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Spiral formation and degeneration in heterogeneous excitable media

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Spontaneous spiral formation occurs when an excitation wave is input to a heterogeneous network of low- and high-light-intensity cells projected onto a light-sensitive Belousov-Zhabotinsky reaction. The range of network conditions where spirals form is increased if two waves are input at critical time intervals. Spirals degenerate to form multiple spirals and spirals trapped within excitable cells. Spiral formation and degeneration is dependent on network excitability, cell size, and network size. Results exhibit parallels with spiral formation in excitable biological systems such as the heart.

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Mechanisms of spiral formation and degeneration play an important role in biology where they are implicated in cardiac arrhythmias and fibrillation [1,2]. Diverse theories exist concerning spiral wave formation in the heart, but many are based around a dispersion of refractoriness [3] in the excitable tissue coupled with a critically timed premature pulse [2].

Spontaneous spiral formation was observed in homogeneous Belousov-Zhabotinsky (BZ) reactions where waves propagate into the vulnerable region of preceding waves [4,5] and where waves interact with impenetrable nonexcitable obstacles [6,7]. We report the same phenomena in a BZ system where light intensity is utilized to impose a controllable heterogeneous network. In our system cell boundaries are permeable to diffusion, providing strong parallels with heterogeneous biological networks.

We used a light-sensitive BZ system [8] with $Ru(bipyridyl)_3^{2+}$ catalyst immobilized on silica gel [9,10]. At the solution composition [11] the medium is oscillatory in the dark. A controllable network of excitable and nonexcitable cells is created by projecting low- (Φ_1) and high- (Φ_2) light-intensity cells (10×10 checkerboard pattern) onto the gel surface ([12,13] and Fig. S1 in Ref. [10]). Numerical studies were carried out using the Oregonator model modified to account for photochemistry, where Φ represents the photoinduced $[Br^-]$ that controls excitability [10,14].

Waves are initiated in an oscillating region (model, $\Phi=0$; experiment, an area masked from projected light) positioned a distance $2x$ (x=spatial resolution of network) below the network [Fig. 1(k)]. If a single wave is input to the network, the behavior is dependent on network excitability. When both Φ_1 and Φ_2 are close to the subexcitable limit (experiment ~5.2 mW cm^{-2}; model ~0.0355), excitability is low and spirals form spontaneously [Figs. 1(a) and 1(f)]. If network excitability is increased, fragmented waves propagate through the network junctions formed by the corners of adjacent excitable cells. Reducing the coupling between cells also induced spiral formation in monolayers of heart cells [15].

If two waves are sequentially introduced to the network, the behavior is dependent on both network excitability and the time interval between the waves. If the time interval is large (>30 s in experiment) two fragmented waves propagate across the network [Figs. 1(b) and 1(g)]. With short time intervals (<20 s in experiment), the second wave fails to propagate. However, spirals form when the time interval is between these two points [Figs. 1(c)–1(e) and 1(h)–1(j); movies S1 and S5 in [10]]. Importantly, spirals form from two waves at network excitability levels where one wave maintained stability. Spiral formation occurs as the first wave induces increased heterogeneity in the network in terms of refractoriness. This increases the chance of subsequent waves reaching junctions at critical points prior to them regaining full excitability. Similar behavior is observed in the heart where only pulses delivered within a "vulnerable region" before the tissue regains full excitability cause "unidirectional blocks" and spiral formation [2,3]. This vulnerable region has previously been shown to exist in homogeneous BZ systems [4].

In our experiments, spirals and multiple spirals have their tips pinned around nonexcitable cells. In the heart, reentrant spiral waves become pinned to small anatomical obstacles, leading to tachycardia [1,16]. The resolution of certain multiple spirals in experiment and model gives spiral waves trapped within one excitable cell [Figs. 2(a), 2(c), and 2(e); movies S2 and S4 in [10]]. As the rotational period of these spirals is shorter than the original multiple spirals, they quickly dominate the network dynamics, giving a proliferation of spiral wave fragments (Fig. S2 in Ref. [10]). The results are qualitatively similar to tachycardia and degeneration to fibrillation in the heart [1–3].

By changing Φ the effect of network heterogeneity was investigated more thoroughly in numerical simulations. In experiments at lower excitability levels, wave transfer was disrupted by additional heterogeneity and the effect of changing light levels could not be mapped reproducibly. Figure 3 shows a phase diagram of the wave behavior for sampled combinations of Φ_1 and Φ_2 ($\Delta\Phi=\Phi_2-\Phi_1 \geqslant 0.0005$). In the region SE the system is weakly excitable and unbounded wave fragments form. At the boundary between fragmented wave propagation (F) and nonpropagation (N) exists region D, where wave fragments travel diagonally after crossing the first junction. At specific Φ_1 and Φ_2 values within D spirals form (S). Therefore, spiral formation is confined to a narrow region adjacent to region SE. As the mean of Φ_1 and Φ_2 is in region SE we can conclude that spirals form in networks with weakly excitable junctions.

The effect of cell size was studied [Figs. 4(a) and 4(b)]. Decreasing cell size but maintaining excitability causes a

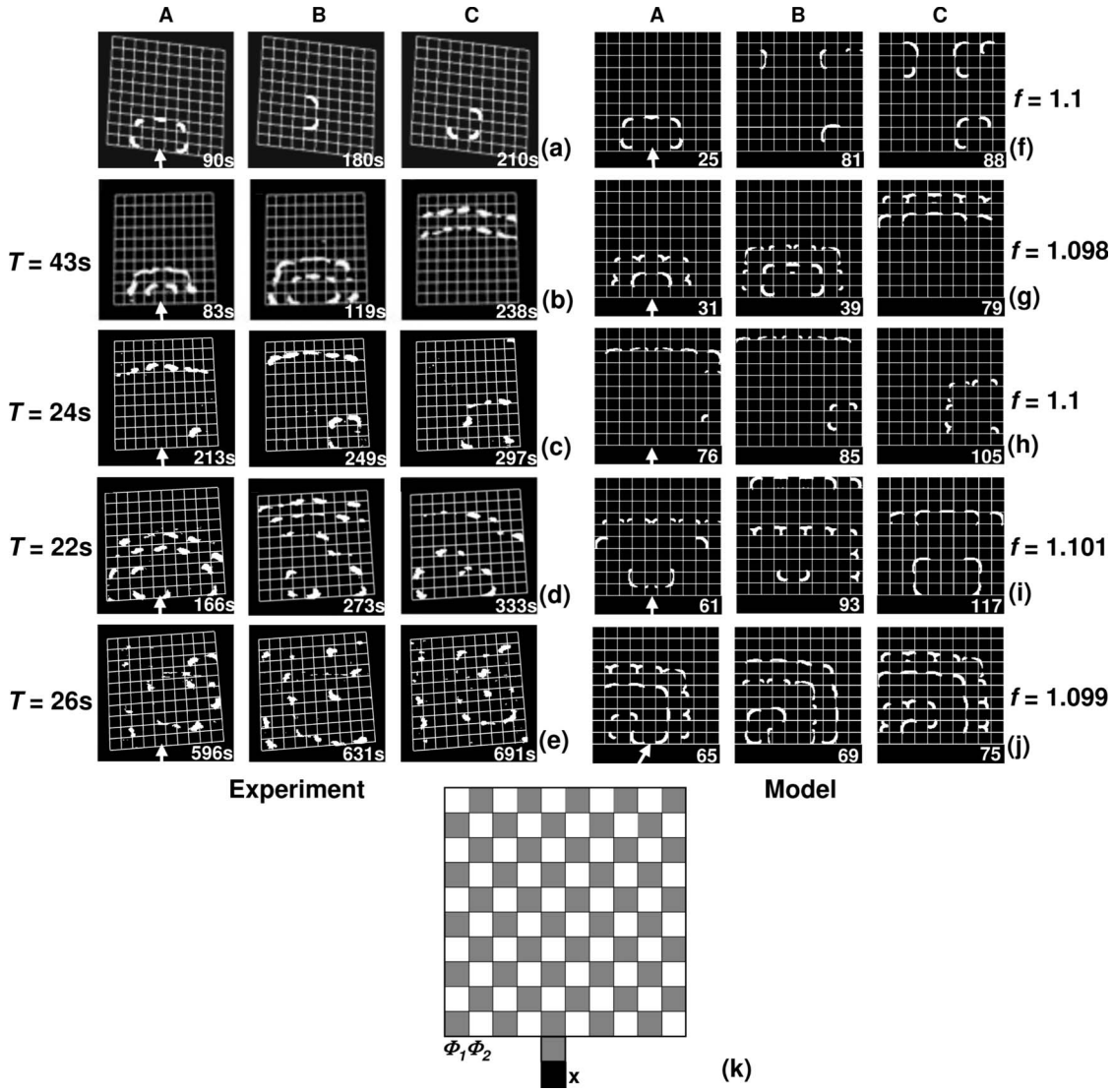

FIG. 1. Spiral formation resulting from one wave input to the network (a),(f) (experiment, $\Phi_1=1.3$, $\Phi_2=8.6$ mW cm^{-2}; model, $\Phi_1=0.0285$, $\Phi_2=0.0409$). Behavior when two waves are input to the network with time interval (T). (Experiment, $\Phi_1=0.394$, $\Phi_2=9.97$ mW cm^{-2}; model, $\Phi_1=0.027$, $\Phi_2=0.042$). In the model waves with different time intervals are initiated by altering the stoichiometric factor f of the entire medium. Two stable waves (b),(g), and single (c),(h), double (d),(i), and multiple spirals (e),(j). Projected checkerboard pattern (k). Wavefronts are shown in white [high concentration of oxidized catalyst $Ru(bpy)_3^{3+}$]. Arrows show the location and direction of initiation, numbers show time after initiation [seconds or dimensionless time units (dtu)]. See also movies S1–S3 in [10].

transition from stable wave propagation → spiral formation → diagonal propagation → wave propagation failure [Fig. 4(b)]. Because cell size controls the relationship between the rotational period of the spiral and the refractory period of the junctions, spirals form at limited cell sizes when compared to other traveling waves. In very small cells, wave curvature reaches a critical value, meaning propagation fails (eikonal equation [17]). Increasing cell size reduces wave curvature and the inhibitory effect of neighboring nonexcitable cells, making junction transition progressively more favorable. In a system incorporating two junctions, the effects of junction geometry and excitability on wave curvature were used to impart a "diodelike" characteristic [18].

Wave transition is favored in networks with lower heterogeneity. If cell size is constant but $\Delta\Phi$ is increased, a progressive change from stable propagation to nonpropagation is observed. This highlights the critical role that nonexcitable cells play in controlling junction transition and occurs because effective junction size (permeability) is controlled by the degree that waves can overlap nonexcitable cells.

In heterogeneous networks where wave transfer is junction controlled spirals may be formed as follows. The original wave input to the network becomes fragmented when it cannot pass through nonexcitable cells. To maintain stability waves break, move around nonexcitable cells (via corner to corner junctions), and rejoin in a collision. As the distance from the initiation site increases, different sequences of junctions exert differential changes in velocity and direction that alter the effective path length to the collision site, and collisions become increasingly disproportionate. Waves must also move through a series of junctions at right angles to the direction of travel (apex of the wave) in order to maintain

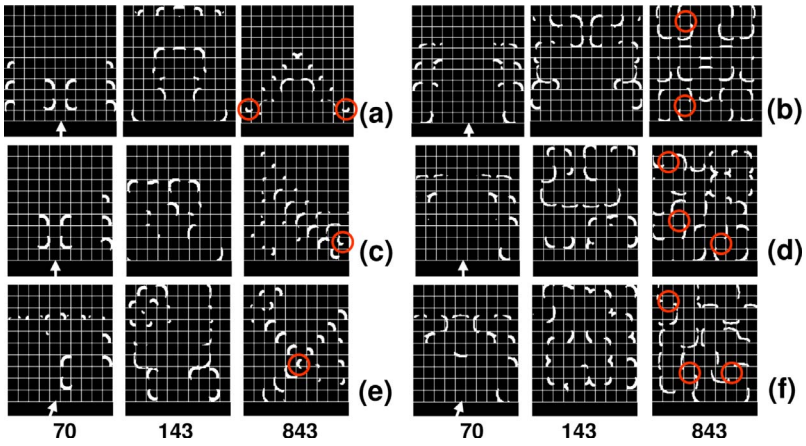

FIG. 2. (Color online) Numerical results showing spiral degeneration in 11×11 (a),(b) and 10×10 (c)–(f) networks, cell size 18. Orthogonal (a)–(d) and nonorthogonal initiation (e),(f) (shown by arrows) ($f=1.1$). (a), (c), (e) $\Phi_1=0.0245$ and $\Phi_2=0.0465$; (b), (d), (f) $\Phi_1=0.028$ and $\Phi_2=0.039$. Images captured at 70, 143, and 843 dtu. Circles indicate spirals trapped in excitable cells (a),(c),(e) and two opposed fragments rotating around a nonexcitable cell (b),(d),(f). See also movies S4 and S5 in [10].

stability. Right-angled junction transition is less stable as lower amounts of activator diffuse through the junction. This is because only the free ends of the wave that possess high curvature sweep past the junction. However, if junctions are opposite the waves' apex where curvature is lower, then higher amounts of activator diffuse and junction transition is stable. Thus diagonal transition caused by unilateral failure of right-angled junctions is more commonly observed than spiral formation (caused by the failure of selected right-angled junctions). Spirals form when the wave becomes disconnected and fragments propagate back through junctions where forward propagation failed. The junctions retain their excitability or have short refractory periods because complete forward propagation failed. As the disconnected wave moves further through the network small excitation waves, on the threshold between expansion and collapse, form at certain junctions and drift large distances across cells before eventually expanding. The resulting waves possess very high curvature and almost form spirals within excitable cells. However, the spiral core is too large and the tip collides with the vacant (due to disconnection of the original wave) lower junction to form spirals with trajectories pinned around adjacent nonexcitable cells (see Fig. S3 and movie S3 in [10]).

In small networks (e.g., 5×5) spiral formation is always stable resulting in double spirals. However, in larger networks spirals spontaneously degenerate, forming complex interacting spirals (Fig. 2). This occurs when the trajectories of spirals overlap and fragments are incident at junctions with short time intervals causing additional junction failures. In Figs. 2(b), 2(d), and 2(f) the system degenerates to a point where two spiral fragments rotate around a single nonexcitable cell. In networks with higher $\Delta\Phi$, degeneration may lead to spiral waves trapped in excitable cells [Figs. 2(a), 2(c), and 2(e)]. In these networks Φ_2 is higher so cell wall permeability is low. However, Φ_1 is lower so cells are more excitable and wave fragments possess higher curvature [19]. The size of a spiral's core also decreases with increasing excitability [20]. When fragments are repeatedly incident at certain junctions in the network, near junction failure occurs and critical excitation waves form (model, $u\sim0.1965$). In networks with higher $\Delta\Phi$ these critical excitation waves can expand and curl back to form spirals trapped within excitable cells [Figs. 2(a), 2(c), and 2(e); Fig. S2 and movies S2 and S4 in [10]]. Because they form large numbers of closely spaced fragments their mechanism of formation has an increasing chance of being repeated at other points in the network and they self-proliferate. Long simulations show the formation of increasing numbers of these spirals.

In conclusion, spiral waves are generated spontaneously when a heterogeneous network is applied to the BZ reaction. The behavior is sensitively dependent on network excitability, network size, and cell size. Spirals form spontaneously

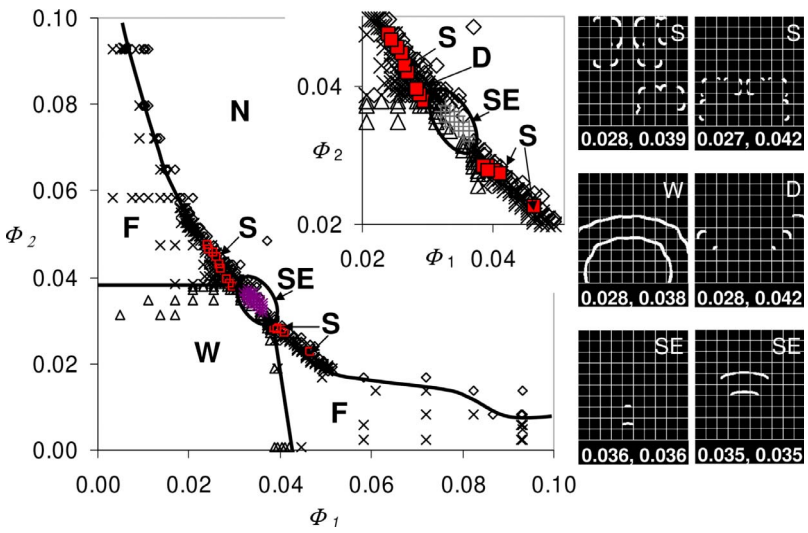

FIG. 3. (Color online) Phase diagram of sampled combinations of Φ_1 and Φ_2 in numerical simulation ($f=1.1$). ×, fragmented wave propagation (F); △, wave does not fragment (W); ◇, wave does not propagate (N); at the boundary between F and N waves fragment but propagate only diagonally (D); □, spiral formation (S); +, unbounded wave fragments (SE). Images illustrate typical wave behaviors; numbers are Φ_1,Φ_2.

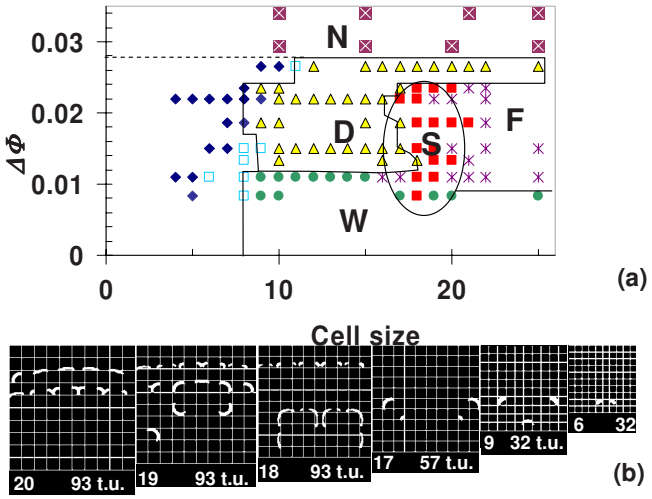

FIG. 4. (Color online) (a) Effects of cell size and $\Delta\Phi$ on wave behavior. ■, spiral (S); ▲, diagonal propagation (D), ∗, fragmented wave (F); ●, continuous wave (W); ☒, no propagation (N); □, diagonal propagation of first wave, second wave annihilated; ◆, both waves fail after crossing first junction. (b) Wave behavior with decreasing cell size. $\Phi_1=0.027$, $\Phi_2=0.042$, $\Delta\Phi=0.015$. Numbers on images show cell size and time after initiation (dtu).

from one wave if network excitability is low and junctions are weakly excitable. Spiral formation can occur at higher network excitability levels if two waves are input to the network with a certain time interval. A mechanism of spiral formation was identified based on complete unidirectional failure of selected junctions. Small excitation waves possessing high curvature formed from the disconnected wave. This gave spirals with their trajectories pinned around nonexcitable cells. In larger networks these spirals degenerated to form a proliferation of interacting spiral fragments. In networks with higher $\Delta\Phi$, this degeneration process leads to critical wave fragments which expand to form spirals trapped within excitable cells.

Our study has shown that spiral formation is in a narrow parameter range (cell size, excitability), indicating that orderly propagation in excitable media is quite robust against heterogeneous conditions. The results reported exhibit parallels with excitable biological systems such as the heart, where a coupling of critical spacing and spatial heterogeneity leads to spiral formation and degeneration [2,3].

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