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Plasticity, Learning, and Complexity in Spiking Networks

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ABSTRACT: Complexity is widespread in neuronal spike trains and propagation of spike activity, in that variations in measurements of neural activity are irregular, heterogeneous, non-stationary, transient, and scale-free. There are numerous possible reasons for this complexity, and numerous possible consequences for neural and behavioral function. The present review is focused on relationships among neural plasticity, learning, and complex spike dynamics in animal nervous systems, including those of humans. The literature on complex spike dynamics and mechanisms of synaptic plasticity are reviewed for the purpose of considering the roles that each might play for the other. That is, the roles of complex spike dynamics in learning and regulatory functions are considered, as well as the roles of learning and regulatory functions in generating complex spike dynamics. Experimental and computational studies from a range of disciplines and perspectives are discussed, and it is concluded that cognitive science and neuroscience have much to gain from investigating the adaptive aspects of complex spike dynamics for neural and cognitive function.

KEY WORDS: Neural dynamics, synaptic mechanisms, scaling laws, critical branching, reinforcement learning, selectionist learning

I. INTRODUCTION

A fundamental fact of nervous systems is that they are perpetually in flux as energy flows through them.[1] Neuron membrane potentials are always fluctuating; action potentials are always being generated; synaptic strengths are constantly being modulated; and network structures never stop changing, albeit these changes may happen on slower or faster time scales.[2] Together, these factors result in complex variations in neural activity.[3] We will define "complex variations" over the course of this review, but to begin, variations range widely in their magnitude and time course, and it has proven challenging to characterize their intricate regularities, irregularities, and dependencies.[4,5]

Whereas complex variations are generally recognized as widespread in nervous systems, their implications for theories of neural function are open to debate. Regularities and dependencies indicate coordination among neural components, in the sense that degrees of freedom in activity are far less than what is possible given the numbers of components.[6,7] But coordination *per se* does not have to be so complex, so why the complexity? One might first reason that complexity could be an inevitable byproduct of *complicated* systems.[8] That is, nervous systems have many different parts made of parts made of parts, and so on. Their aggregated effects may end up appearing as complex variations in measurements. If so, complex variations may be nothing more than noise with respect to functions like perception, attention, memory, and motor control. Complex variations would be a hindrance to neural function in this case, unless noise is helpful for signal encoding and transmission, as in stochastic resonance.[9]

Plasticity is particularly interesting with regard to complex variations in neural activity. Learning theories and algorithms tend to rely on stable, stationary relations between neural activity and the environmental conditions in which learning occurs.[10] Also, plasticity is hypothesized to aid in regulating and

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stabilizing activity.[11] The prevalence of complex variations suggests either that the goals of stabilization and stationarity are not fully achieved or that these are not primary goals of learning and regulatory functions.[12] If stability and stationarity are indeed primary goals, then complex variations would seem to interfere with the shaping of neural activity on the basis of stable relations among sensory inputs, and between sensory inputs and behavioral actions.[13] But what if complex spike dynamics play an important role in learning and regulatory function, and what if plasticity contributes to this complexity? Then it would seem that stabilization and stationarity are not primary goals of neural function.[14]

In the present article, we selectively review the literature on complex variations in neural activity and mechanisms of plasticity in nervous systems, with an emphasis on exploring how findings in these studies might inform one another. We focus on action potentials, often referred to as "spikes," as a level of analysis that bridges neural dynamics, neural mechanisms of plasticity, and behavior. Our overarching goal is to highlight questions, hypotheses, and research directions at the junction of plasticity, complexity, and function in neuroscience and cognitive science.

II. COMPLEX VARIATIONS IN SPIKE DYNAMICS

One of the most basic issues in the physical and life sciences is how the physical becomes informational.[15,16] In neuroscience, this issue goes by the name *neural coding*. Nearly all contemporary theories and approaches identify spikes as central to how neurophysiological processes encode information.[17] Specifically, spikes are treated as discrete events that occur at instantaneous points in time, which means that spikes code information only in terms of when they occur. Temporal coding is the hypothesis that information is coded in the precise timing of individual spikes from individual neurons, whereas rate coding is the hypothesis that information is coded more coarsely in the numbers of spikes occurring within given windows of time. Rate coding is nearly universally accepted as playing some role in neural function. There is less consensus on whether precise spike times carry information,[17] but evidence has been mounting that temporal coding does indeed play a role in neural function (e.g., Van Rulen et al.[18] and Dan and Poo[19]).

Given our focus on spikes, what kinds of variations are found in spike activity, and what is the evidence for complexity in these variations? Recordings of spike trains from individual neurons tend to yield spike times that deviate from any kind of simple, regular pattern. Instead, spike trains generally show highly irregular interspike intervals (ISIs) in raster plots (Figure 1a). One might assume that these irregularities can be explained as random variations in synaptic inputs and other factors that cause membrane potentials to fluctuate. However, Softky and Koch[20] showed that irregularities in single-cell recordings from macaque visual and extrastriate cortices cannot be explained by random inputs to neurons. The coefficients of variation for ISIs are far too high. These authors and others have interpreted such high coefficients as evidence that inputs to neurons are *temporally correlated*,[21] despite the apparent irregularities in spike times. This is our first hint of complexity in spike variations—dependencies among irregular sources of input are indicative of complex coordination among neurons that reduces potential degrees of freedom in spike activities.

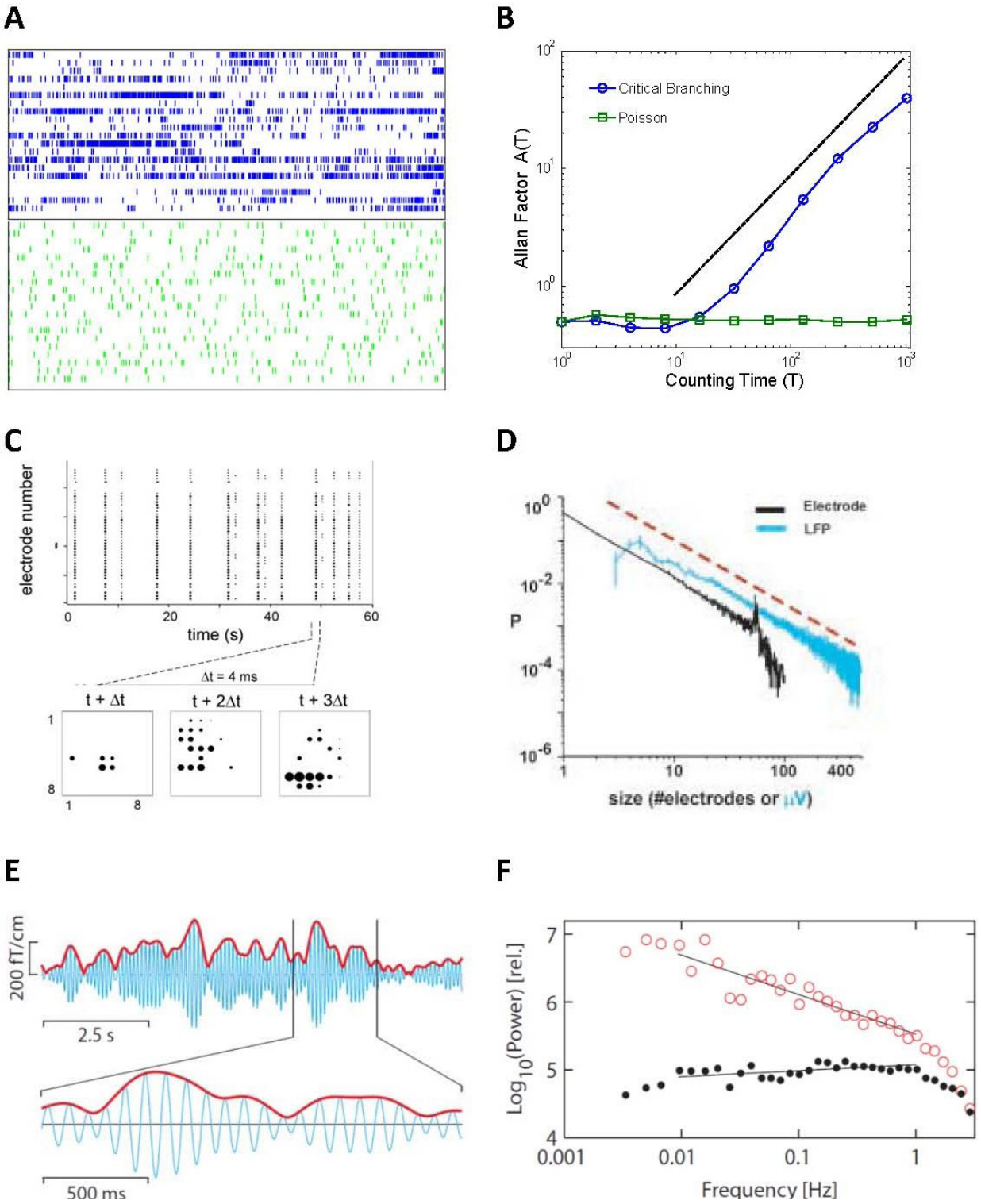
We can also consider whether irregularities in spike trains are themselves complex. Irregular spike times appear as intermittent bursts and clusters of spikes, but such bursts and clustering are not necessarily complex. Even simple, randomly timed spikes exhibit bursts and clustering by chance. Random spike times can be described by Poisson point processes, which have high coefficients of variation near unity and ISI distributions with exponential tails.[22] Some studies, such as that of Softky and Koch,[20] have treated spike trains as Poisson point processes, which is to treat their irregularities as correlated but random nonetheless, and hence non-complex. However, a comprehensive review of the literature yields evidence for many different statistical patterns in spike trains. ISI distributions,

for instance, have been associated with bimodal,[23] bi-exponential,[24] lognormal,[25] inverse Gaussian,[26] gamma,[27] and power-law ISI distributions.[28] This heterogeneity is our second hint at complexity in spike time variations. ISI distributions are useful but limited as expressions of spike time statistics, because ISI distributions discard the temporal distributions of spikes. Thus we can ask further whether spike trains exhibit complex temporal structures. The Poisson process again serves as our baseline of simplicity, because each Poisson spike time provides no information about previous or subsequent spike times—Poisson spike trains exhibit no temporal correlations beyond chance. To the contrary, Teich et al.[29,30] found temporal correlations in spike clustering beyond what is expected from Poisson processes. They used methods like Fano factor and Allan factor analyses to examine clustering as a function of temporal scale in spike trains from cat visual and auditory systems. Results showed that, starting at the time scale of seconds, the smallest spike clusters are nested within larger ones, which are nested within even larger ones, in a lawful scaling relation over more than three orders of magnitude in time (Figure 1b). These temporal correlations add to the complexity of spike trains by showing that irregularities in spike times at least partly reflect fractal clustering of spikes.

Investigations of spike trains emphasize the neuron as a unit of analysis, but spikes are signals that propagate among neurons in networks. Studies of propagating potentials have found that activity spreads in so-called "neuronal avalanches" under a range of preparations and measurement conditions.[31] These avalanches are complex in that their sizes follow a scaling law similar to clustering in spike trains. Spontaneous local field potentials were examined *in vitro* in the original studies of neuronal avalanches in rat somatosensory cortical slice preparations.[32,33] Activity was measured as voltages from electrodes on a microarray, and most activities were short-lived, isolated events over just one or two adjacent electrodes. But sometimes activity spread to a few adjacent electrodes, and sometimes it spread farther and for longer periods of time (Figure 1c). Beggs and Plenz[32] measured these spreading events as avalanches of various sizes, where size was measured as the cumulative voltage over contiguous events on electrodes.[32] Results showed that avalanche sizes followed a scaling law similar to earthquakes: most avalanches were very small, and the probability of observing avalanches decreased with their size according to $P(S) \sim 1/S^{\alpha}$, where $\alpha \sim 3/2$ (Figure 1d). Neuronal avalanches are complex partly because sizes vary over a wide range of scales but also because avalanches are transient, even when exogenous factors are held constant. Avalanches do not comprise a simple, steady "hum" of background noise in neural activity.

Another kind of transience in the propagation of neural activity is found in the synchronization of spikes across neurons. It is well known that large-scale brain activity is characterized by oscillatory waves generated by synchronous neural activity. Synchronization has been hypothesized as a temporal basis for neural information processing,[34] and as such, synchronizations are theorized and observed to be locally transient. For instance, Bressler and Kelso showed that 12-Hz oscillations in local field potentials at striate and inferotemporal cortical sites come into phase with each other transiently, with the onset of visual stimuli.[7] These and other local transients create fluctuations that extend over a wide range of frequencies and amplitudes.[35] Once again, we find these fluctuations to follow a scaling relation, this time between power and frequency: $P(f) \sim 1/f^{\alpha}$, where α is often observed to be near one (Figure 1e and 1f).[36,37]

In summary, neurons continually produce complex variations in spike dynamics that can be characterized as irregular, heterogeneous, non-stationary, transient, and scale-free. The term "complex" can refer to other characteristics of neural networks as well, most notably chaotic dynamics[38] and complex network structures.[39] These characteristics are all interrelated, and together they pose basic questions for neural theories of learning and regulatory function: Do complex variations help or hinder these functions as expressed in spike dynamics, and do these functions themselves contribute to such complex variations? Before we address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t + 3Δt
D
Electrode
LFP
P
size (#electrodes or μV)
E
200 fT/cm
2.5 s
500 ms
F
Log10(Power) [rel.]
Frequency [Hz]

FIGURE 1: A: Example spike train raster plots from model neurons in Kello's (2012)[1] critical branching reservoir (blue), and Poisson spike trains (green) for comparison. **B:** Mean Allan factor analyses of spike trains showing that clustering scales with window size T, $A(T) \sim T^{\alpha}$, with dashed line showing $\alpha = 1$. **C:** Local field potentials recorded from multi-electrode array in Beggs and Plenz (2003),[32] with raster plot showing spontaneous periods of synchronized activity, and bubble plot showing example avalanche "burst" of activity. **D:** Probability density function from Beggs and Plenz showing avalanche probability scales with size, $P(S) \sim 1/S^{3/2}$, with size measured as either summed electrodes or voltages. **E:** Band-pass filtered signal (6.7–13.3 Hz, thin blue lines) from a single channel (0.1–100 Hz) of magnetoencephalography recording from (118), shown at two time scales and filtered through a Morlet wavelet. **F:** Log-log power spectrum of the amplitude envelope of oscillations from data like those shown in **E**. Evidence for 1/f scaling is seen in the negatively sloped line for data (open red circles), and evidence against an artifactual explanation is seen in the flat line for reference channel control data (filled black circles).

these questions directly, we first review neural mechanisms of plasticity as a basis for considering relations with complex spike dynamics.

III. NEURAL PLASTICITY IN SPIKING NETWORKS

Nervous systems are fundamentally adaptive, in that their structures and activities change in response to conditions and stimuli in the environment, and within the organism itself. These adaptations happen across many different time scales, from evolutionary to developmental to experiential. Plasticity refers to all adaptive processes in nervous systems that unfold on time scales no longer than the lifespan, and result in lasting, structural changes. Learning refers to plasticity that improves specific cognitive functions like perception, memory, motor planning, and language. Other mechanisms of plasticity are more regulatory in nature, resulting in changes that establish and maintain the capacity for learning and healthy function.

With respect to spike dynamics, plasticity most often refers to processes that affect the potentiation of synapses as well as the response properties of neurons. Synapses connect presynaptic neurons to postsynaptic neurons. Synaptic potentiation and depression govern the efficacy of presynaptic spikes in triggering or suppressing postsynaptic spikes, as supported by neurotransmitter release and regulation. Spikes on some neurons are caused primarily by spikes on neurons projecting into them, and spikes on other neurons are caused by sensory transduction and other inputs from outside of the nervous systems in question. Thus learning and regulatory mechanisms are theorized to modulate spike dynamics primarily via synaptic efficacies, and in conjunction with extrinsic factors.

Our first hint that plasticity might give rise to complexity is found in the very mechanisms hypothesized to modulate synaptic efficacy. Because spikes are the primary currency of neural information transmission and processing, spike times are hypothesized to play a primary role in mechanisms of synaptic plasticity. Thus, spike dynamics affect synaptic dynamics, and vice versa. Moreover, effects of plasticity on synaptic efficacies are relatively long lasting, whereas spike dynamics unfold on much faster time scales. The result is bi-directional interactions between processes on disparate time scales. These interactions are potential sources of complexity, as explained later, but first we elaborate the mechanisms of plasticity.

The most widely studied and accepted mechanisms of learning are long-term potentiation (LTP) and long-term depression (LTD). LTP generally occurs when two neurons connected by a synapse are depolarized together, resulting in a long-term increase in synaptic efficacy.[40] By contrast, LTD generally occurs when the presynaptic neuron remains hyperpolarized while the postsynaptic neuron becomes depolarized, resulting in a long-term decrease in synaptic efficacy.[41] Depolarization and hyperpolarization correspond with the presence and absence of spike activity, respectively. While

there are numerous exceptions to these rules,[42] synaptic efficacies generally change in relation to coincidences of depolarized and hyperpolarized states.

Spike dynamics come into play more precisely in a phenomenon known as spike timing dependent plasticity (STDP).[43] STDP is characterized by opposite effects on synaptic efficacy depending on whether a spike on the presynaptic neuron is shortly preceded or followed by a spike on the postsynaptic neuron. Canonically, efficacy increases when a postsynaptic spike shortly follows a presynaptic spike, and it decreases for the opposite timing relation. STDP captures properties of both LTP and LTD, but experiments have shown non-additive effects for multiple spikes in close succession, suggesting that the latter cannot be simply subsumed by the former.[44,45] Regardless, all three are theorized as Hebbian learning mechanisms because they can be used to learn associations among stimulus-driven and behavior-driven spike patterns.[46] A fundamental challenge for these learning mechanisms is to explain how changes in efficacy support new learning while also preserving older learning—the stability–plasticity dilemma.[47]

Stability of learning is a regulatory function that must be integrated with mechanisms of synaptic plasticity used for learning. Another regulatory function associated with synaptic plasticity is homeostasis of spike rates.[48] Spikes cannot support neural function if there are too few or too many spikes per unit of time. These extremes would result in insufficient variability in spike times, not to mention insufficient capacity for complex variations. Synaptic scaling is a term used for homeostatic mechanisms hypothesized to potentiate and de-potentiate synapses in non-specific ways to regulate overall spike rates. Another challenge for theories of plasticity, akin to the stability–plasticity dilemma, is to explain how homeostatic and learning mechanisms are integrated.[49] Otherwise, changes in efficacy for purposes of learning could be "undone" by changes for purposes of homeostasis, and vice versa. STDP is promising in this regard because it inherently balances potentiation and de-potentiation.

Finally, we have focused on mechanisms of plasticity driven by spike times and spike rates, but there are also neuromodulatory mechanisms driven by rewards and reward prediction. For instance, concentrations of the neuromodulator dopamine at some synapses serve to gate mechanisms of learning like LTP, LTD, and STDP.[50,51] Gating is hypothesized to increase probability of reward, or increase reward expectation, because major dopamine nuclei are associated with both actual and predicted rewards.[52] These neuromodulatory mechanisms are hypothesized to implement reinforcement learning,[53] but neuromodulators also have been associated with arousal, attention, motivation, and other regulatory functions.[54] Again, it appears that learning and regulatory functions are supported by common mechanisms of plasticity, presumably helping to minimize interference between synaptic changes driven by learning versus regulation.

IV. ROLES OF NEURAL PLASTICITY IN COMPLEX SPIKE DYNAMICS

Our review of plasticity highlights the bi-directional relationship between spike dynamics and synaptic dynamics. This relationship suggests that explanations of complex variations in spike dynamics will include roles for mechanisms of plasticity, but what these roles might be is an open question. Here we review three of possible roles: Mechanisms of plasticity may generate complex variations 1) as a byproduct of regulation, 2) as an adaptive aspect of learning, and 3) as a reflection of complexity in the environment.

Most generally, mechanisms of plasticity may directly generate complex variations in the course of implementing their learning and regulatory functions. As mentioned previously, we have some inkling of this possibility in the fact that spike times unfold on a much faster time scale than the effects of learning and plasticity on synapses. This disparity in time scales means that the bi-directional relationship between spike and synaptic dynamics spans a wide range of time scales. Interactions between processes at disparate

time scales are sometimes associated with *critical point* dynamics,[55] and models of criticality exhibit complex variations in the form of pervasive scaling laws.[56–59] Critical points generally occur when interactions among system components are poised between two or more phases of dynamics.[60] Theorists have argued that nervous systems operate near such critical points,[4] and findings of complex variations are interpreted by some as evidence for criticality.[61]

Mechanisms of plasticity are usually theorized to affect excitation and inhibition in neural networks, so these mechanisms are implicated in phases of spike dynamics defined in terms of excitation and inhibition. If plasticity draws spike dynamics toward a critical point between such phases, then spike dynamics are predicted to exhibit scaling laws. Kello[1] recently investigated these hypotheses and predictions in a spiking neural network model. The model was developed around a mechanism of homeostasis that draws spike dynamics to a critical point by virtue of bi-directional interactions between spiking and synaptic time scales.

The critical point was formulated in terms of *critical branching*,[62] whereby spike propagation is formulated as a *branching process*.[63] Each spike may branch into some number of future spikes, where the number of branches is the number of times a neuron is "blamed" for subsequent spikes. A neuron may be blamed each time one of its spikes is followed by a subsequent spike on one of the neurons toward which it projects. Excitatory neurons can trigger spikes, and inhibitory neurons can fail to prevent spikes, so blame can be assigned to either type of neuron. Critical branching is the state whereby each spike is blamed for one subsequent spike, on average. This state is homeostatic because spikes are conserved, statistically speaking, as they propagate through a network. This conservation requires a balance between excitation and inhibition, which previously has been associated with complex, chaotic variations in neural activity.[64]

The mechanism formulated by Kello[1] targets critical branching by tracking blame locally for individual neurons. If a neuron is blamed more than once during an ISI, one of its axonal synapses is depotentiated with some probability, to reduce the chance of being blamed more than once in the future. If a neuron is not blamed during an ISI, one of its axonal synapses is potentiated with some probability, to increase the chance of being blamed in the future. To create disparity between spike and synaptic time scales, rates of synaptic change were set to be much slower than spike rates by setting a low probability of (de)potentiation (5%). Thus bi-directional interactions between slow synaptic dynamics and relatively fast spike dynamics resulted in continual adjustments in connectivity toward and around critical branching. As predicted, these adjustments caused complex variations in the form of scaling laws in spike clustering, ISI distributions, neuronal avalanches, and $1/f$ scaling.

The critical branching model shows how plasticity for maintaining homeostasis can result in complex variations in spike dynamics. The critical branching mechanism was not designed for learning, but as mentioned earlier, neuroscientific evidence indicates that learning and regulatory functions are supported by common mechanisms. This evidence leads to the consideration of whether a learning mechanism like STDP might be integrated with critical branching. Kello[1] showed that adding an STDP mechanism to the critical branching model does not interfere with homeostasis and complex variations in spike dynamics. It remains to be seen whether STDP or other mechanisms might support learning in conjunction with critical branching, and thereby also play a role in complex variations.

The homeostatic function of critical branching is adaptive in itself, but by virtue of attraction to a critical point, critical branching spike dynamics also are associated with computational benefits. This association raises the possibility of a second role for plasticity in the creation of complex variations in spike dynamics. Learning mechanisms may draw spike dynamics toward critical points and thereby benefit from associated computational capacities. For instance, Kinouchi and Copelli[65] reported a stochastic model of critical branching that was not mechanistic and self-tuning like Kello's,[1] but nonetheless showed that the dynamic range of sensory

systems is maximized at the critical branching point (*see also* Chen et al.[66]). Similarly, Haldeman and Beggs[67] showed that critical branching maximizes information transmission, and Kello[1] showed that critical branching maximizes the memory and encoding capacities of spike dynamics. These modeling results suggest that some learning mechanisms may be designed to target these maxima near critical points, and thereby produce complex variations in spike activity. Thus far, we have considered ways that mechanisms of plasticity *intrinsically* might result in complex spike dynamics. That is, critical point dynamics have been hypothesized to emerge regardless of stimuli or task conditions, or any other factors extrinsic to nervous systems themselves. The third and final role of plasticity that we consider is the shaping of spike dynamics to reflect complex variations that originate *outside* of nervous systems. Natural environments are irregular, heterogeneous, non-stationary, transient, and scale-free, just like neural and behavioral activity. Therefore, it may be adaptive for nervous systems to shape themselves to match the complex variations that constantly impinge upon them.[68] For instance, the long-run statistics of both visual and auditory stimuli are characterized by $1/f$ scaling.[69,70] Recent evidence shows that eye movements and the response properties of auditory nerve cells also exhibit scaling laws.[71–74] Complex spike dynamics underlying these scaling laws may reflect the statistics of visual and auditory inputs.

V. ROLES OF COMPLEX SPIKE DYNAMICS IN NEURAL PLASTICITY

In the previous section, a wealth of studies were reviewed, illustrating different ways that mechanisms of plasticity may be at least partly responsible for complex variations in spike dynamics. In all cases, adaptive qualities of these mechanisms led to complex variations. However, as mentioned at the outset, complex variations may appear to pose a challenge for learning and regulatory functions if neurally mediated relations among sensory inputs and behavioral actions are made less stable or reliable. If this is the case, then complex variations need to be shaped for purposes of learning. In fact, some evidence from brain imaging studies is consistent with this possibility.[75] Complex variations also need to be overcome by learning mechanisms. Consistent with this idea, a spike-based mechanism akin to back-propagation has been shown to support learning in the face of complex variations treated as noise.[76] But rather than treat complexity as something to be overcome, we turn to modeling studies in which complex variations were hypothesized and shown to be adaptive for learning and regulatory functions. The overarching theme of these studies is that the heterogeneity of complex spike dynamics makes available a wide range of useful patterns and nonlinear functions of external inputs.

We begin with two studies that exploited the complexity of critical branching dynamics for purposes of learning nonlinear functions. First, de Arcangelis and Herrmann[77] formulated a simple mechanism of synaptic plasticity for learning nonlinear functions like XOR embedded in critical branching networks. Their method of learning effectively "farmed" the intrinsic variability in spike dynamics to identify local patterns that could be modified slightly to compute particular functions. The heterogeneity of complex variations afforded a variety of spike patterns for the learning mechanism to exploit. Kello similarly exploited variations in critical branching dynamics to compute XOR functions,[1] but unlike de Arcangelis and Herrmann,[77] the functions were not learned by a mechanism of synaptic plasticity. Instead, ordinary least squares regression was used to map unlearned spike dynamics onto XOR functions, a technique known as *reservoir computing*.[78,79] Mappings were computed for inputs at progressively distant points in time to show that fading memory inherent in recurrent network dynamics can be exploited as well, as demonstrated in previous reservoir-computing studies.[80]

XOR functions are useful probes for demonstrating computational properties inherent in complex, recurrent dynamics. However, XOR functions are not easily relatable to functions like perception and memory under more natural con-

ditions. To go beyond XOR, other reservoir computing studies have demonstrated the usefulness of complex dynamics for learning more naturalistic tasks, such as speech recognition,[81] syntactic processing,[82] visual object motion tracking,[83] motor control,[84] and navigation.[85] In fact, a precursor to Kello's critical branching model[1] was applied to visual motion processing.[86]

The reservoir-computing results are useful proofs of concept for our purposes, but learning mechanisms are not part of the theoretical commitments made by these models. One exception to this rule of reservoir computing was reported by Sussillo and Abbott.[87] They developed a learning algorithm specifically designed for *echo state networks*, which represent a particular kind of reservoir computing model with feedback from outputs back to the reservoir. The authors parameterized recurrent connections in the reservoir to generate chaotic reservoir dynamics, and they used feedback connections to adjust synaptic weights on the basis of error between target and output time series. This method of integrating learning with echo state networks was shown to learn complex time series from the Lorenz attractor, and from dynamics of human walking and running. Performance was best when reservoir dynamics were chaotic (i.e., complex).

We have claimed that complex variations in spike activity will result in heterogeneous dynamics useful for nonlinear functions and input-output mappings *in general*. This claim may seem overblown if one assumes that specific functions require specific network architectures and input representations. If this assumption is correct, then complex spike dynamics *per se* would not be sufficient to ensure robust learning. However, Cover's Theorem shows that, just by projecting inputs randomly and nonlinearly into high-dimensional spaces, one can increase the likelihood of learning arbitrary binary classifications using only linear learning mechanisms.[88] The likelihood of learning increases with dimensionality of the space being projected into. This property suggests that robust learning can be ensured, as long as networks can be sufficiently large.

A. Reinforcement Learning and Generalization

Cover's Theorem provides a basis for understanding the effectiveness of reservoir computing models, but it may also serve as a basis for understanding the role of complex spike dynamics in other kinds of learning models. A recent study by Rodny and Noelle[89] provides an excellent case in point. They developed a model of reinforcement learning that took advantage of Cover's Theorem in its use of complex spike dynamics. The goal of reinforcement learning is to maximize actual and expected rewards by increasing the probabilities of behaviors that lead to them.[90] Generally speaking, an agent explores actions in an environment to find and then exploit those that increase rewards. Rodny and Noelle showed that complex variations can help a model find good action policies for obtaining rewards.

The challenge addressed by these authors was to learn action policies online, in environments whose states cannot be exhaustively enumerated and explored. Reinforcement learning algorithms like the adaptive *actor-critic model*[91] use temporal difference errors to learn relationships between states and reward values, both actual and expected. The action policy is based directly on these learned relationships. Sutton proved that, when states can be fully enumerated and explored, the actor-critic algorithm is guaranteed to find an optimal action policy.[92] The algorithm also has support from neuroscience evidence of temporal difference error signals in the basal ganglia, in the form of firing rates of midbrain dopamine neurons.[53,93] Several models have also combined reinforcement learning and spiking networks, with different objectives than the work described above. For instance, Rao and Sejnowski[94] showed that temporal difference learning can be expressed in terms of STDP, and Florian[95] and Izhikevich[96] simulated reinforcement learning by modulating STDP using a global reward signal. More recently, spiking networks have been formulated to simulate the actor-critic framework.[97] These models are limited, however, in that they do not capture the full capacity of ac-

tor-critic models to extrapolate information from experienced to novel states of the environment. The issue of extrapolating learning to novel states has been addressed by introducing *value function approximators* (VFAs) into reinforcement learning models. VFAs serve to generalize learning about expected rewards from experienced states in the environment to novel ones. VFAs have proven useful for environmental states that are continuous with respect to spatial and/or temporal extents, and for speeding up the learning process.[98] However, the actor-critic model is no longer guaranteed to converge with VFAs, and Boyan and Moore[99] provided a demonstration of failure to learn using VFAs. The authors investigated a set of relatively challenging problems, including navigation of a simulated agent in "puddle world"—a two-dimensional grid in which reward was located in the corner of a grid, bordered by two elliptical, perpendicular puddles that represented punishment (negative reward). Finding an optimal action policy was challenging because the state space was large, and the conjunction of horizontal and vertical dimensions was required. The VFA failed to support learning of an optimal action policy.

A number of solutions to this problem with VFAs have been explored.[99,100] While each has strengths and weaknesses, they all sidestep the original problem of online learning using VFAs for environments with very large numbers of states. Rodny and Noelle[89] addressed the problem directly by using a VFA based in complex spike dynamics. The rationale was that traditional VFAs using methods like back-propagation learning are overly restricted to interpolating between known states, rather than extrapolating beyond the space of experienced environmental states.[101,102] The authors reasoned that, if states are represented as spike patterns, then complex variations in spike dynamics might serve to expand the representational state space, as supported by Cover's Theorem. This expansion should facilitate learning and generalization because interpolation in the expanded space results in extrapolation in the environmental space. In fact, previous work using an echo state VFA network established that complex variations could be used to support online reinforcement learning when extrapolating to new environmental states.[103]

The model by Rodny and Noelle[89] used a critical branching reservoir[1] as the VFA for online actor-critic learning in puddle world (Figure 2). Their results showed that the model converges to a near-optimal action policy, similar to the offline model reported by Boyan and Moore.[99] While these results call for further analyses and modeling to understand the specific role of critical branching dynamics, we can tentatively conclude that spike patterns in the VFA reservoir captured the types of conjunctive feature representations needed to support generalization in the service of actor-critic learning.

B. Selectionist Learning via Reinforcement

The heterogeneity of complex spike dynamics is central to their usefulness for learning in reservoir computing models, and also for generalization in reinforcement learning models. In both cases, the associated nonlinearities in complex variations are used to support classifications and input-output mappings. Here we review a different possible role for heterogeneity in complex spike dynamics, one that is based on selectionist learning theories.[104–107]

In evolutionary theory, genotypic variation is created by processes of genetic mutation, recombination, drift, and flow. These processes are not adaptive in and of themselves, because they produce variations that may be more or less fit than their starting points. Instead, these processes serve to instill heterogeneity in the genetic variations found in a given population, and natural selection preserves only the more fit variations as generations pass. Evolutionary adaptation is on a longer time scale than mechanisms of plasticity, but the basic process of selecting from heterogeneous variations may be recapitulated at the level of spike dynamics. Complex variations in spike activity may reflect a diverse repertoire of spatiotemporal patterns, and selectionist learning mechanisms may increase the probability of certain patterns on the basis of fitness, for example, as measured by

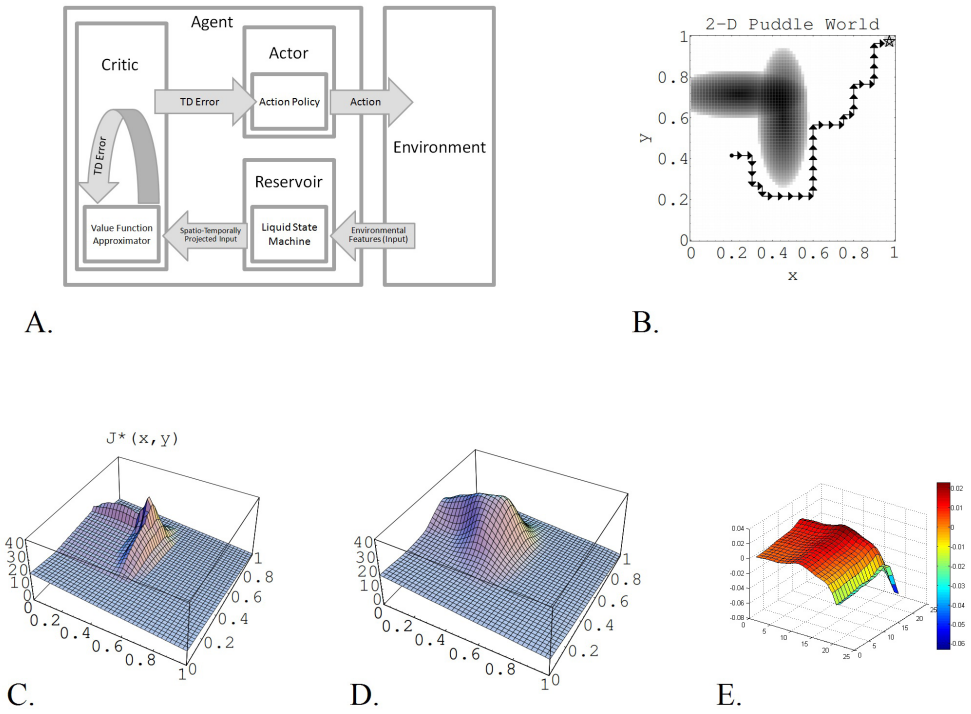

FIGURE 2: A: Schematic of the actor-critic architecture with the addition of a reservoir-supported VFA introduced by Rodny and Noelle (2012).[89] **B**, **C**, and **D** reproduced from Boyan and Moore (1995).[99] **B:** Top-down view of the puddle world grid. Reward is located at top right, and negative reward is within the two elliptical "puddles". **C:** Topology of optimal solution for puddle world value function. Z axis is cost-to-goal (future punishment) starting from each location to reach reward. **D:** Solution to puddle world from Boyan and Moore, using an off-line *grow-support* learning method. **E:** Solution to puddle world from Rodny and Noelle (2012), using the reservoir-supported VFA. For **E**, Z axis is the inverse of mean VFA output, and results are aggregated over 40 simulations.

association with expected or actual rewards.

Heterogeneity in spike activity can arise by virtue of mechanisms of plasticity, as discussed previously, but this is not the only source. Heterogeneity can also arise from complexity in the connectivity patterns of neural networks, and complexity in the structures of nervous systems in general. Complexity from these structural sources may arise early in development through epigenetic processes. By analogy, network structures would be the genotypes and spike dynamics would be the phenotypes. Reward-driven learning would serve to select only those network structures and processes that increase the probability of "fit" spike dynamics. Heterogeneity in spike activity would be critical for maximizing chances that more fit spike dynamics are expressed and available to be selected.

A potential mechanism of selectionist learning was already discussed in the context of reinforcement learning: Reward-modulated STDP[108] can selectively amplify or alter patterns of network con-

nectivity through both LTP and LTD. Izhikevich[109] showed that spiking networks can have intrinsically diverse, *polychronous* patterns of spike activity, and that STDP could be used to amplify specific patterns. A subsequent model[96] demonstrated that dopamine modulation of STDP is a viable mechanism for selectionist learning, while also shedding light on a number of classical and operant conditioning principles. This model demonstrated learning only for selecting relative patterns of spike times between neurons—it is not expected to work for selecting more coarse-grained patterns of firing rates.

An excellent example of selectionist learning via reinforcement can be found in a recent study by Warlaumont,[110] who used the Izhikevich model to simulate selectionist learning of speech-like babbling patterns (Figure 3A). Intrinsic, complex spike dynamics were generated by the spiking neural network, and a subset of model neurons were chosen as controllers of muscle inputs to a simulated vocal tract. Spikes on muscle neurons were summed, smoothed, and then input to the simulated vocal tract as lip and jaw muscle activations. At the start of the simulation, a wide variety of sounds were generated by the vocal tract model, reflecting the diversity of polychronous patterns produced (Figure 3B). Segments of vocal-tract output were judged by human listeners as being more or less speech-like, and judgments were used to modulate the release of dopamine that encoded a reward signal. STDP was increased by dopamine, selecting for those spike dynamics that resulted in more speech-like sounds.

Warlaumont found that, over the course of learning, the model exhibited increasingly mature syllabicity in its vocalizations compared to a yoked control model (Figure 3C).[110] In the control model, dopamine signals were applied at the same rate, but at random with respect to vocalization segments. These results demonstrate how rewards, be they social or intrinsic, can shape behavioral dynamics through selection of spike patterns. In this case, the shaping of behavior resulted in the development of canonical-like babbling, which is marked by speech-like timing, and is considered to be a critical milestone of early speech development.[111] Complex variations in spike dynamics were a part of the selectionist learning process, and related work suggests that heterogeneity in spike activity is useful when networks must learn in the context of background noise.[112] Further simulations are needed to test whether complex variations are necessary or facilitative for selectionist learning as implemented by reward-modulated STDP, and to test whether selectionist learning can promote complex, heterogeneous spike dynamics.

C. Probabilistic Inference

For our last example of how complex spike dynamics may play a role in plasticity, we turn to the general framework of probabilistic inference. Bayes' rule holds that optimal learning and reasoning from uncertainty requires computations over conditional probability distributions. That is, inferences require estimations of probabilities for some variables, given known or hypothesized states of other variables.[113] Exhaustive evaluations of these distributions quickly become intractable in spiking networks as the numbers of variables and states grow (but see Steimer et al.[114]). To address this problem, heuristic methods have been developed based on efficient sampling from conditional probability distributions.[115] Sampling in this case is typically viewed as a stochastic process,[116] but deterministic chaos also may serve as a sampling method.[117] Regardless, nervous systems may have learning mechanisms that implement probabilistic inference by sampling. If so, complex variations may reflect the process of sampling from conditional probability distributions. This is a new, promising direction of research in spiking network models.

VI. CONCLUSION

We began with the question of whether complex spike dynamics might be a hindrance or a benefit to neural function, as supported by neural plasticity. We then reviewed a diverse range of studies,

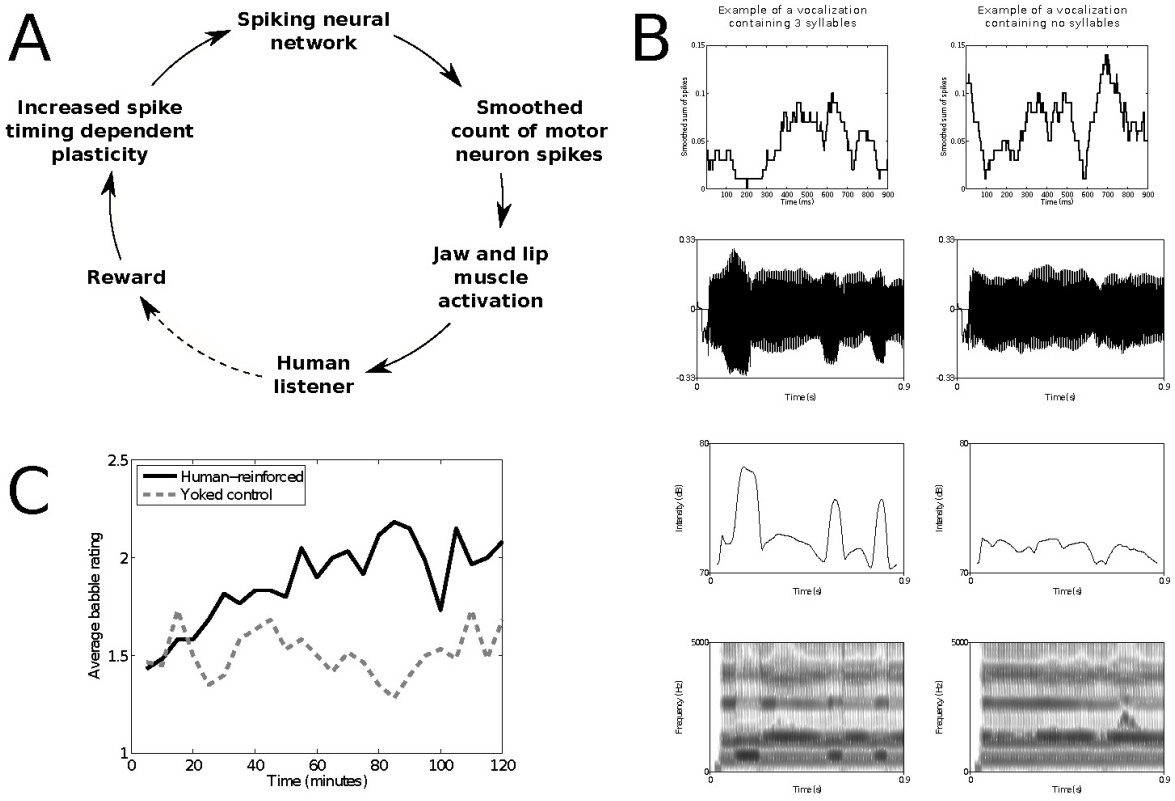

FIGURE 3: A: Schematic depiction of Warlaumont's (2012) model.[110] A neural network with complex spike dynamics was used to control muscle inputs to a vocalization synthesizer simulating the human vocal tract. A human listener selectively rewarded the model's vocalization. Reward generated a surge of dopamine, temporarily increasing STDP. **B:** Examples of two sounds produced by the model. The sound on the left contains three canonical-like syllables while the one on the right contains none. The top row shows the muscle activations as a function of time, the second row shows the sound waveforms, the third row shows the sound intensity envelope, and the fourth row shows spectrograms for each sound. **C:** Across learning, the human-reinforced model exhibited increased production of canonical-like sounds while a yoked control model did not.

from a diverse range of perspectives, to elucidate some possible relationships between neural plasticity and complex variations in spike activity. All of these studies support the idea that complexity is integral to neural plasticity and therefore is not a hindrance for neural function. Instead, complex spike dynamics were theorized to be adaptive in many cases, especially when expressed as scaling laws. In other cases, complex spike dynamics were theorized as byproducts of synaptic plasticity that do not interfere with learning and regulatory functions.

Despite all these studies, it is still possible that complex variations in spike activity sometimes reflect noise in the detrimental sense of the word, at least for certain processes in certain contexts. Our perspective is that researchers must always consider seriously whether a given observation of complexity is just noise. But that said, cognitive science and neuroscience have much more to gain from studying cases in which complex spike dynamics are shown to reflect integral, adaptive aspects of learning and regulatory functions as expressed in neural plasticity.

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