomous clique performs optimally for a particular value of γ. The basic idea is as follows. If γ is small, then not enough information is obtained by observing the first wave of decisions (Fig. 9a). The vast majority of remaining agents are undecided and the time required for everyone to make a decision is long. On the other hand, if γ is too large, then all deliberate agents choose correctly after observing the first decision (Fig. 9b). But too many agents are sacrificed to the wrong decision for this result. These two effects counteract at a critical γ value (Fig. 9c). **Thus, not only do heterogeneous cliques decide more accurately, they also decide more quickly due to the hasty initial decision!**

This result holds for more general θ distributions. For example, taking $\theta \sim U[\theta_{\min}, \theta_{\max}]$ yields similar results (see Fig. 10). Due to the relevance of this work, particularly during election years, we received significant media attention.

3.2. **Further Reading.**

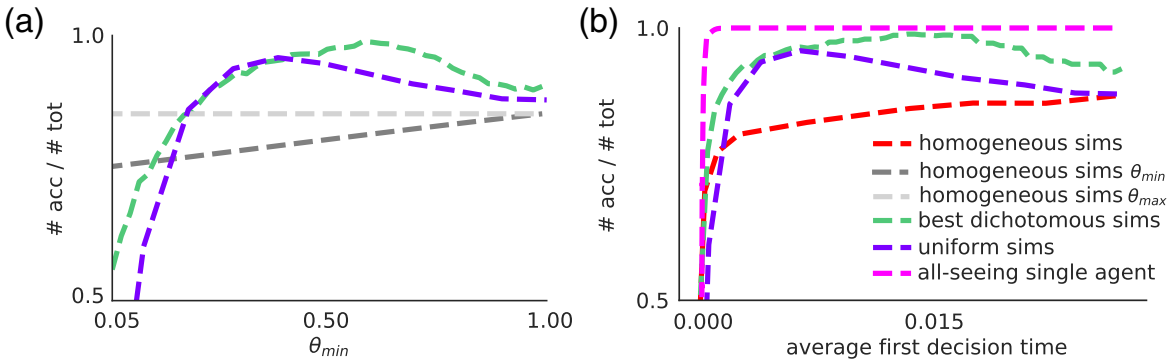

Figure 10. Simulations showing heterogeneous cliques outperforming homogeneous cliques in accuracy and time.

(1) **B. R. Karamched**, S. Stolarczyk, Z. P. Kilpatrick, and K. Josic. Bayesian Evidence Accumulation on Social Networks. SIADS 19 (3) 1884-1919 (2020). (summarized above)
(2) **B. R. Karamched**, M. Stickler, B. Lindner, Z. P. Kilpatrick, W. Ott, and K. Josic. Heterogeneity Improves Speed and Accuracy in Social Networks. Physical Review Letters 125, 218302 (2020)
(3) M. Stickler, W. Ott, Z. P. Kilpatrick, K. Josic, and **B. R. Karamched**. Impact of Correlated Information on Pioneering Decisions. Physical Review Research. 5, 033020. (2023)
(4) S. Linn, S. D. Lawley, **B. R. Karamched**, Z. P. Kilpatrick, K. Josic. Fast decisions reflect biases, slow decisions do not. Physical Review E 110, 024305 (2024).

4. Other Work

Here I briefly summarize other work I've done in each of the subdisciplines.

4.1. **Biomolecular Feedback Systems.** Since 2021, I have modeled biological processes involving biomolecular feedback and developed theory regarding it. In collaboration with *Krešimir Josić* and *Will Ott*, I developed a stochastic model of synthetic *E. coli* cells in spatially extended domains and showed that if two types of cells communicate via positive feedback, they dynamically alter their spatiotemporal patterns [69]. Building on this, with *Shawn Ryan*, I developed a PDE-DDE model demonstrating that synchronization of oscillations in protein concentrations in synthetic *E. coli* across a large spatial domain was facilitated by positive feedback loops coupled with negative feedback loops. The delay here captured the time for protein production and diffusion across the domain [32]. I developed a model of neuronal polarization—the process by which symmetrically arranged protrusions from the unpolarized neuron suddenly break symmetry and choose exactly one of their protrusions to be the axon (electrical signal propagator) and the rest to be dendrites (signal receivers) [7]. Analysis involved understanding how additive noise facilitated transfer of a particle between potential wells in a positive feedback system. Our model was the first rigorous theoretical formulation of the neuron polarization problem. These were done in collaboration with *Richard Bertram* and my first Ph. D. student, *Fan Bai*. Furthermore, with *Will Ott* and *Dave Albers*, I investigated the role delay plays in onset of chaos in a paradigmatic glucose-insulin model. We followed up by investigating some clinical implications [37, 42]

4.2. **Lattice Models.** With my student *Fan Bai* and *Richard Bertram*, I developed detailed biophysical models of motor transport to bring to light how interactions of the motors themselves at the subneuronal level affect length homeostasis in the neurons [6]. The motor interactions were captured via nonlinear PDEs derived from a Totally Asymmetric Simple Exclusion Process (TASEP).

Our model coincided with two previously published experimental reports and predicted that a traffic jam of motors would cause neuronal length to grow. With *David Dingli* and *Claudia Neuhauser*, I developed a lattice model to investigate spatial dynamics of oncolytic virotherapy—a relatively novel therapeutic for treating cancerous tumors [44]. With *Krešimir Josič* and *Will Ott*, I devised a lattice model to demonstrate how multi-strain microbial consortia with distinct shapes for each strain can be used to control spatiotemporal patterns [69].

4.3. **Social Decision-Making.** In follow up papers from what is described above, we developed a rigorous model of decision-making when information is correlated, which is more pertinent to real world settings [60]. Thereafter, we developed our models for including initial bias from the observers [48]. Each model has been an incremental improvement on classical evidence accumulation models, with each becoming more pertinent to real life. These models allow for comparison with data from social network sites and can divulge where humans deviate from optimality. This work was done with *Zachary Kilpatrick*, *Krešimir Josič*, and *Will Ott*.

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