

# **A general, noise driven mechanism for the $1/f$ -like behavior of neural field spectra**

Running title: *A general mechanism for  $1/f$ -like neural field spectra*

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**Consistent observations across recording modalities, experiments, and neural systems find neural field spectra with  $1/f$ -like scaling, eliciting many alternative theories to explain this universal phenomenon. We show that a general dynamical system with stochastic drive and minimal assumptions generates  $1/f$ -like spectra consistent with the range of values observed *in vivo*, without requiring a specific biological mechanism or collective critical behavior.**

## **1 Introduction**

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Transient oscillations are a prominent feature of macroscopic neural field activity (Buzsaki, 2011), linked to brain function (Buzsaki & Draguhn, 2004) and dysfunction (Gibbs & Lennox, 2002; Uhlhaas & Singer, 2012). Oscillations appear as narrowband increases in the spectrum above an aperiodic background in which the power  $P$  decreases proportional to the frequency  $f$  raised to an exponent  $\beta$ :  $P \propto f^\beta$ . Characterizing oscillations while accounting for the aperiodic background is important for understanding neural spectra (Donoghue et al., 2020). Sophisticated methods (Donoghue et al., 2020; Wen & Liu, 2016; L. E. Wilson et al., 2022) support estimation of the 1/f-like, scale-free (He, 2014) or power-law (Newman, 2005) behavior of neural field spectra. Changes in  $\beta$ , the aperiodic exponent, have been investigated in many domains, including sleep (Bódizs et al., 2021; Freeman & Zhai, 2009; G. Horváth et al., 2022; Lendner et al., 2020), aging (Cesnaite et al., 2023; Schaworonkow & Voytek, 2021; Voytek et al., 2015), and disease (Lanzone et al., 2022; Numan et al., 2022; Robertson et al., 2019). While many factors impact estimation of the aperiodic exponent (e.g., the frequency range analyzed (Gerster et al., 2022; Stumpf & Porter, 2012)), values of the exponent reported at higher frequencies (>20 Hz) typically range between  $-4$  and  $-2$  (Table 1).

**Table 1: Example aperiodic exponents for human voltage spectra reported in the literature.** The mean value of the aperiodic exponent ( $\beta$ ) reported in the reference listed in column Reference. Additional details include recording modality, number of subjects  $n$ , frequency range analyzed, and experimental condition. Considering only those studies with minimum frequencies  $\geq 20$  Hz (yellow highlighted rows), the aperiodic exponent has mean -3.1, lower quartile -4, and upper quartile -2.5.

$\beta$	Reference	Recording modality	Frequency	Experimental condition
-0.08	(Colombo et al., 2019)	Scalp EEG (n=5)	20-40 Hz	Anesthesia (Ketamine)
-1.12	(Lanzone et al., 2022)	Scalp EEG (n=16)	1-40 Hz	Eyes closed
-1.3	(Adelhöfer et al., 2021)	Scalp EEG (n=74)	2-40 Hz	Behavioral experiments
-1.44	(Adelhöfer et al., 2021)	Scalp EEG (n=74)	2-40 Hz	Behavioral experiments
-1.48	(Lanzone et al., 2022)	Scalp EEG (n=18)	1-40 Hz	Stroke patients
-1.51	(Robertson et al., 2019)	Scalp EEG (n=78)	4-50 Hz	Resting state
-1.67	(Robertson et al., 2019)	Scalp EEG (n=76)	4-50 Hz	Resting state
-1.84	(Lendner et al., 2020)	Scalp EEG (n=9)	30-45 Hz	Wakefulness
-1.86	(Fransson et al., 2013)	Scalp EEG (n=7)	0.2-30 Hz	Sleep

–1.87	(Lendner et al., 2020)	Scalp EEG (n=14)	30-45 Hz	Resting state
–2.03	(Colombo et al., 2019)	Scalp EEG (n=5)	20-40 Hz	Wakefulness
–2.07	(Fransson et al., 2013)	Scalp EEG (n=15)	0.2-30 Hz	Sleep
–2.32	(Freeman et al., 2000)	Intracranial EEG (n=5)	0.5-150 Hz	Resting state
–2.33	(Bódizs et al., 2021)	Scalp EEG (n=175)	2-48 Hz	NREM sleep
–2.44	(He et al., 2010)	Intracranial EEG (n=5)	1-100 Hz	Wakefulness
–2.48	(Colombo et al., 2019)	Scalp EEG (n=5)	20-40 Hz	Wakefulness
–2.71	(G. Horváth et al., 2022)	Scalp EEG (n=251)	2-48 Hz	NREM sleep
–2.73	(Bódizs et al., 2021)	Scalp EEG (n=175)	2-48 Hz	NREM sleep
–2.75	(Lendner et al., 2020)	Intracranial EEG (n=12)	30-45 Hz	Wakefulness
–2.87	(He et al., 2010)	Intracranial EEG (n=5)	1-100 Hz	Slow wave sleep
–2.99	(Lendner et al., 2020)	Intracranial EEG (n=10)	30-45 Hz	Wakefulness
–3.1	(Lendner et al., 2020)	Scalp EEG (n=9)	30-45 Hz	Anesthesia
–3.13	(Colombo et al., 2019)	Scalp EEG (n=5)	20-40 Hz	Wakefulness
–3.46	(Lendner et al., 2020)	Scalp EEG (n=14)	30-45 Hz	N3 Sleep
–3.59	(Colombo et al., 2019)	Scalp EEG (n=5)	20-40 Hz	Anesthesia (Xenon)

–3.67	(Lendner et al., 2020)	Scalp EEG (n=14)	30-45 Hz	N2 Sleep
–3.69	(Lendner et al., 2020)	Intracranial EEG (n=10)	30-45 Hz	N3 Sleep
–4	(Miller et al., 2009)	Intracranial EEG (n=20)	80-500 Hz	Behavioral experiments
–4.15	(Lendner et al., 2020)	Intracranial EEG (n=10)	30-45 Hz	REM sleep
–4.34	(Lendner et al., 2020)	Intracranial EEG (n=12)	30-45 Hz	Anesthesia
–4.36	(Colombo et al., 2019)	Scalp EEG (n=5)	20-40 Hz	Anesthesia (Propofol)
–4.73	(Lendner et al., 2020)	Scalp EEG (n=14)	30-45 Hz	REM sleep

The universal observation of  $1/f$ -like neural field spectra with a restricted range of aperiodic exponents across different recording modalities, experiments, and neural systems suggests a common generative mechanism. However, many complex, specific mechanisms have been identified to generate this phenomenon. These include excitatory/inhibitory balance (Gao et al., 2017), low-pass frequency filtering by dendrites (Buzsáki et al., 2012) or the extracellular medium (Bédard et al., 2006), nonideal resistive components in the cell membrane (Bédard & Destexhe, 2008), stochastic firing of neurons convolved with an exponential relaxation process (Bédard et al., 2006; Miller et al., 2009; Milstein et al., 2009), stochastic synaptic conductances (Rudolph et al., 2005), stochastically driven damped oscillators with different relaxation rates (Evertz et al., 2022), local homogenous connectivity (Jirsa, 2009), combinations of many transient oscillations at different frequencies and amplitudes (He et al., 2010), or network mechanisms linking slower rhythms to broad neuronal recruitment and therefore larger amplitude field potentials (Buzsáki et al., 2012). Theoretically, scale-free phenomena – with  $1/f$ -like behavior – have been linked to fractal properties (Pritchard, 1992), critical transitions (Newman, 2005; O’Byrne & Jerbi, 2022), and self-organized criticality (Bak et al., 1987; Cocchi et al., 2017). How these proposed biological and mathematical mechanisms contribute – separately or combined – to the range of aperiodic exponents observed across diverse neural field spectra remains unclear.

Here we demonstrate how, in general, a dynamical system with stochastic drive generates  $1/f$ -like behavior at higher frequencies in neural field spectra. We show that two

noise terms – representing correlated and uncorrelated noise inputs – produce the range of aperiodic exponents observed *in vivo*. We illustrate these general results in nonlinear models of neural and non-neural activity to demonstrate the ambiguity in determining the specific mechanisms given only the observed 1/f-like behavior in the spectrum. While more complex underlying mechanisms may exist, we instead illustrate how the range of aperiodic exponents observed *in vivo* occurs in general for dynamical systems with stochastic drive, without requiring a specific biological mechanism or tuning to collective critical behavior.

## 2 Results

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As a general model of neural activity, we consider the n-dimensional dynamical system:

$$\frac{dX_k}{dt} = f_k(X_1, X_2, \dots, X_n) + \sum_{j=1}^m B_{kj} \epsilon_{X,j} \quad (1)$$

where, for each  $k = \{1, 2, \dots, n\}$ ,  $X_k$  is a 1-dimensional variable,  $f_k$  is a nonlinear function,  $B_{kj}$  is a constant, and  $\epsilon_{X,j}$  is an independent Gaussian white noise source with mean 0 and variance  $\sigma_{X,j}^2$ . We assume the variable  $X_1$  (i.e.,  $k = 1$ ) is an observable quantity (e.g., the voltage recorded in the EEG or LFP) and all other variables ( $X_2, X_3, \dots, X_n$ ) represent n-1 unobserved or latent variables impacting the observable dynamics. The unspecified model (1) is general and therefore consistent with diverse models of neural activity. To

derive the main result from this general model requires no specific biophysical mechanism. In what follows, we illustrate these general results by making specific model choices for the variables ( $X_k$ ) and functions ( $f_k$ ).

We assume an equilibrium exists in the noise-free model so that  $\left(\frac{dX_1}{dt}, \frac{dX_2}{dt}, \dots, \frac{dX_n}{dt}\right) = (0, 0, \dots, 0)$  at  $(X_1, X_2, \dots, X_n) \equiv \vec{X}^0$ . Near this equilibrium, the dynamics for one variable ( $X_k$ ) of the nonlinear system (1) can be approximated by the corresponding linear system,

$$\frac{dx_k}{dt} = \left(\frac{\partial f_k}{\partial X_1}\bigg|_{\vec{X}^0}\right)x_1 + \left(\frac{\partial f_k}{\partial X_2}\bigg|_{\vec{X}^0}\right)x_2 + \dots + \left(\frac{\partial f_k}{\partial X_n}\bigg|_{\vec{X}^0}\right)x_n + \sum_{j=1}^m B_{kj} \epsilon_{X,j} \quad (2)$$

where  $x_k$  represent small deviations of the variable  $k$  from the equilibrium  $X_k^0$ , we evaluate the partial derivatives of the nonlinear function  $f_k$  at the equilibrium  $\vec{X}^0$  (Guckenheimer, John & Holmes, Philip, 1983; Izhikevich, E, 2007), and we include the same stochastic perturbations as in the nonlinear system (1). We express the system (2) as

$$\frac{d}{dt}\vec{x} = \mathbf{A}\vec{x} + \mathbf{B}\vec{\epsilon}, \quad (3)$$

where  $\vec{x}$  is the  $n$ -by-1 vector of deviations from the equilibrium,  $\vec{\epsilon}$  is the  $m$ -by-1 vector of independent  $\delta$ -correlated Gaussian white noise sources,  $\mathbf{A}$  is the  $n$ -by- $n$  Jacobian matrix of the nonlinear system (1) evaluated at the equilibrium, and  $\mathbf{B}$  is the  $n$ -by- $m$  noise matrix,



$$\mathbf{B} = \begin{bmatrix} B_{11} & B_{12} & B_{13} & \dots & B_{1m} \\ B_{21} & B_{22} & B_{23} & \dots & B_{2m} \\ B_{31} & B_{32} & B_{33} & \dots & B_{3m} \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ B_{n1} & B_{n2} & B_{n3} & \dots & B_{nm} \end{bmatrix},$$

where the parameter  $B_{ij}$  determines the contribution of noise source  $j$  to variable  $i$ . We assume that  $\mathbf{A}$  satisfies the conditions required for the linear system (3) to accurately approximate the dynamics of the noise-free nonlinear system (1) near the equilibrium (i.e., we assume  $\mathbf{A}$  has no eigenvalues with zero real part and the equilibrium is therefore hyperbolic (Guckenheimer, John & Holmes, Philip, 1983; Izhikevich, E, 2007)).

For the linear system (3), the cross-spectral matrix  $\mathbf{S}[\omega]$  can be obtained from the expression (Gardiner, C, 2004; Kleeman, 2011; Thomas & Lindner, 2019),

$$\mathbf{S}[\omega] = \frac{1}{2\pi} (\mathbf{A} + i\omega\mathbf{I})^{-1} (\mathbf{B} \mathbf{B}^T) (\mathbf{A}^T - i\omega\mathbf{I})^{-1}, \quad (4)$$

where  $\mathbf{A}$  is real,  $\mathbf{I}$  is the identity matrix,  $i = \sqrt{-1}$ , and  $\omega = 2\pi f$  is the frequency. Evaluating the asymptotic behavior of the cross-spectral matrix at high frequencies ( $\omega$  larger than any frequency associated with a natural rhythm of the linear system in (3)), the spectrum of the observable variable  $X_1$  is

$$S_{11}[\omega] = (B_{11}^2 + B_{12}^2 + \dots + B_{1m}^2) \mathcal{O}(\omega^{-2}) + \mathcal{O}(\omega^{-4}), \quad (5)$$

where  $\mathcal{O}(\omega^k)$  indicates the limiting behavior of the spectrum as a function of the  $k^{\text{th}}$  power of  $\omega$  as  $\omega \rightarrow \infty$ ; see Appendix.

The result in (5) shows that, at high frequencies, the aperiodic exponent  $\beta$  depends on the relative noise to the observable variable. Without stochastic drive to the observable variable  $X_1$  (i.e., with  $B_{1k} = 0$  for all  $k$  in  $\{1, 2, \dots, m\}$ ),

$$S_{11}[\omega] = \mathcal{O}(\omega^{-4}),$$

so that the aperiodic exponent  $\beta = -4$  at high frequencies (i.e., as  $\omega \rightarrow \infty$ ). Alternatively, with stochastic drive to the observable variable  $X_1$  (i.e., with  $B_{1k} \neq 0$  for any  $k$  in  $\{1, 2, \dots, m\}$ ),

$$S_{11}[\omega] = \mathcal{O}(\omega^{-2}),$$

so that the aperiodic exponent  $\beta = -2$  at high frequencies (i.e., as  $\omega \rightarrow \infty$ ). We note that, in this case, random walk dynamics dominates the spectrum at high frequencies, with well-known power-law behavior (e.g., see (Milotti, 2002)).

To summarize, we consider a general,  $n$ -dimensional nonlinear dynamical system with stochastic drive (1). We assume an equilibrium exists in this system with dynamics well-approximated by the linearized system (2). Near this equilibrium, the dynamics produce aperiodic exponents between  $-4$  and  $-2$  at high frequencies (i.e., frequencies beyond

the natural frequencies or spectral peaks of the system) consistent with the range of values observed *in vivo*. The value of the aperiodic exponent depends on the noise in the observed and latent variables; when noise in the observable variable  $X_1$  dominates, the aperiodic exponent  $\approx -2$ , while when the noise in the latent variables dominates, the aperiodic exponent  $\approx -4$ .

The main result (5) and implications for the aperiodic exponent ( $-4 \leq \beta \leq -2$ ) are for the general model (1). These general results do not require a specific biophysical model of neural activity. In what follows, we illustrate the generality of these results in four example models, in which we choose the nonlinear functions  $f_k$  in model (1) and assume a square diagonal noise matrix  $\mathbf{B}$  to simplify the presentation of numerical simulation results. In doing so, we show that each model produces aperiodic exponents consistent with *in vivo* data ( $-4 \leq \beta \leq -2$ ) but with different physical interpretations.

## **2.1 A reduced model of single neuron activity**

We first consider a reduced Hodgkin-Huxley type model (Hodgkin & Huxley, 1952; Izhikevich, E, 2007) to simulate the subthreshold dynamics of a single neuron. The model equations describe the dynamics of an observable voltage ( $V$ ) and a latent membrane current ( $M$ ),

$$\frac{dV}{dt} = I_0 + g_M M(E_M - V) + \epsilon_V \quad (6)$$

$$\frac{dM}{dt} = \alpha_M[V](1 - M) - \beta_M[V]M + \epsilon_M .$$

In the first equation, three terms drive the voltage dynamics: a constant input current ( $I_0$ ), a dynamic membrane current ( $g_M M(E_M - V)$ ), and voltage noise ( $\epsilon_V$ , Gaussian distributed with mean 0 and variance  $\sigma_V^2$ ). In the second equation, the dynamics of the latent membrane current depend on forward ( $\alpha_M[V]$ ) and backward ( $\beta_M[V]$ ) rate functions,

$$\alpha_M[V] = \frac{0.02}{1 + \exp(\frac{-V - 20}{5})}$$

$$\beta_M[V] = 0.01 \exp(\frac{-V - 43}{18}) .$$

We choose these functions to simulate a muscarinic receptor suppressed potassium current (M-current, see Table A2 of (Traub et al., 2003)) (Kopell et al., 2011; Kramer, Roopun, et al., 2008). We omit other membrane currents (e.g., fast sodium and potassium currents) to focus on the subthreshold membrane dynamics without action potential generation. A stochastic drive also impacts the membrane current dynamics ( $\epsilon_M$ , Gaussian distributed with mean 0 and variance  $\sigma_M^2$ ).

Choosing the model parameters  $I_0 = 1$ ,  $g_M = 4$ , and  $E_M = -95$ , we find an equilibrium of the noise-free model (6) at

$$(V_0, M_0) \approx (-48.15, 0.00534) .$$

The noise-driven linearized system near this equilibrium is approximately,

$$\frac{d\bar{V}}{dt} = -0.0213 \bar{V} - 187.38 \bar{M} + \epsilon_V$$

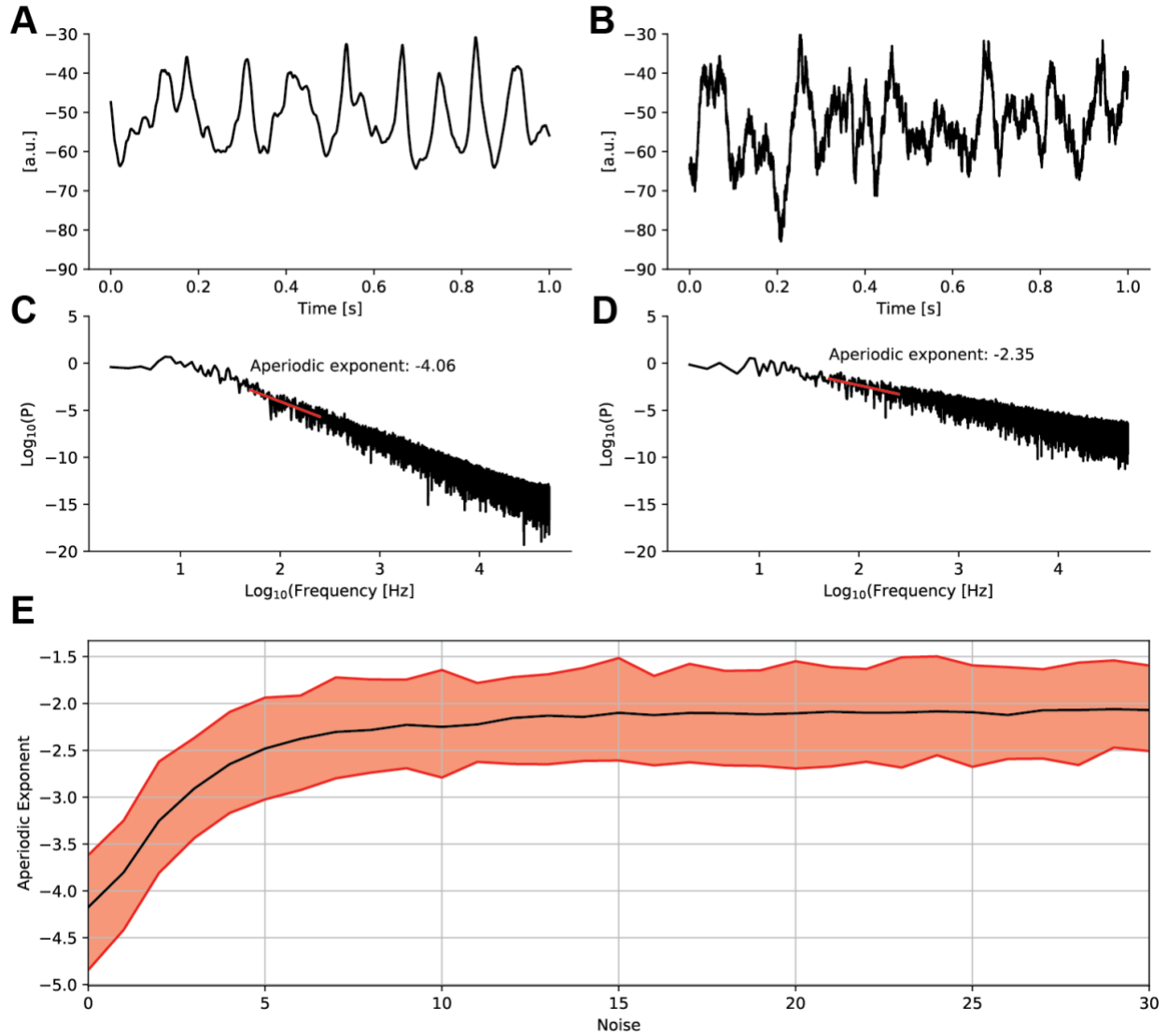
$$\frac{d\bar{M}}{dt} = 0.0000181 \bar{V} - 0.0134 \bar{M} + \epsilon_M ,$$

and the equilibrium is hyperbolic with eigenvalues  $-0.0174 \pm 0.0581 i$ .

Consistent with the general theory, we expect this nonlinear Hodgkin-Huxley type model (6) to produce spectra with aperiodic exponents  $-4 \leq \beta \leq -2$ , depending on the values of the stochastic drives  $(\epsilon_V, \epsilon_M)$ . To show this, we simulate the Hodgkin-Huxley type model (6) and estimate the spectrum of the voltage variable  $V$  with fixed current noise ( $\sigma_M = 0.01$ ) and variable voltage noise ( $0 \leq \sigma_V \leq 30$ ). In agreement with the general theory (Figure 1), as the voltage noise increases, the aperiodic exponent increases from near  $\beta \approx -4$  when  $\sigma_V = 0$  to  $\beta \approx -2$  when  $\sigma_V = 30$ .

We conclude that this nonlinear model of single neuron subthreshold dynamics (6) produces aperiodic exponents consistent with the range of values observed *in vivo*. In agreement with the general theory, the value of the aperiodic exponent depends on the

relative noise in the observable voltage variable and latent current variable. In this case, when noise in the membrane current dominates, the aperiodic exponent approaches  $-4$ ; when noise in the voltage dominates, the aperiodic exponent approaches  $-2$ .



**Figure 1:** In a reduced model of single neuron activity, the aperiodic exponent increases from approximately  $-4$  to  $-2$  with the voltage noise. (A,B) Example voltage time series when (A) the voltage noise is 0 (i.e.,  $\epsilon_V = 0$ ), or (B) the voltage noise is non-zero (i.e.,  $\sigma_V = 20$ ). (C,D) The corresponding spectra (black) and linear fits (red,

50 Hz to 250 Hz) for the time series in (A,B). **(E)** Estimates of the aperiodic exponent for increasing values of voltage noise. Black (red) indicates mean (standard deviation) of estimates across 100 simulations. In all simulations, the current noise ( $\sigma_M = 0.01$ ) is fixed and numerical integration is performed using the Euler–Maruyama method with timestep 0.01 ms. Code to simulate the model and create this figure is available at <https://github.com/Mark-Kramer/Aperiodic-Exponent-Model>

## 2.2 A 2-dimensional model of neural population activity

We now consider the Wilson-Cowan equations as a model of neural population activity (H. R. Wilson & Cowan, 1972). The equations describe the interacting dynamics of an excitatory ( $E$ ) and inhibitory ( $I$ ) neural population,

$$\begin{aligned}\tau_e \frac{dE}{dt} &= -E + (k_e - r_e E) \mathcal{S}_e[c_1 E - c_2 I + P] + \epsilon_E \\ \tau_i \frac{dI}{dt} &= -I + (k_i - r_i I) \mathcal{S}_i[c_3 E - c_4 I + Q] + \epsilon_I,\end{aligned}\tag{7}$$

where  $\mathcal{S}$  is the sigmoid function,

$$\mathcal{S}_k[x] = \frac{1}{1 + \exp(-a_k (x - \theta_k))} - \frac{1}{1 + \exp(a_k \theta_k)},$$

and  $\epsilon_E$  and  $\epsilon_I$  are noise terms (Gaussian distributed with mean 0 and variances  $\sigma_E^2$  and  $\sigma_I^2$ , respectively). We choose the parameters to produce damped oscillatory behavior (see Figure 10 of (H. R. Wilson & Cowan, 1972) and the caption of Figure 2 for the parameter values used here). With these parameters, we find an equilibrium of the noise-free model (7),

$$(E_0, I_0) \approx (0.1511, 0.1585) .$$

The noise-driven linearized system near this equilibrium is approximately,

$$\frac{d\bar{E}}{dt} = 1.482 \bar{V} - 2.660 \bar{I} + \epsilon_E$$

$$\frac{d\bar{I}}{dt} = 3.963 \bar{V} - 1.981 \bar{I} + \epsilon_I ,$$

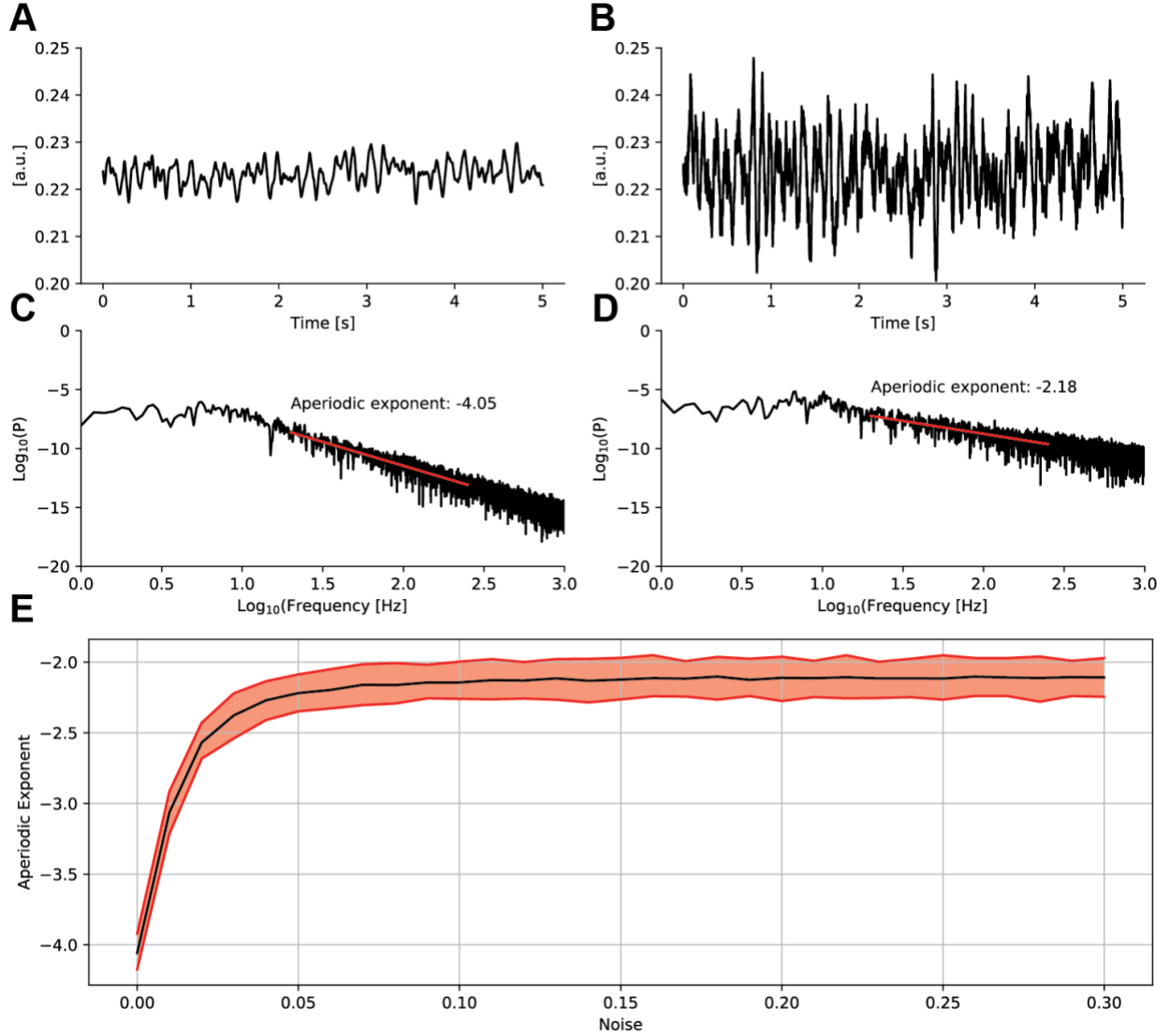
and the equilibrium is hyperbolic, with eigenvalues  $-0.250 \pm 2.75 i$ .

According to the general theory, we expect the nonlinear system (7) near the equilibrium  $(E_0, I_0)$  to produce spectra with aperiodic exponents  $-4 \leq \beta \leq -2$ , depending on the values of the stochastic drives  $(\epsilon_E, \epsilon_I)$ . To show this, we simulate the nonlinear system (7) and estimate the spectrum of the excitatory variable  $E$  with fixed inhibitory noise ( $\sigma_I = 0.1$ ) and variable excitatory noise ( $0 \leq \sigma_E \leq 0.3$ ). In agreement with the general theory (Figure



2), as the excitatory noise increases, the aperiodic exponent increases from near  $\beta \approx -4$  when  $\sigma_E = 0$  to  $\beta \approx -2$  when  $\sigma_E = 0.3$ .

We conclude that this nonlinear model of neural population activity (7) produces aperiodic exponents consistent with the range of values observed *in vivo*. In this model, the value of the aperiodic exponent depends on the relative noise in the observable variable  $E$  and latent variable  $I$ . When noise in the inhibitory population dominates, the aperiodic exponent approaches  $-4$ ; when noise in the excitatory population dominates, the aperiodic exponent approaches  $-2$ .



**Figure 2: In a 2-dimensional model of neural population activity, the aperiodic exponent increases from approximately  $-4$  to  $-2$  with the excitatory noise. (A,B)** Example excitatory population time series when (A) the excitatory noise is 0 (i.e.,  $\epsilon_E = 0$ ), or (B) the excitatory noise is non-zero (i.e.,  $\sigma_E = 0.2$ ). **(C,D)** The corresponding spectra (black) and linear fits (red, 20 Hz to 250 Hz) for the time series in (A,B). **(E)** Estimates of the aperiodic exponent for increasing values of excitatory noise. Black (red) indicates mean (standard deviation) of estimates across 100 simulations. In all simulations, the inhibitory noise ( $\sigma_I = 0.1$ ) is fixed. We use the model parameters:  $c_1 = 15, c_2 = 15, c_3 =$

15,  $c_4 = 7$ ,  $a_e = 1$ ,  $\theta_e = 2$ ,  $a_i = 2$ ,  $\theta_i = 2.5$ ,  $\tau_e = 50$ ,  $\tau_i = 50$ ,  $r_e = 1$ ,  $r_i = 1$ ,  $k_e = 1$ ,  $k_i = 1$ ,  $P = 1.25$ ,  $Q = 0$ , and perform numerical integration using the Euler–Maruyama method with timestep 0.1 ms. Code to simulate the model and create this figure is available at <https://github.com/Mark-Kramer/Aperiodic-Exponent-Model>.

### 2.3 A 10-dimensional model of neural population activity

To illustrate an application of the main result (5) to a higher-dimensional neural model, we consider a mean-field model of neural population activity consisting of the coupled differential equations:

$$\tau_k \frac{dh_k}{dt} = (h_k^{rest} - h_k) + \psi_{ek}[h_e] I_{ek} + \psi_{ik}[h_i] I_{ik} \quad (8)$$

$$\left( \frac{d}{dt} + \gamma_k \right)^2 I_{kl} = \left( N_{kl}^\beta S_k[h_k] + P_{kl} \right) G_k \gamma_k e$$

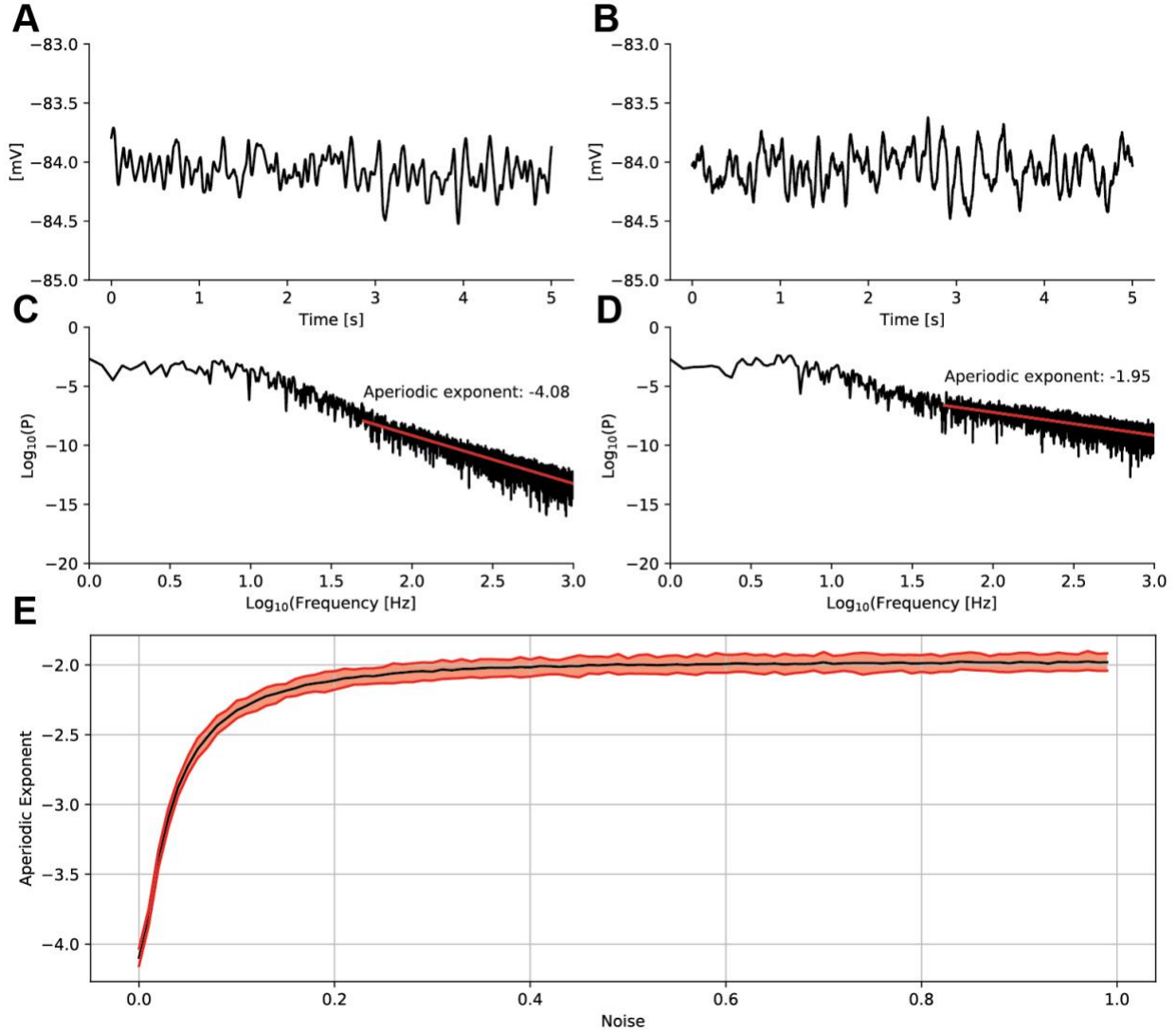
where  $k = \{e, i\}$  and  $l = \{e, i\}$  denote excitatory ( $e$ ) and inhibitory ( $i$ ) neural populations,  $\psi_{kl}$  are normalized weighting functions, and  $S_k[h_k]$  are sigmoidal transfer functions (M. L. Steyn-Ross et al., 2003). The model variables simulate the macrocolumn-averaged transmembrane soma voltage of an excitatory ( $h_e$ ) and inhibitory ( $h_i$ ) neural population, and synaptic input ( $I_{kl}$ ) from population  $k$  to population  $l$ . Expressing the second-order differential equations for the synaptic inputs  $I_{kl}$  as first-order differential equations results in a system of 10 coupled first-order differential equations. The variable  $h_e$  is observable

(M. L. Steyn-Ross et al., 1999) and the other variables are latent. We include independent stochastic drive to the dynamics of the observable variable (mean 0 and variance  $\sigma_{h_e}^2$ ) and each latent variable (mean 0 and variance  $\sigma_L^2$  to all latent variables). Applications of the model include simulating electroencephalogram (EEG) dynamics during sleep (D. Steyn-Ross et al., 2005; M. L. Steyn-Ross et al., 2005; M. T. Wilson, Steyn-Ross, et al., 2006), seizures (Kramer et al., 2005, 2007; M. L. Steyn-Ross et al., 2012; M. T. Wilson, Sleight, et al., 2006), and anesthesia (M. L. Steyn-Ross et al., 1999, 2003).

Fixing all model parameters to the default values in (M. L. Steyn-Ross et al., 2003), a stable equilibrium exists (M. L. Steyn-Ross et al., 2003). We therefore expect, consistent with the general theory, the model (8) to produce spectra with aperiodic exponents  $-4 \leq \beta \leq -2$ , depending on the values of the stochastic drives. To show this, we simulate the model (8) and estimate the spectrum of the voltage variable  $h_e$  with fixed noise ( $\sigma_L = 50$ ) to all latent variables and variable noise to the observable variable ( $h_e, 0 \leq \sigma_{h_e} \leq 1$ ). In agreement with the general theory (Figure 3), as the noise to the excitatory neural population increases, the aperiodic exponent increases from near  $\beta \approx -4$  when  $\sigma_{h_e} = 0$  to  $\beta \approx -2$  when  $\sigma_{h_e} = 1$ .

We conclude that this high-dimensional (10<sup>th</sup> order) nonlinear model of macroscopic neural population activity (8) produces aperiodic exponents consistent with the range of values observed *in vivo*. In agreement with the general theory, the value of the aperiodic exponent depends on the relative noise in the observable variable ( $h_e$ ) and latent variables. In this case, when noise outside of the excitatory population dominates, the

aperiodic exponent approaches  $-4$ ; when noise in the excitatory population ( $h_e$ ) dominates, the aperiodic exponent approaches  $-2$ .



**Figure 3: In a 10-dimensional model of neural population activity, the aperiodic exponent increases from approximately  $-4$  to  $-2$  with increasing noise to the excitatory neural population. (A,B) Example excitatory neural population activity when (A) noise to the excitatory neural population is 0 (i.e.,  $\epsilon_{h_e} = 0$ ), or (B) non-zero (i.e.,  $\sigma_{h_e} = 1$ ). (C,D) The corresponding spectra (black) and linear fits (red, 50 Hz to 1000 Hz) for the**

time series in (A,B). **(E)** Estimates of the aperiodic exponent for increasing values of excitatory neural population noise. Black (red) indicates mean (standard deviation) of estimates across 100 simulations. In all simulations, the latent noise terms ( $\sigma_L = 50$ ) are fixed and numerical integration performed using the Euler–Maruyama method with timestep 0.1 ms. Code to simulate the model and create this figure is available at <https://github.com/Mark-Kramer/Aperiodic-Exponent-Model>

## 2.4 A model of predator-prey interactions

To illustrate the generality of the main result (5) beyond models of neural activity we consider a nonlinear model of predator-prey interactions,

$$\frac{dx}{dt} = \frac{x(\gamma - x)}{\gamma} - x y + \epsilon_x \quad (9)$$

$$\frac{dy}{dt} = -\alpha y + x y + \epsilon_y ,$$

where  $x$  and  $y$  represent prey and predator populations, respectively, and the prey population includes self-regulation;  $(\alpha, \gamma)$  are positive constants; and  $\epsilon_x$  and  $\epsilon_y$  are noise terms (Gaussian distributed with mean 0 and variances  $\sigma_x^2$  and  $\sigma_y^2$ , respectively) (Edelstein-Keshet, 2005). The non-trivial equilibrium of the deterministic system is,

$$(x_0, y_0) = \left( \alpha, 1 - \frac{\alpha}{\gamma} \right) ,$$

and we require  $\gamma > \alpha$  so that the equilibrium predator population is positive. The linearized system near this equilibrium is,

$$\frac{d}{dt} \begin{pmatrix} \bar{x} \\ \bar{y} \end{pmatrix} = A \begin{pmatrix} \bar{x} \\ \bar{y} \end{pmatrix} + \begin{pmatrix} \epsilon_x \\ \epsilon_y \end{pmatrix},$$

where  $A = \begin{pmatrix} -\frac{\alpha}{\gamma} & -\alpha \\ 1 - \frac{\alpha}{\gamma} & 0 \end{pmatrix}$ . Because the trace of  $A = -\frac{\alpha}{\gamma}$  is negative, and the determinant

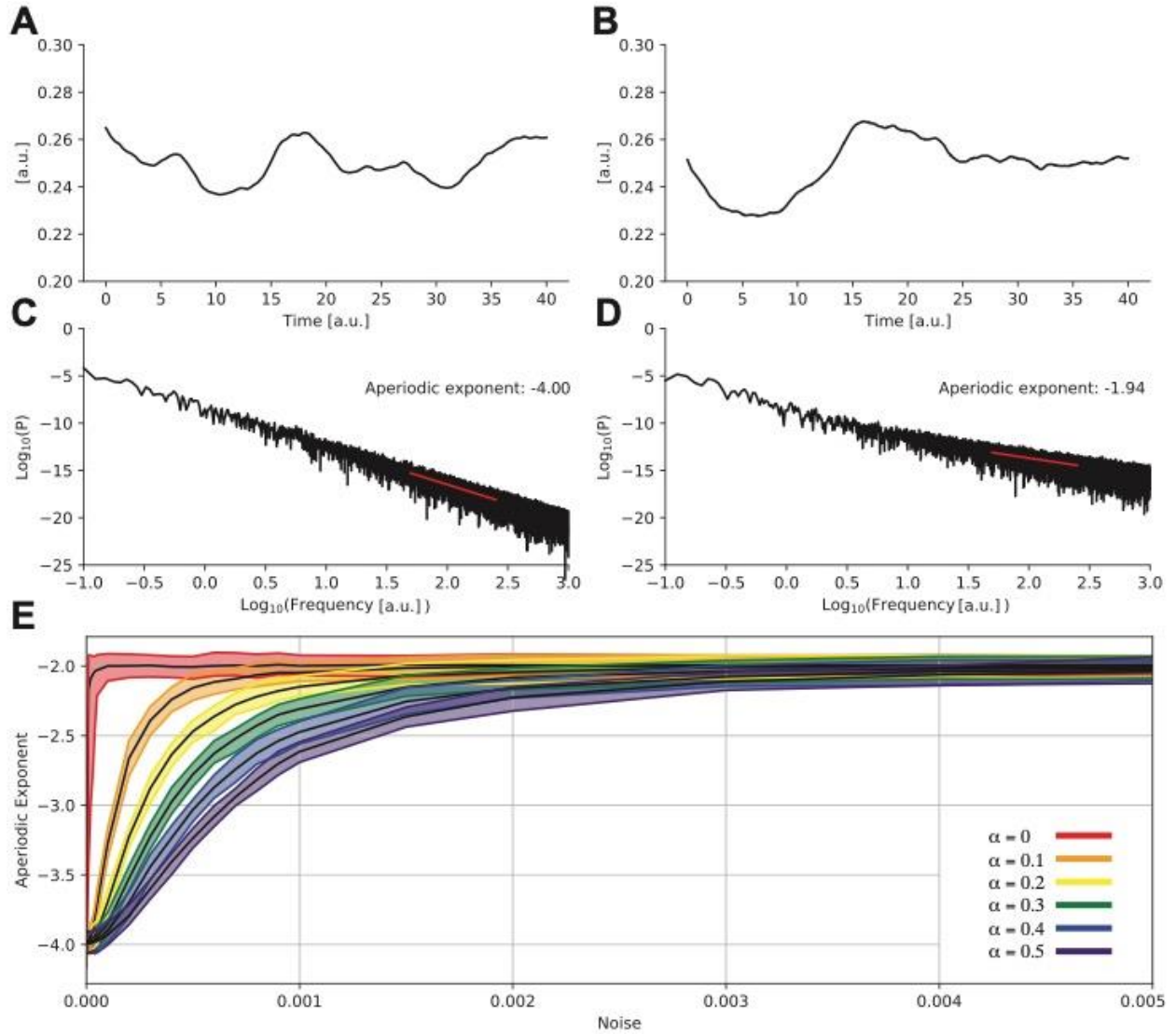
of  $A = \alpha y_0$  is positive, the equilibrium is stable and hyperbolic (Edelstein-Keshet, 2005).

Consistent with the general theory, we expect this predator-prey model (9) to produce spectra with aperiodic exponents  $-4 \leq \beta \leq -2$ , depending on the values of the stochastic drives  $(\epsilon_x, \epsilon_y)$ . To show this, we simulate the predator-prey model (9) over a range of parameters  $(\alpha, \gamma)$  and estimate the spectrum of the prey variable  $x$  with fixed predator noise ( $\sigma_y = 1$ ) and variable prey noise ( $0 \leq \sigma_x \leq 0.005$ ). In agreement with the general theory, the values of the aperiodic exponent in this non-neural model lie within the range  $-4 \leq \beta \leq -2$ , depending on the relative noise in the prey variable (Figure 4). As the prey noise increases, the aperiodic exponent increases from near  $\beta \approx -4$  when  $\sigma_x = 0$  to  $\beta \approx -2$  when  $\sigma_x = 0.005$  across a range of model parameters  $(\alpha, \gamma)$ .

We note that, mathematically, three of the four models are essentially the same; the Hodgkin-Huxley type model (6), the 2-dimensional neural population model (7), and the

predator-prey model (9) are all planar models with independent additive noise in the two variables. We choose to illustrate the theory with these example simulations to demonstrate the difficulty of interpreting the biological mechanism producing the aperiodic exponent. While the four models (6-9) simulate different neural and non-neural dynamics, each model produces 1/f-like scaling with aperiodic exponents consistent with *in vivo* observations of neural activity. The interpretation of the mechanism affecting the aperiodic exponent depends on the model choice; the aperiodic exponent  $\beta$  approaches  $-2$  with increasing voltage noise in (6), noise to an excitatory neural population in (7) and (8), or prey noise in (9). Because each model satisfies the general conditions (i.e., a hyperbolic equilibrium exists), the spectral results derived for the general model in (5) capture the 1/f-like spectrum for each specific, biophysical model considered here.





**Figure 4:** In a predator-prey population model, the aperiodic exponent increases from approximately  $-4$  to  $-2$  with the prey noise. (A,B) Example prey population dynamics when (A) the prey noise is 0 (i.e.,  $\epsilon_x = 0$ ), or (B) the prey noise is non-zero (i.e.,  $\sigma_x = 0.01$ ). (C,D) The corresponding spectra (black) and linear fits (red, 50 Hz to 250 Hz) for the time series in (A,B). (E) Estimates of the aperiodic exponent for increasing values of prey noise. Black (color) indicates mean (standard deviation) of estimates across 100 simulations. In all simulations, the predator noise ( $\sigma_y = 1$ ) is fixed and the model

*simulated for 250,000 steps with numerical integration performed using the Euler–Maruyama method with timestep 0.0002 a.u.; to avoid an initial large amplitude transient, we omit the first 50,000 steps of simulated data from analysis. In (A–D), the parameters are  $\alpha = 0.25$ ,  $\gamma = 0.6$ . In (E), colors indicate simulations at  $\alpha = \{0.0, 0.1, \dots, 0.5\}$ . For each fixed  $\alpha$ , we simulate 10 instances of the model at 10 different values of  $\gamma = \{0.1, 0.2, 0.3, \dots, 1.0\}$  for a total of 100 simulations. Code to simulate the model and create this figure is available at <https://github.com/Mark-Kramer/Aperiodic-Exponent-Model>*

### 3 Discussion

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A universal feature of neural field potential spectra is  $1/f$ -like scaling at high frequencies. To produce this power-law, many generative mechanisms have been proposed with diverse biological implementations and interpretations. Here, we do not propose a specific biological mechanism produces  $1/f$ -like scaling. Instead, to understand how different neural mechanisms can produce similar  $1/f$ -like scaling, we consider a general nonlinear dynamical system with stochastic drive. We show that dynamics near a (hyperbolic) equilibrium in this general model of neural activity produce aperiodic exponents between  $-4$  and  $-2$ , consistent with the range of values reported *in vivo* for higher frequencies (e.g.,  $>20$  Hz). We illustrate these results in neural and non-neural models. We propose that the range of aperiodic exponents observed across recording modalities, experiments, and neural systems is a natural consequence of a noise driven dynamical system.

We considered here a single statistic – the aperiodic exponent – reflecting the  $1/f$ -like feature of the neural field spectrum. We note that many different models can explain the same observed statistic. For example, observations from neural systems can produce spectra with broadband peaks in the gamma band (approximately 30-80 Hz) (Fries et al., 2007). Many approaches exist to explain these observed spectral peaks, including statistical approaches (e.g., an autoregressive model of order two (Spyropoulos et al., 2020)), mechanical approaches (e.g., a damped driven oscillator (Spyropoulos et al., 2020)), or biophysical approaches (e.g., the interneuron network model, or the pyramidal-interneuron network model (Whittington et al., 2000)). Our understanding of the gamma rhythm in a particular experiment depends on the model choice. In the same way, observations from neural systems produce spectra with  $1/f$ -like scaling at high frequencies, and many models exist to explain this scaling. Our results show mathematically why many models can produce the range of aperiodic exponents observed *in vivo* ( $-4 \leq \beta \leq -2$ ). Due to the general nature of this mathematical result, it is not surprising that many different proposals exist to explain the  $1/f$ -like spectrum. In practice, we expect biological mechanisms must exist to create the scaling observed *in vivo*. Identifying these biological mechanisms, and their expression in the diverse observations of  $1/f$ -like scaling reported, remains an important challenge.

Our results are consistent with previous work showing that power-law scaling occurs in simple stochastic or physical systems. For example, in (Gao et al., 2017) the authors relate estimates of the aperiodic exponent to changes in the balance between excitation

and inhibition. To do so, the authors simulate the local field potential as the summed synaptic current generated by independent stochastic spiking excitatory and inhibitory cells. In (Bédard et al., 2006) the authors propose that  $1/f$ -like scaling in the spectrum does not rely on critical states, but instead depends on the filtering properties of the extracellular medium (although this mechanism remains debated (Logothetis et al., 2007)). In (Touboul & Destexhe, 2010) the authors show that simple models of stochastic processes (high-frequency shot-noise processes or Ornstein-Uhlenbeck processes) produce peak-amplitude distributions consistent with power-law distributions. In (Priesemann & Shriki, 2018) the authors show that inhomogeneous Poisson processes can produce approximate power law distributions in the size and duration distributions of avalanches (i.e., activity cascades). Consistent with our results, these examples generate power-law scaling without requiring a sophisticated biological or mathematical mechanism. Distinct from these previous works, we consider an (unspecified)  $n$ -dimensional dynamical system and show that, near a (hyperbolic) equilibrium, stochastic drive produces aperiodic exponents consistent with values observed *in vivo*.

Under the general framework considered here, the range of aperiodic exponents reflects different types of noise. The observable dynamics (e.g., variable  $X_1$  in (1)) depend directly on the stochastic drives to the observable variable, and indirectly on the stochastic drives to the latent variable(s). The latent dynamics introduce correlations in the uncorrelated latent noise process before this noise reaches the observable dynamics. Therefore, the observable dynamics depend on both uncorrelated and correlated noise inputs, and we may interpret the aperiodic exponent in terms of these different types of noise; an

aperiodic exponent near  $\beta = -4$  indicates correlated noise inputs dominate the high frequency observable dynamics, while  $\beta = -2$  indicates uncorrelated noise dominates the high frequency observable dynamics. These general results are independent of a specific biophysical mechanism, and a biophysical interpretation of the relationship between different types of noise and the aperiodic exponent depends on the specific model choice.

Some observations report aperiodic exponents greater than  $-2$  (i.e.,  $\beta > -2$ ), beyond the range of aperiodic exponents derived here. Experimental factors, such as measurement noise, which flattens the spectrum and shifts  $\beta$  towards 0, might contribute to these observations. In addition, analysis factors may impact reported results. For example, the frequency range considered varies widely across studies (see discussion of fitting ranges in (Gerster et al., 2022)). In general, larger aperiodic exponents (more negative  $\beta$ ) occur at higher frequencies (Bédard et al., 2006; Colombo et al., 2019; Ibarra Chaoul & Siegel, 2021; Racz et al., 2021), although not always (Chaudhuri et al., 2018). In the framework considered here, the result  $-4 \leq \beta \leq -2$  holds in the high frequency limit (when the measured frequency exceeds any natural frequency of the neural population) and near an equilibrium of the dynamical system. In lower frequency bands (e.g., below the natural frequency), our simplifying asymptotic arguments no longer apply, and the relationship between power and frequency will depend on the model parameters. In addition, we note that the aperiodic exponent reported in lower frequency bands is more difficult to interpret. To assess low frequency rhythms requires long durations of data, which increases the chance of nonstationarity. Artifacts (e.g., slow drifts) and

analysis choices (e.g., whether to subtract the signal mean) also impact the low frequency power. Finally, while power-law features at low frequencies may reflect the same power-law features at high frequencies, these different phenomena unlikely reflect the same neural mechanisms. We also expect analyzing dynamics away from an equilibrium of the dynamical system will increase the aperiodic exponent. Away from an equilibrium, nonlinear terms in the model have a greater impact on the dynamics and resulting spectrum. These nonlinearities may increase power at high frequencies (e.g., (Kramer, Tort, et al., 2008)) and therefore increase the aperiodic exponent beyond the range derived for the linear dynamics near the equilibrium.

Here we considered additive Gaussian noise as the stochastic inputs to the dynamical models. However, alternative noise sources would provide more accurate approximations of biological systems. For example, for conductance based neural models, more biophysically plausible noise sources would include noise in the membrane current dynamics with variance proportional to the opening and closing rates of ion channels (Pu & Thomas, 2020, 2021). For models of neural population activity, stochastic perturbations may be scaled by the square root of the population size (Benayoun et al., 2010; Candia et al., 2021). For models of ecological population dynamics, noise may be scaled proportional to the birth and death rates (Barendregt & Thomas, 2023; Huynh et al., 2023; Strang et al., 2019). Understanding the impact of more biologically realistic noise (e.g., state-dependent noise) on the power-law behavior remains an important topic for future investigation.

Many well-supported observations of power-laws appear in neuroscience (e.g., avalanches of population voltage discharges (Beggs & Plenz, 2003), amplitudes of narrowband oscillations (Linkenkaer-Hansen et al., 2001)). Here, we consider one type of power-law: the  $1/f$ -like neural field spectrum and a general, noise-driven dynamical system. Under this general model, the aperiodic exponent represents the impact of noise in the observable and latent dynamics, without requiring a sophisticated biological or dynamical mechanism. We propose that the range of aperiodic exponents  $-4 \leq \beta \leq -2$  observed *in vivo* represents the expected dynamics near an equilibrium in a nonlinear dynamical system driven by noise. The generality of the model is consistent with the universality of  $1/f$ -like field spectra, reflecting a basic dynamical feature present in many different neural systems. However, this simplicity may also limit the computational utility of this mechanism and the role of the aperiodic exponent in measuring neural computations.

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## Materials And Methods

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To estimate the aperiodic exponent  $\beta$ , we first compute the spectrum in the standard way. To a simulated voltage time series ( $V_t$ ) with sampling interval  $\Delta$  and duration  $T$ , we subtract the mean, apply a Hanning taper, compute the Fourier transform ( $V_f$ ), and multiply by the complex conjugate ( $V_f^*$ ):

$$P = \frac{2 \Delta^2}{T} V_f V_f^* .$$

We note that the square of the Fourier coefficients is essential for consistent interpretation of the aperiodic exponent across studies; omitting the square is a common mistake identified in previous work (see discussion in (Milotti, 2002)). For frequencies  $f$ , we fit a linear model to the logarithm base 10 of the spectrum ( $\log_{10} P$ ) with predictor logarithm base 10 of the frequency ( $\log_{10} f$ ),

$$\log_{10} P = c + \beta \log_{10} f ,$$

where  $\beta$  is the estimate of the aperiodic exponent. Code to compute the spectrum and estimate the aperiodic exponent is available at <https://github.com/Mark-Kramer/Aperiodic-Exponent-Model>.



### *Derivation of the asymptotic behavior of the cross-spectral matrix*

Consider the two terms of (4) that involve the Jacobian,

$$(\mathbf{A} + i\omega\mathbf{I})^{-1} \text{ and } (\mathbf{A}^T - i\omega\mathbf{I})^{-1}.$$

In general, these  $n$ -by- $n$  matrices are complicated expressions of the constants in  $\mathbf{A}$  and powers of  $\omega$ . To characterize the limiting behavior of these matrices for large values of  $\omega$ , we express each of these two terms using asymptotic notation,

$$\begin{bmatrix} \mathcal{O}(\omega^{-1}) & \mathcal{O}(\omega^{-2}) & \mathcal{O}(\omega^{-2}) & \dots & \mathcal{O}(\omega^{-2}) \\ \mathcal{O}(\omega^{-2}) & \mathcal{O}(\omega^{-1}) & \mathcal{O}(\omega^{-2}) & \dots & \mathcal{O}(\omega^{-2}) \\ \mathcal{O}(\omega^{-2}) & \mathcal{O}(\omega^{-2}) & \mathcal{O}(\omega^{-1}) & \dots & \mathcal{O}(\omega^{-2}) \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ \mathcal{O}(\omega^{-2}) & \mathcal{O}(\omega^{-2}) & \mathcal{O}(\omega^{-2}) & \dots & \mathcal{O}(\omega^{-1}) \end{bmatrix}$$

where terms on the diagonal grow proportional to  $\omega^{-1}$  and terms off the diagonal grow proportional to  $\omega^{-2}$  as  $\omega \rightarrow \infty$ . The noise matrix  $\mathbf{B}$  enters the calculation only as a symmetric matrix  $\mathbf{D}$ ,

$$\mathbf{D} = \mathbf{B} \mathbf{B}^T = \begin{bmatrix} D_{11} & D_{12} & D_{13} & \dots & D_{1n} \\ D_{12} & D_{22} & D_{23} & \dots & D_{2n} \\ D_{13} & D_{23} & D_{33} & \dots & D_{3n} \\ \vdots & \vdots & \vdots & \ddots & \vdots \\ D_{1n} & D_{2n} & D_{3n} & \dots & D_{nn} \end{bmatrix},$$

and we note  $D_{11} = B_{11}^2 + B_{12}^2 + \dots + B_{1m}^2$ , the sum of each squared element in the first row of  $\mathbf{B}$ .

The first row of

$$(\mathbf{A} + i\omega\mathbf{I})^{-1} \mathbf{D}$$

then becomes,

$$\begin{aligned} & [D_{11}\mathcal{O}(\omega^{-1}) + (D_{12} + D_{13} + \dots + D_{1n})\mathcal{O}(\omega^{-2}), \\ & D_{12}\mathcal{O}(\omega^{-1}) + (D_{22} + D_{23} + \dots + D_{2n})\mathcal{O}(\omega^{-2}), \\ & D_{13}\mathcal{O}(\omega^{-1}) + (D_{23} + D_{33} + \dots + D_{3n})\mathcal{O}(\omega^{-2}), \\ & \vdots \\ & D_{1n}\mathcal{O}(\omega^{-1}) + (D_{2n} + D_{3n} + \dots + D_{nn})\mathcal{O}(\omega^{-2})]. \end{aligned}$$

To determine the spectrum of the observable variable ( $S_{11}[\omega]$ ), we compute the first entry of the cross-spectral matrix  $\mathbf{S}[\omega]$  in (4)

$$(\mathbf{A} + i\omega\mathbf{I})^{-1} \mathbf{D} (\mathbf{A}^T - i\omega\mathbf{I})^{-1},$$

which corresponds to multiplying the two vectors,

$$\begin{array}{ll}
[D_{11}\mathcal{O}(\omega^{-1}) + (D_{12} + D_{13} + \dots + D_{1n})\mathcal{O}(\omega^{-2}) , & [\mathcal{O}(\omega^{-1}), \\
D_{12}\mathcal{O}(\omega^{-1}) + (D_{22} + D_{23} + \dots + D_{2n})\mathcal{O}(\omega^{-2}), & \mathcal{O}(\omega^{-2}), \\
D_{13}\mathcal{O}(\omega^{-1}) + (D_{23} + D_{33} + \dots + D_{3n})\mathcal{O}(\omega^{-2}), & \mathcal{O}(\omega^{-2}), \\
\vdots & \vdots \\
D_{1n}\mathcal{O}(\omega^{-1}) + (D_{2n} + D_{3n} + \dots + D_{nn})\mathcal{O}(\omega^{-2})] & \mathcal{O}(\omega^{-2})]
\end{array}
\quad \text{and}$$

element by element. Doing so, we find,

$$S_{11}[\omega] = D_{11}\mathcal{O}(\omega^{-2}) + \mathcal{O}(\omega^{-4}) ,$$

or

$$S_{11}[\omega] = (B_{11}^2 + B_{12}^2 + \dots + B_{1m}^2)\mathcal{O}(\omega^{-2}) + \mathcal{O}(\omega^{-4}) , \quad (\text{A1})$$

where we note that the  $\mathcal{O}(\omega^{-3})$  terms vanish due to the symmetry of  $\mathbf{D}$ .

To illustrate these general results, we consider a 2-dimensional dynamical system with

$$\mathbf{A} = \begin{pmatrix} a & b \\ c & d \end{pmatrix} \text{ and } \mathbf{B} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix} .$$

Evaluating the cross-spectral matrix (4) for the observable variable ( $S_{XX}[\omega]$ ) we find,

$$\begin{aligned}
& S_{XX}[\omega] \\
&= \frac{1}{2\pi} \frac{b^2 (B_{21}^2 + B_{22}^2) - 2 b d (B_{11}B_{21} + B_{12}B_{22}) + (B_{11}^2 + B_{12}^2)(d^2 + \omega^2)}{(b c - a d)^2 + (a^2 + 2 b c + d^2)\omega^2 + \omega^4} .
\end{aligned} \quad (\text{A2})$$

Isolating the  $\omega^2$  term in the numerator, this expression becomes,

$$S_{XX}[\omega] = \frac{\mathcal{O}(1) + (B_{11}^2 + B_{12}^2)\mathcal{O}(\omega^2)}{\mathcal{O}(\omega^4)} = (B_{11}^2 + B_{12}^2)\mathcal{O}(\omega^{-2}) + \mathcal{O}(\omega^{-4}),$$

as  $\omega \rightarrow \infty$ , equivalent to the general expression (A1).

To illustrate the cross-spectral matrix  $S_{XX}[\omega]$  for a specific 2-dimensional dynamical system, we consider the predator-prey model (9). In this case,  $B_{11} = \sigma_x$ ,  $B_{12} = B_{21} = 0$ , and  $B_{22} = \sigma_y = 1$ , so that (A2) becomes,

$$S_{XX}[\omega] = \frac{1}{2\pi} \frac{b^2 + \sigma_x^2 (d^2 + \omega^2)}{(b c - a d)^2 + (a^2 + 2 b c + d^2)\omega^2 + \omega^4}.$$

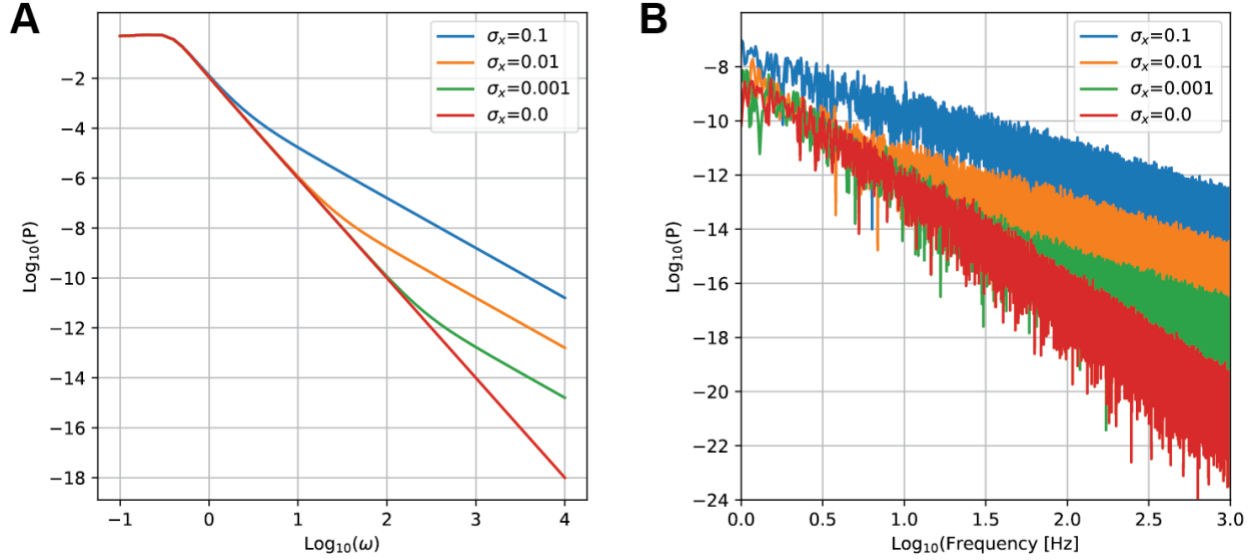
Fixing  $\alpha = 0.25$  and  $\gamma = 0.6$  as in Figure 5A-D, we find

$$a = -\frac{0.25}{0.6}, \quad b = -0.25, \quad c = 1 - \frac{0.25}{0.6}, \quad d = 0,$$

so that the cross-spectrum for the observable (prey) variable becomes,

$$S_{XX}[\omega] = \frac{1}{2\pi} \left( \frac{\sigma_x^2 \omega^2}{0.0212674 - 0.118056 \omega^2 + \omega^4} + \frac{0.0625}{0.0212674 - 0.118056 \omega^2 + \omega^4} \right). \quad (\text{A3})$$

We show in Figure 5A this cross-spectrum for different values of prey noise ( $\sigma_x$ ). When  $\sigma_x = 0$ , the first term in (A3) is zero and the  $\omega^{-4}$  term dominates the spectrum (red curve in Figure 5A). When  $\sigma_x$  increases to 0.001, the first term in (A3) becomes non-zero, and the  $\omega^{-2}$  term impacts the spectrum. However, because  $\sigma_x$  is small, the effect of the  $\omega^{-2}$  term only appears at large  $\omega$ , where the second term of  $S_{XX}[\omega]$  in (A3) is small (green curve in Figure 5A). As  $\sigma_x$  increases, the impact of the first ( $\omega^{-2}$ ) term increases and emerges above the second ( $\omega^{-4}$ ) term at lower frequencies (orange and blue curves in Figure 5A). A knee in the curve occurs at the transition from  $\omega^{-4}$  behavior (at lower frequencies) to  $\omega^{-2}$  behavior (at higher frequencies). We note that estimating a single aperiodic exponent over the high frequency range produces a single estimate for the slope of  $S_{XX}[\omega]$ ; a more representative approach would instead identify the knee in the curve and estimate the two separate slopes. However, in practice, noisy spectral estimates from time series data obfuscate this change in aperiodic exponent (example in Figure 5B).



**Figure 5: Analytic and estimated spectra for the predator-prey model. (A)** The analytic spectrum (A3) for the prey variable at different values of prey noise ( $\sigma_x$ , see legend). When prey noise is 0, the aperiodic exponent is  $-4$  at high frequencies (red curve). As the prey noise increases, a knee appears in the curve and the aperiodic exponent becomes  $-2$  at high frequencies. **(B)** Spectra estimated from numerical simulations of the predator-prey model at the same values of prey noise ( $\sigma_x$ , see legend). The shift to an aperiodic exponent of  $-2$  at high frequencies becomes difficult to detect. For all curves, we fix  $\alpha = 0.25$ ,  $\gamma = 0.6$ , and  $\sigma_y = 1$ , and perform numerical integration using the Euler–Maruyama method with timestep 0.0002 a.u. Code to create this figure is available at <https://github.com/Mark-Kramer/Aperiodic-Exponent-Model/>

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