Stable Irregular Dynamics in Complex Neural Networks

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Irregular dynamics in multidimensional systems is commonly associated with chaos. For infinitely large sparse networks of spiking neurons, mean field theory shows that a balanced state of highly irregular activity arises under various conditions. Here we analytically investigate the microscopic irregular dynamics in finite networks of arbitrary connectivity, keeping track of all individual spike times. For delayed, purely inhibitory interactions we demonstrate that any irregular dynamics that characterizes the balanced state is not chaotic but rather stable and convergent towards periodic orbits. These results highlight that chaotic and stable dynamics may be equally irregular.

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Highly irregular dynamics is a prominent feature of multidimensional complex systems and often attributed to chaos [1,2]. Networks of spiking neurons, which interact by sending and receiving electrical pulses (spikes), exhibit very irregular dynamics for a wide range of conditions [3–8]. For instance, networks of sparse random connectivity may display a balanced state [3] in which excitatory (positive) and inhibitory (negative) inputs to each neuron balance on average and only the fluctuations create spikes at irregular times [3,4]. Their dynamics resemble random (e.g., Poisson) processes with low correlations across different neurons. Mean field theory [4] shows that such irregular balanced activity occurs in networks with excitatory and inhibitory recurrent feedback as well as in networks that receive external excitatory inputs and exhibit recurrent inhibition only [4,5]. Interestingly, for inhibitory networks recent work [9,10] suggests that this irregular dynamics is stable in globally coupled and slightly diluted networks without delay. In particular, Zillmer *et al.* [10] numerically measured a negative maximal Lyapunov exponent of the irregular, seemingly chaotic spiking dynamics.

In this Letter, we analytically investigate the microscopic spiking dynamics in finite neural networks with delayed inhibitory interactions [11] and arbitrarily complicated connectivity [Fig. 1(a)]. We show that any spiking dynamics characterizing the irregular balanced state in any of the above networks is stable, i.e., two close-by trajectories converge towards each other over time. Moreover, we show that any generic periodic orbit is stable [13]. In particular, this excludes generic unstable periodic orbits and indicates that the typical irregular dynamics is not chaotic in any of these systems.

Consider N neurons that interact on a directed network by sending and receiving spikes. The subthreshold dynamics of the neurons' membrane potentials $V_i(t)$ at time t are given by

$$\frac{d}{dt}V_i = f_i(V_i) + \sum_{j=1}^{N}\sum_{k\in\mathbb{Z}} \varepsilon_{i,j}\delta(t - t^s_{j,k} - \tau_{i,j}), \qquad (1)$$

where a smooth function f_i specifies the internal dynamics, $\varepsilon_{i,j} \leq 0$ is the inhibitory coupling strength and $\tau_{i,j} > 0$ the delay time of a synaptic interaction from neuron j to neuron i, and $t^s_{j,k}$ determines the time of the kth spike sent by neuron j. Here $\delta(\cdot)$ is the Dirac delta function. If a neuron j reaches the threshold potential, $V_j(t^-) = V_{\Theta,j}$, it generates a spike at $t =: t^s_{j,k}$ for some k and is reset, $V_j(t^s_{j,k}) = 0$. We require here that the f_j for all j satisfy $f_j(V_j) > 0$ and $f'_j(V_j) < 0$ for all $V_j \leq V_{\Theta,j}$ such that in isolation each neuron exhibits oscillatory dynamics. Sending and receiving of spikes are the only events occurring in these systems that interrupt the continuous time evolution.

The neurons are equivalently described [14] by a phase-like variable $\phi_j(t) \in (-\infty, \phi_{\Theta,j}]$ satisfying the linear differential equation

$$d\phi_j/dt = 1 \qquad (2)$$

at all nonevent times. Upon reaching a phase threshold $\phi_j(t^{s-}_{j,k}) = \phi_{\Theta,j}$, this phase is reset, $\phi_j(t^s_{j,k}) = 0$, and a spike is generated. After a delay time $\tau_{i,j}$ that spike is received by postsynaptic neuron i of neuron j and its phase changes according to

$$\phi_i(t^s_{j,k} + \tau_{i,j}) = H^{(i)}_{\varepsilon_{i,j}}(\phi_i((t^s_{j,k} + \tau_{i,j})^-)). \qquad (3)$$

This interaction is mediated by the transfer function

$$H^{(i)}_{\varepsilon}(\phi) = U_i^{-1}[U_i(\phi) + \varepsilon], \qquad (4)$$

where $U_i(t)$ is the free (all $\varepsilon_{i,j} = 0$) solution of (1) through the initial condition $U_i(0) = 0$, yielding $U'_i > 0$ and $U''_i < 0$, and $\phi_{\Theta,j} = U_j^{-1}(V_{\Theta,j})$; cf. [15]. We remark that for these systems there is a one-to-one mapping between the potential representation (1) and the phase representation (2)–(4) that is specified by $V_i = U_i(\phi_i)$; cf. also [14]. For

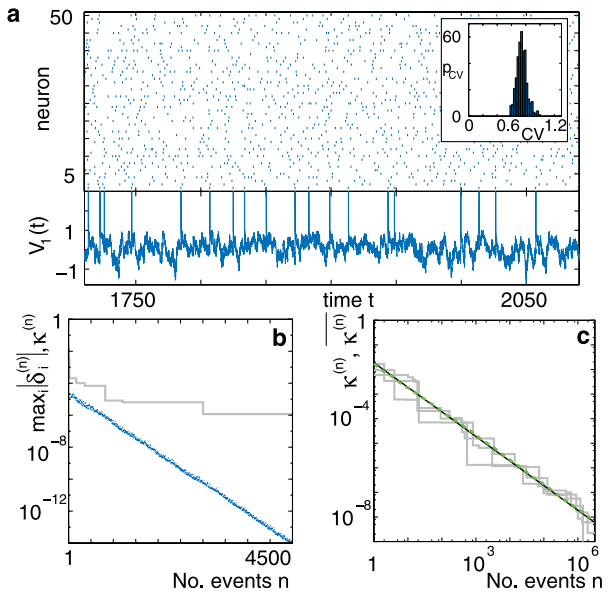

FIG. 1 (color). Stable irregular dynamics in a random network ($N = 400$, $\gamma_i^{-1} \equiv 1.0$, $I_i \equiv 4.0$, $\phi_{\Theta,i} \equiv 1$, $\tau_{i,j} \equiv 0.1$, connection probability $p = 0.2$, $\sum_j \varepsilon_{i,j} \equiv -16$). (a) Upper panel displays the spiking times (blue lines) of the first 50 neurons. Lower panel displays the membrane potential trajectory of neuron $i = 1$ (spikes of height $\Delta V = 2$ added at firing times). Inset shows a histogram of the coefficients of variation $CV_i := \sigma_i/\mu_i$, $\mu_i := \langle t^s_{i,k+1} - t^s_{i,k}\rangle$, $\sigma_i^2 := \langle (t^s_{i,k+1} - t^s_{i,k} - \mu_i)^2\rangle$ averaged over time. (b) Exponential decay of the maximal perturbation $\max_i|\delta_i^{(n)}|$ (blue dots) and the minimal margin $\kappa^{(n)}$ (gray line) for one given microscopic dynamics. (c) Algebraic decay of the average minimal margin, $\overline{\kappa^{(n)}}$ (green dashed line, averaged over 250 random initial conditions), and the analytical prediction (no free fit parameter) of $\overline{\kappa^{(n)}}$ (black solid line). We also show the minimal margin $\kappa^{(n)}$ for three exemplary initial conditions (gray lines), including that of (b).

instance, for standard leaky integrate-and-fire neurons, where $f_i(V) = I_i - \gamma_i V$ with time scale $\gamma_i^{-1} > 0$ and equilibrium potential $\gamma_i^{-1} I_i > V_{\Theta,i}$, we have $U_i(\phi) = \gamma_i^{-1} I_i (1 - \exp(-\gamma_i \phi))$. Whereas the analysis below is valid for general $U_i(\phi)$, all numerical simulations are presented for leaky integrate-and-fire neurons. In the following we consider arbitrary generic spike sequences in which all neurons are active [i.e., there is a finite constant $T > 0$, arbitrarily large, such that in every time interval $[t, t + T)$, $t \in \mathbb{R}$, every neuron fires at least once] and no two events occur at the same time.

Highly irregular spiking sequences [cf. Fig. 1(a)] constitute a typical form of activity in these networks, suggesting that the underlying dynamics may be chaotic. However, as we show below for networks of arbitrary connectivity, this dynamics generically is stable and not chaotic. To show this, we first analytically study the exact microscopic dynamics of an original trajectory, as defined by the (arbitrarily irregular) sequence of events generated by the network, and a slight perturbation to it that keeps the order of events as in the original.

The time of the nth event (sending or receiving) occurring in the entire network is denoted by t_n in the original sequence, and by $\tilde{t}_n$ in the perturbed sequence. Here simultaneous reception of the same spike at different neurons constitutes one event. Analogously, at a given time t, we denote the phases of neuron i by $\phi_i(t)$ and $\tilde{\phi}_i(t)$, respectively. Let

$$\Delta_i^{(n)} = [\phi_i(t_n) - \tilde{\phi}_i(\tilde{t}_n)] - (t_n - \tilde{t}_n) := \delta_i^{(n)} - \delta t^{(n)} \quad (5)$$

denote the difference of the phases of neuron i between the two sequences after the nth and before the $(n + 1)$st event, corrected for the time shift $\delta t^{(n)} = t_n - \tilde{t}_n$ between the sequences. If at the $(n + 1)$st event some neuron j^* sends a spike, the phase shifts

$$\Delta_i^{(n+1)} = \Delta_i^{(n)} \quad (6)$$

of all neurons i stay unchanged. Because of the linear phase dynamics (2) between the spikes, $\Delta_{j^*}^{(n+1)} = \Delta_{j^*}^{(n)} = -\delta t^{(n+1)}$ also specifies the temporal shift of the $(n + 1)$st event. At some $t_l = t_{n+1} + \tau_{i,j^*}$ postsynaptic neuron i receives the spike sent by j^*. The resulting phase shifts are given by

$$\Delta_i^{(l)} = H^{(i)}_{\varepsilon_{i,j^*}}(\phi_i(t_l^-)) - H^{(i)}_{\varepsilon_{i,j^*}}(\tilde{\phi}_i(\tilde{t}_l^-)) - \delta t^{(l)}, \quad (7)$$

where $\phi_i(t_l^-) = \phi_i(t_{l-1}) + t_{n+1} + \tau_{i,j^*} - t_{l-1}$ and $\tilde{\phi}_i(\tilde{t}_l^-) = \tilde{\phi}_i(\tilde{t}_{l-1}) + \tilde{t}_{n+1} + \tau_{i,j^*} - \tilde{t}_{l-1}$ are the phases just before spike reception. Using the identities $\phi_i(t_l^-) = \tilde{\phi}_i(\tilde{t}_l^-) + \Delta_i^{(l-1)} + \delta t^{(n+1)}$ and $\delta t^{(l)} = \delta t^{(n+1)} = -\Delta_{j^*}^{(n)}$, we apply the mean value theorem to Eq. (7) and obtain

$$\Delta_i^{(l)} = c_i^{(l)} \Delta_i^{(l-1)} + (1 - c_i^{(l)}) \Delta_{j^*}^{(n)}, \quad (8)$$

where $c_i^{(l)}$ is given by the derivative $c_i^{(l)} = dH^{(i)}_{\varepsilon_{i,j^*}}(\phi)/d\phi$ for some ϕ between $\phi_i(t_l^-)$ and $\tilde{\phi}_i(\tilde{t}_l^-)$. If neuron j^* is not connected to neuron i, $\varepsilon_{i,j^*} = 0$, the function $H^{(i)}_{\varepsilon_{i,j^*}}(\phi) = H_0^{(i)}(\phi) = \phi$ is the identity map, such that the phase shift stays unchanged, $\Delta_i^{(l)} = \Delta_i^{(l-1)}$; indeed $c_i^{(l)} = dH_0^{(i)}(\phi)/d\phi = 1$, independent of ϕ. If neuron j^* is connected to i we find $c_i^{(l)}$ bounded by

$$c_{\mathrm{mn}} := \inf_{\phi,k}\{(H^{(k)}_{\varepsilon_{mn}})'(\phi)\} \le c_i^{(l)} \le \sup_{\phi,k}\{(H^{(k)}_{\varepsilon_{mx}})'(\phi)\} =: c_{\mathrm{mx}}, \quad (9)$$

where $\varepsilon_{\mathrm{mx}} = \max_{i,j:\varepsilon_{i,j}\neq 0}\{\varepsilon_{i,j}\}$ and $\varepsilon_{\mathrm{mn}} = \min_{i,j:\varepsilon_{i,j}\neq 0}\{\varepsilon_{i,j}\}$. The phases are confined to certain finite intervals, $\phi_i \in [\phi_{\mathrm{mn}}, \phi_{\Theta,i}]$, which depend on the network parameters. Given that $dH^{(i)}_{\varepsilon}(\phi)/d\phi = U_i'(\phi)/U_i'(U_i^{-1}(U_i(\phi) + \varepsilon))$ and using the monotonicity $U_i' > 0$ and concavity $U_i'' < 0$, we find $c_{\mathrm{mn}} > 0$ and $c_{\mathrm{mx}} < 1$, independent of the sequence and of the network realization (including its connectivity). Thus the phase shift after receiving a spike is a weighted average of earlier shifts.

Consider that the perturbed sequence differs from the original one by perturbations of the phases of all neurons

and the sending times of all spikes sent but not received at time $t = 0$. (We denote the maximum of these perturbations after the event at t_n by $\Delta^{(n)}_{\mathrm{mx}}$ and the minimum by $\Delta^{(n)}_{\mathrm{mn}}$.) Since the maximum phase shift cannot increase and the minimum cannot decrease according to (8) and (9), the dynamical trajectories are Lyapunov stable. In particular, it follows that generic irregular dynamics cannot be unstable and thus not chaotic.

For strongly connected networks [16] where every neuron can be reached by following a sequence of presynaptic connections from any other neuron, the averaging (8) that yields Lyapunov stability leads also to exponential convergence between the perturbed and original trajectory: Briefly, the relations (8) and (9) already imply that the maximum perturbation cannot increase and the minimum cannot decrease. So the difference $\Delta^{(n)}_{\mathrm{mx}} - \Delta^{(n)}_{\mathrm{mn}}$ decreases (by a factor smaller than 1 dependent on $c_{\mathrm{mx}} < 1$ and $1 - c_{\mathrm{mn}} < 1$) or it stays unchanged during an event. For the perturbation to stay unchanged when neuron i receives a spike from neuron j^*, both its previous perturbation $\Delta^{(l-1)}_i$ and the perturbation $\Delta^{(n)}_{j^*}$ of the sending neuron at sending time need to be identical, i.e., $\Delta^{(l)}_i = \Delta^{(l-1)}_i = \Delta^{(n)}_{j^*}$ in Eq. (8). Following this argument throughout the network (to the presynaptic neurons of the presynaptic neurons, etc.) implies that before some finite time the perturbations of all neurons must have been identical. Because of time translation invariance, a globally identical (trivial) perturbation is equivalent to a time shift of the original orbit. So any nontrivial perturbation converges to a trivial one because the difference $\Delta^{(n)}_{\mathrm{mx}} - \Delta^{(n)}_{\mathrm{mn}}$ converges to zero exponentially. Thus the considered trajectory is exponentially asymptotically stable; cf. also [5,17].

A main condition for stability of trajectories was that the order of events stays the same in the perturbed and original trajectories, a condition that was already found useful to assume in globally coupled systems [9]. In the system considered here, for arbitrary generic spike sequences, there is a nonzero perturbation size keeping the order unchanged in any *finite* time interval. However, the requirement of an unchanged event order yields more and more conditions over time such that the allowed size of a perturbation could decay more quickly with time than the actual perturbation. This will be excluded if the temporal *margin* $\mu^{(n)}$ (cf. also [9]) between two subsequent potential future events stays larger than the dynamical perturbation for *infinite* time. Specifically, after time t_n denote the kth potential future event time (of the original trajectory) that would arise if there were no future interactions by $\theta_{n,k}$, $k \in \mathbb{N}$, and the temporal *margin* by $\mu^{(n)} := \theta_{n,2} - \theta_{n,1}$. A sufficiently small perturbation, satisfying $\Delta^{(n)}_{\mathrm{mx}} - \Delta^{(n)}_{\mathrm{mn}} < \mu^{(n)}$, cannot change the order of the $(n+1)$st event.

This directly implies that all generic *periodic orbits* (all those with nondegenerate event times t_n) consisting of a finite number of P events are stable because there is a minimal margin

$$\kappa^{(P)} := \min_{n\in\{1,\ldots,P\}} \mu^{(n)} > 0. \tag{10}$$

To further analyze stability properties of *irregular nonperiodic spike sequences*, we consider the minimal margin $\kappa^{(n)}$ over the first n events. For simplicity, we here consider delays τ_{ij} independent of i. In the balanced regime the spiking activity is well modeled by a Poisson point process [4]. Assuming that, along with the irregular dynamics, the temporal margins are generated by a Poisson process with rate ν, the distribution function of margins is given by $P(\mu^{(n)} \le \mu) = 1 - e^{-\nu\mu}$. The probability that the minimal margin $\kappa^{(n)}$ after n events is smaller or equal to μ is determined by the probabilities that not all individual margins $\mu^{(n)}$ are larger than μ such that

$$P(\kappa^{(n)} \le \mu) = 1 - \prod_{m=1}^{n} P(\mu^{(m)} > \mu) = 1 - e^{-n\nu\mu} \tag{11}$$

with density $\rho_n(\mu) := dP(\kappa^{(n)} \le \mu)/d\mu = n\nu\exp(-n\nu\mu)$. This implies an algebraic decay with the number n of events for the expected minimal margin

$$\overline{\kappa^{(n)}} = \int_0^\infty \mu\rho_n(\mu)d\mu = (n\nu)^{-1} \tag{12}$$

that depends only on the network event rate ν and is independent of the specific network parameters. Numerical simulations [cf. Fig. 1(c)] confirm this *algebraic* decay (12) of the expected minimal margin with the number of network events n. Similarly, we find that the distributions of the margins $\mu^{(n)}$ are indeed exponential such that the margins' fluctuations are sufficiently small [17]. Together

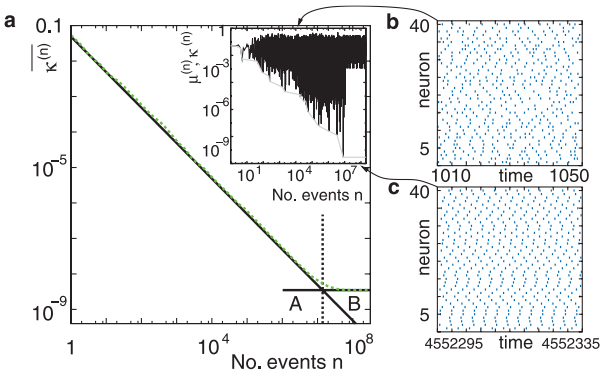

FIG. 2 (color). Convergence towards a periodic orbit in a random network ($N = 40$, $\gamma_i^{-1} \equiv 1.0$, $I_i \equiv 3.0$, $\phi_{\Theta,i} \equiv 1.0$, $\tau_{i,j} \equiv 0.1$, $p = 0.2$, $\sum_j \varepsilon_{i,j} \equiv -3.3$). (a) The average minimal margin $\overline{\kappa^{(n)}}$ [as in Fig. 1(c)] decays as a power law (region A) and saturates after about 10^7 events (region B) when the periodic orbit is reached. Inset: Margin $\mu^{(n)}$ (black) and minimal margin $\kappa^{(n)}$ (gray) for a trajectory started from one specific initial condition. The margin $\mu^{(n)}$ fluctuates strongly on the transient; after the sequence becomes periodic the minimal margin $\kappa^{(n)}$ does not decrease further for future events n. (b),(c) Snapshots of (b) irregular spike sequences after $n \approx 2 \times 10^4$ events on the transient and (c) after $n \approx 10^8$ events on the periodic orbit.

with the *exponential* decay of dynamical perturbations in strongly connected networks this indicates that for a sufficiently small perturbation the order of events stays unchanged for *infinite* time.

Interestingly, arbitrary irregular spike sequences converge to a periodic orbit after finite time (cf. Fig. 2) because (i) there is some finite number E such that two sequences that share the same order of E events are equally ordered for all future events because any initial difference decays exponentially and (ii) there is only a finite number of orderings of events in a finite network such that a given sequence of length E repeats after finite time. Nevertheless, we find that the transient time until a periodic orbit is reached rapidly increases with network size N and with the interaction strengths, in agreement with observations in Ref. [10]; cf. also [17].

In summary, we analytically studied the microscopic dynamics of inhibitory networks of spiking neurons with arbitrarily complicated connectivity and delayed interactions. We showed that all generic trajectories in the balanced regime are stable, even though they generate highly irregular dynamics. Curiously, the assumption of events generated by a maximally irregular (Poisson) random process led us to show stability of the deterministic trajectories.

These results analytically confirm recent numerical findings [10] that irregular dynamics in spiking neural networks may exhibit stable behavior. Moreover, as for globally coupled systems without delay [9] our results show that also networks with more complex structure and delayed interactions exhibit stable periodic orbits. However, highly irregular yet stable transient trajectories dominate the dynamics in all large and nonglobally coupled networks, in stark contrast to the fast convergence to attractor dynamics found recently in globally coupled networks [9] and also opposite to the long chaotic (and thus unstable) transients in randomly diluted networks of excitatory neurons [6]. Interestingly, the above analysis also indicates that the numerically found spike timing sequences [18] are generated by *stable* trajectories; in particular, the systems considered in that work do not exhibit chaotic (unstable) dynamics for any delays and the phase switching observed for long delays is predictable and occurs at well-defined times.

More generally, our results underline that multidimensional deterministic dynamics with statistical properties close to that of a random system need not be generated by deterministic chaos. As we have shown analytically for spiking neural networks with delayed interactions, dynamical irregularity may well be generated by stable trajectories, in particular on the transient. This is in stark contrast to the dynamics on chaotic attractors and chaotic transients [19] that is often irregular as well but induced by unstable periodic orbits. In the systems studied here the irregularity is not induced by unstable periodic orbits but generated due to the coaction of complicated network connectivity, interaction delays, and heterogeneity. Understanding the details of the dynamical origin of long stable transients in such high-dimensional systems constitutes a challenging open problem.

Future work also needs to investigate closer the key consequences for systems in which irregular dynamics is stable. For instance, stable irregular dynamics may lift the important practical constraint of long-term unpredictability that irregular dynamics bears if it is generated by chaos. Stable irregular dynamics, even in multidimensional systems, may well be predictable in practice. For the networks of spiking neurons studied above, this has the astounding consequence that the dynamics in only a small time window, even in the presence of some errors, defines the entire future of the highly irregular spiking dynamics.

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