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Coherence resonance in random Erdős-Rényi neural networks : mean-field theory

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2 ABSTRACT

3 Additive noise is known to tune the stability of nonlinear systems. Using a network of two
4 randomly connected interacting excitatory and inhibitory neural populations driven by additive
5 noise, we derive a closed mean-field representation that captures the global network dynamics.
6 Building on the spectral properties of Erdős-Rényi networks, mean-field dynamics are obtained
7 via a projection of the network dynamics onto the random network's principal eigenmode. We
8 consider Gaussian zero-mean and **Poisson-like** noise stimuli to excitatory neurons and show that
9 these noise types induce coherence resonance. Specifically, the stochastic stimulation induces
10 coherent stochastic oscillations **in the γ -frequency range** at intermediate noise intensity. We further
11 show that this is valid for both global stimulation and partial stimulation, i.e. whenever a subset of
12 excitatory neurons is stimulated only. The mean-field dynamics exposes the coherence resonance
13 dynamics **in the γ -range** by a transition from a stable non-oscillatory equilibrium to an oscillatory
14 equilibrium via a saddle-node bifurcation. We evaluate the transition between non-coherent
15 and coherent state by various power spectra, spike-field coherence and information-theoretic
16 measures.

17 **Keywords:** coherence resonance, phase transition, stochastic process, excitable system, mean-field, random networks

1 INTRODUCTION

18 Synchronisation is a well characterized phenomenon in natural systems [1]. A confluence of experimental
19 studies indicate that synchronization may be a hallmark pattern of self-organisation [2, 3, 4]. While
20 various mechanisms are possible, synchronization may emerge notably through an enhancement of internal
21 interactions or via changes in external stimuli statistics. A specific type of synchronisation can occur due
22 to random external perturbations, leading to a noise-induced coherent activity. Such a phenomenon is
23 called coherence resonance (CR) and has been found experimentally in solid states [5], nanotubes [6]
24 and in **neural systems** [7, 8]. Theoretical descriptions of CR have been developed for single excitable
25 elements [9, 10, 9], for excitable populations [11] and for clustered networks [12].

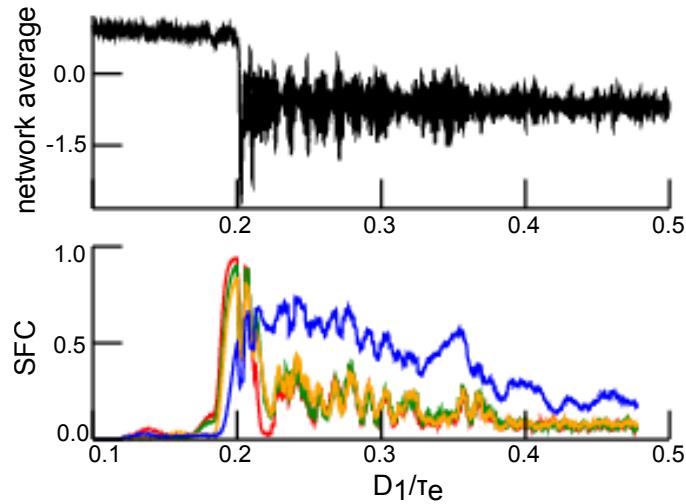


Figure 1. Synchronisation dependent on noise intensity as a marker of coherence resonance. The top panel shows the network average of V in Eq. (1) and the bottom panel provides the spike-field coherence (SFC) in the Θ –(red), α –(green), β –(orange) and γ –(blue) frequency range. For low noise intensity D_1/τ_e there is no SFC, intermediate noise intensity yields strong SFC while large noise intensities diminish SFC again. To gain the SFC values, we have integrated in time the model system with 10^4 time steps while increasing the noise variance according to (7). For illustration reasons, the SFC-values have been averaged by a sliding window of length $\Delta(D_1/\tau_e) = 0.004$. Definitions are given in section 2 and parameters are the same as in Fig. 4 with $q = 1$.

26 In general, stimulus-induced synchronisation is well-known in neural systems [2]. Synchronisation has
 27 been observed intracranially in the presence of noise between single neurons in specific brain areas [13, 14]
 28 and between brain areas [15, 16, 17]. The source of these random perturbations is still under debate. In this
 29 context, it is interesting to mention that [18] have found that the ascending reticular arousal system (ARAS)
 30 affects synchronisation in the visual cortex. The ARAS provides dynamic inputs to many brain areas
 31 areas [19, 20, 21]. It has thus been hypothesized that synchronisation in the visual system represents a CR
 32 effect triggered by ARAS-mediated drive. This hypothesis has been supported recently by [22] showing in
 33 numerical simulations that an intermediate intensity of noise maximises the interaction in a neural network
 34 of Hodgkin-Huxley neurons. Furthermore, recent theoretical work [21] has provided key insights on how
 35 human occipital electrocorticographic γ -activity (40-120Hz) commonly observed with open eyes [21] is
 36 closely linked to CR. Coherence resonance has further been associated with states of elevated information
 37 processing and transfer [22], which are difficult to assess in the absence of mean-field descriptions.
 38 For illustration, Fig. 1(upper panel) shows average network activity for increasing noise intensities D_1 and
 39 one observes a jump from non-oscillatory to oscillatory activity. Moreover, the figure presents very low
 40 coherence in the network under study for weak and strong noise intensities D_1 , whereas high coherence
 41 emerges for intermediate noise intensities (bottom panel). In the present work, we will explain this
 42 noise-induced coherence by a mean-field description.

43 To better understand the mechanisms underlying CR and its impact on information processing, we
 44 consider a simple two-population Erdős-Rényi network of interconnected McCulloch-Pitts neurons. Our
 45 goal is to use this model to provide some insight into the emergence of stimulus-induced synchronisation
 46 in neural systems and its influence on the neural network's information content. The neural network under
 47 study has random connections, a simplification inspired from the lack structure neural circuits possess
 48 at microscopic scales. Previous studies [23] have shown that such systems are capable of noise-induced

49 CR. Building on these results, we here provide a rigorous derivation of a mean-field equation based on an
 50 appropriate eigenmode decomposition to highlight the role of the network's connectivity - Erdős-Rényi
 51 more specifically - eigenspectrum in supporting accurate mean-field representations. We extend previous
 52 results by further considering both global (all neurons are stimulated) and partial (some neurons are
 53 stimulated) stochastic stimulation and its impact on CR similar to some previous studies [24, 25, 26].
 54 This partial stimulation is both more general and realistic than global stimulation as considered in most
 55 previous studies [23, 11, 27]. We apply our results to both zero-mean Gaussian and **Poisson-like** stochastic
 56 stimuli, and derive the resulting mean-field description. It is demonstrated rigorously that partial stochastic
 57 stimulation shifts the system's dynamic topology and promotes CR, compared to global stimulation. We
 58 confirm and explore the presence of CR using various statistical measures.

2 MATERIAL AND METHODS

59 We first introduce the network model under study, motivate the mean-field description, mention the
 60 nonlinear analysis employed and provide details on the statistical evaluation.

61 2.1 The network model

62 Generically, biological neuronal networks are composed of randomly connected excitatory and inhibitory
 63 neurons, which interact through synapses with opposite influence on post-synaptic cells. We assume neural
 64 populations of excitatory \mathcal{E} and inhibitory \mathcal{I} neurons with N neurons in each population. Excitatory neurons
 65 in \mathcal{E} excite each other through the connectivity matrix \mathbf{F} , and excite inhibitory neurons in \mathcal{I} through the
 66 connectivity matrix \mathbf{M} . Similarly, neurons in \mathcal{I} inhibit each other by \mathbf{F} and inhibit excitatory neurons
 67 through the connectivity matrix \mathbf{M} . Hence, \mathbf{F} and \mathbf{M} represent the intra-population and inter-population
 68 synaptic connections, respectively. Mathematically, such neural population interactions are described
 69 by a $2N$ -dimensional non-linear dynamical system governing the evolution of the state variable vectors
 70 $\mathbf{V}, \mathbf{W} \in \mathbb{R}^N$,

$$\begin{aligned} \tau_e \frac{d\mathbf{V}}{dt} &= -\mathbf{V} + \mathbf{F}\mathbf{S}_1[\mathbf{V}] - \mathbf{M}\mathbf{S}_2[\mathbf{W}] + eI^e + \boldsymbol{\xi}^e(t) \\ \tau_i \frac{d\mathbf{W}}{dt} &= -\mathbf{W} + \mathbf{M}\mathbf{S}_1[\mathbf{V}] - \mathbf{F}\mathbf{S}_2[\mathbf{W}] + eI^i + \boldsymbol{\xi}^i(t). \end{aligned} \quad (1)$$

71 This formulation is reminiscent of many rate-based models discussed previously [28], where it is as-
 72 sumed that neuronal activity is asynchronous and synaptic response functions are of first order. The state
 73 variables \mathbf{V} and \mathbf{W} represent excitatory and inhibitory dendritic currents, respectively. The terms $\boldsymbol{\xi}^{e,i}$
 74 represent respective stochastic inputs from various sources, such as ion channel fluctuations, stochastic
 75 input from other brain areas or external stimuli not directly accounted for in the model [29].
 76 More specifically, we assume noise $\boldsymbol{\xi}^{e,i} \in \mathbb{R}^N$, constant input $I^{e,i}$ with $e = (1, \dots, 1)^t$. The con-
 77 nectivity matrices are defined by $\mathbf{F}, \mathbf{M} \in \mathbb{R}^{N \times N}$ while the nonlinear transfer function is given by
 78 $\mathbf{S}_{1,2}[\mathbf{u}] \in \mathbb{R}^N$ with $(\mathbf{S}_1[\mathbf{u}])_n = H_0 S(u_n)$, $(\mathbf{S}_2[\mathbf{u}])_n = S(u_n)$, $H_0 > 0$ and the scalar transfer function
 79 $S(u) > 0 \forall u \in \mathbb{R}$. Specifically, we will consider the transfer function $S(u) = \Theta(u)$ with the Heaviside
 80 function $\Theta(u) = 0 \forall u < 0$, $\Theta(u) = 1 \forall u \geq 0$. In addition, the synaptic time scales are $\tau_{e,i}$.

81

82 The present work considers directed Erdős-Rényi networks (ERN) with connection probability density
 83 $c = 0.95$, i.e. both neuron populations exhibit intra-population and inter-population non-sparse random

84 connections. Let us assume $\mathbf{F} = \mathbf{A}F_0$, $\mathbf{M} = \mathbf{A}M_0$ and \mathbf{A} is the non-symmetric adjacency matrix of the
 85 ERN for which $(\mathbf{A})_{nm} = 0$ with probability $1 - c$ and $(\mathbf{A})_{nm} = 1/cN$ with probability c .
 86 At first, let $\mathbf{A} = \mathbf{S} + \mathbf{U}$ with the symmetric matrix $\mathbf{S} = (\mathbf{A} + \mathbf{A}^t)/2$, the antisymmetric matrix
 87 $\mathbf{U} = (\mathbf{A} - \mathbf{A}^t)/2$ and the eigenvalues λ_A and λ_S of the matrix \mathbf{A} and \mathbf{S} , respectively. Then $\mathcal{R}e(\lambda_A) = \lambda_S$,
 88 i.e. the real part of the eigenvalue spectrum in the directed (i.e. non-symmetric) and non-directed (i.e.
 89 symmetric) random matrix \mathbf{A} and \mathbf{S} is identical. Moreover, for non-directed ERNs with symmetric
 90 adjacency matrix and $N \rightarrow \infty$ its edge spectrum contains the maximum eigenvalue $\lambda_1 = 1$ with eigenvector
 91 $\mathbf{v}_1 = (1, 1, \dots, 1)^t$ [30, 31, 32, 33] and the bulk spectrum has the maximum eigenvalue

$$\begin{aligned} \lambda_2 &= \frac{2\sigma\sqrt{N}}{cN} \\ &= \frac{2\sqrt{1-c}}{\sqrt{cN}} \end{aligned} \quad (2)$$

92 with the corresponding Bernoulli distribution variance $\sigma^2 = c(1 - c)$. It is obvious that $\lambda_2 \ll \lambda_1$ and
 93 $\lambda_2 \approx 0$ for large mean degree cN . Since $\mathcal{R}e(\lambda_A) = \lambda_S$, the finite-size non-symmetric connectivity matrix
 94 \mathbf{F} (\mathbf{M}) has a maximum eigenvalue $\lambda_1 \approx F_0$ and $\lambda_{n>1} \approx 0$ ($\lambda_1 \approx M_0$, $\lambda_{n>1} \approx 0$). If c decreases, then λ_2
 95 increases, i.e. the spectral gap decreases, and this approximation does not hold anymore. The Appendix
 96 illustrates the limits of this approximation in numerical simulations.

97 Figure 2(A) shows the single maximum eigenvalue λ_1 of \mathbf{A} representing the edge spectrum and the other
 98 very small eigenvalues of the bulk spectrum. Hence, the matrix \mathbf{F} has maximum eigenvalue F_0 and the other
 99 eigenvalues vanish. The same holds for matrix $\mathbf{M} = M_0\mathbf{A}$ with a maximum eigenvalue M_0 . Figure 2(B)
 100 shows the real and imaginary part of the eigenvectors. The eigenvectors of the bulk spectrum ($i > 1$)
 101 have uniformly distributed elements in good accordance with theory of symmetric ER networks [34]. The
 102 eigenvector of the edge spectrum is $\Phi_1 = (1, \dots, 1)^t$, see Figure 2(C).

103 Moreover, we assume that each noise process at inhibitory neurons $(\eta^i)_n = \eta_n^i$ at network node n is
 104 Gaussian distributed with zero mean, noise intensity D_2 and uncorrelated in time

$$\langle \xi_n^i(t) \xi_m^i(\tau) \rangle = 2D_2 \delta_{nm} \delta(t - \tau).$$

105 Conversely each noise process at excitatory neurons ξ_n^e belongs to a certain class \mathcal{G}_m , $m = 1, \dots, M$ of M
 106 classes [23]. Noise processes in a specific class \mathcal{G}_m , i.e. $n \in \mathcal{G}_m$, share their mean $\bar{\xi}_m^e$ and variances D_1^m ,
 107 i.e.

$$\langle \xi_k^e(t) \xi_l^e(\tau) \rangle = 2D_1^m \delta_{kl} \delta(t - \tau) \quad , \quad k, l \in \mathcal{G}_m.$$

108 In the following, we assume two classes $M = 2$ with $\bar{\xi}_1^e \neq 0$, $D_1^1 = D_1$ and $\bar{\xi}_2^e = 0$, $D_1^2 = 0$, i.e. only
 109 a subset of nodes $n \in \mathcal{G}_1$ are stimulated. Hence we consider a partial stimulation at number of nodes
 110 $N_1 = |\mathcal{G}_1|$.

111 In biological neural systems, the input to a neural population is well-described by incoming spike trains
 112 that induce dendritic currents at synaptic receptors. According to renewal theory, neurons emit spike trains
 113 whose interspike interval obeys a Poisson distribution [35]. Then incoming spike trains at mean spike
 114 rate r induce random responses at excitatory synapses with time constant τ_{in} . This random process $I_{\text{in}}(t)$
 115 has the ensemble mean $E[I_{\text{in}}] = w_{\text{in}} r \tau_{\text{in}}$ and ensemble variance $\text{Var}[I_{\text{in}}] = w_{\text{in}}^2 r \tau_{\text{in}} / 2$ [36] assuming the

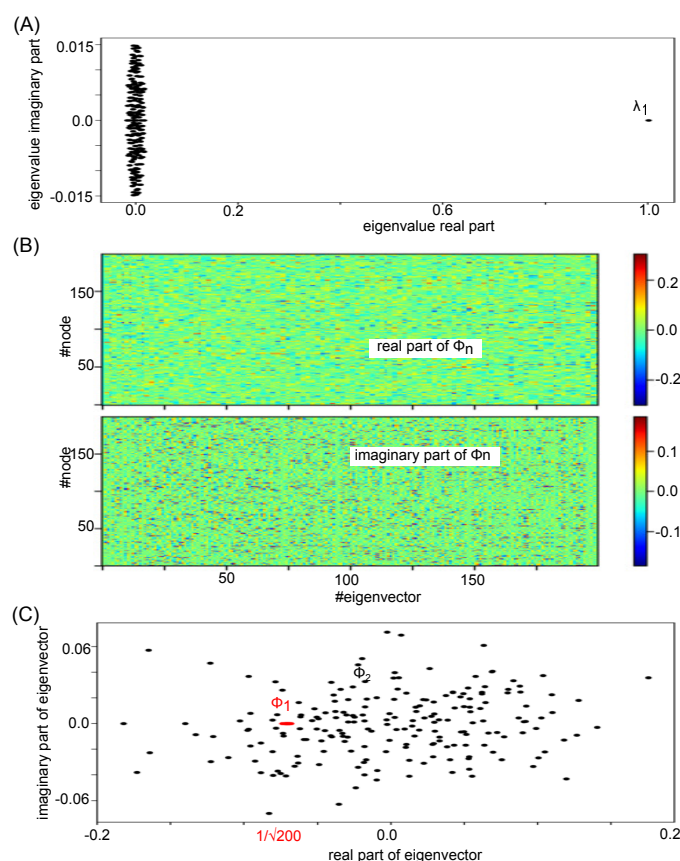


Figure 2. Eigenvalue spectrum of an Erdős-Rényi adjacency matrix A under study and its eigenbasis. (A) The plot shows the eigenvalues in the complex plane demonstrating a clear spectral gap between the first eigenvalue λ_1 and the other eigenvalues $\lambda_{n>1}$. (B) The panels show the real (top) and imaginary (bottom) part of all unit-normalized eigenvectors for illustration. They appear to be random reflecting the random network topology. (C) The normalized eigenvector $\Phi_1 \approx (1, \dots, 1)/\sqrt{N}$ with maximum eigenvalue $\lambda_1 \approx 1$ plotted in complex plane together with the eigenvector Φ_2 of the second largest eigenvalue $\lambda_2 = 0.015 + i0.0006$. Each dot corresponds to a complex-numbered vector entry in the complex plane. This result confirms the choice $\Phi_1 \approx (1, \dots, 1)$ in Eqs. (9).

116 synaptic coupling weight w_{in} . Since a Poisson distribution converges to a Gaussian distribution for large
 117 enough mean, we implement this input current as a Gaussian random process with mean $E[I_{in}]$ and variance
 118 $\text{Var}[I_{in}]$ while ensuring the validity of this approximation by a large enough input firing rate λ_{in} . It is
 119 important to point out that for Poisson noise, in contrast to the zero-mean Gaussian noise, both mean and
 120 variance are proportional to the input firing rate.

121

122 2.2 Conventional Mean-Field Analysis

123 To compare mesoscopic neural population dynamics to macroscopic experimental findings, it is
 124 commonplace to describe the network activity by the mean population response, i.e. the mean-field dynam-
 125 ics [37, 38, 39]. A naive mean-field approach was performed in early neuroscience studies [40, 41, 42], in
 126 which one blindly computes the mean network activity to obtain

$$\begin{aligned}
 \tau_e \frac{dE[V]}{dt} &= -E[V] + fS_1[V] - mS_2[W] + eI^e \\
 \tau_i \frac{dE[W]}{dt} &= -E[W] + mS_1[V] - fS_2[W] + eI^i
 \end{aligned}
 \tag{3}$$

127 with the network average $E[x] = \sum_k x_k/N$ and $(\mathbf{f})_k = \sum_l \mathbf{F}_{lk}/N$, $(\mathbf{m})_k = \sum_l \mathbf{M}_{lk}/N$ assuming
 128 zero-mean external noise with $\sum_k (\xi^{e,i})_k = 0$. In addition, one may assume identical network interactions
 129 with $(\mathbf{f})_k = f_0/N = \text{const}$, $(\mathbf{m})_k = m_0/N = \text{const}$ and the simplifying but questionable linear assumption

$$E[S_{1,2}(x)] = S_{1,2}(E[x]). \quad (4)$$

130 Combined, these assumptions lead to mean-field equations

$$\begin{aligned} \tau_e \frac{dE[V]}{dt} &= -E[V] + f_0 S_1[E[V]] - m_0 S_2[E[W]] \\ &\quad + eI^e \\ \tau_i \frac{dE[W]}{dt} &= -E[W] + m_0 S_1[E[V]] - f_0 S_2[E[W]] \\ &\quad + eI^i \end{aligned} \quad (5)$$

131 In this approximate description, additive noise does not affect the system dynamics.

132 The assumption (4) is very strong and typically not valid. In a more reasonable ansatz

$$\begin{aligned} E[S_{1,2}(x)] &= E \left[S_{1,2}(x_0) + \sum_{n=1}^{\infty} \frac{1}{n!} S_{1,2}^{(n)}(x_0) (x - x_0)^n \right] \\ &= S_{1,2}(x_0) + \sum_{n=1}^{\infty} \frac{1}{n!} S_{1,2}^{(n)}(x_0) E[(x - x_0)^n] \\ &= \mathcal{F}(E[x], E[x^2], E[x^3], \dots) \end{aligned} \quad (6)$$

133 with $S_{1,2}^{(n)} = \partial S_{1,2}^n(x)/\partial x^n$ computed at an arbitrary point $x = x_0$ and a function $\mathcal{F}_{1,2} \in \mathbb{R}$. Hence the
 134 dynamics of the mean-field $E[V]$ depends on the higher-order statistical orders $E[V^n]$ via the nonlinear
 135 function $E[S_{1,2}(V)]$. This is called the closure problem that is solvable in specific cases only [43].

136 Motivated by previous studies on stochastic bifurcations [44, 45, 46, 47, 48, 49, 50, 51, 52, 53], in which
 137 additive noise may tune the stability close to the bifurcation point, the present work shows how additive
 138 noise strongly impacts the nonlinear dynamics of the system for arbitrary noise intensity and away from
 139 the bifurcation. Previous *ad-hoc* studies have already used mean-field approaches [23, 54, 55] which
 140 circumvents the closure problem (6) through a different mean-field ansatz. These motivational studies left
 141 open a more rigorous derivation. This derivation will be given in the present work: presenting in more
 142 detail its power and its limits of validity.

143 2.3 Equilibria, stability and quasi-cycles

144 The dynamic topology of a model differential equation system may be described partially by the number
 145 and characteristics of its equilibria. In general, for the non-autonomous differential equation system

$$\dot{\mathbf{z}} = \mathbf{A}\mathbf{z} + \mathbf{N}(\mathbf{z}) + \mathbf{I}(t)$$

146 with state variable $\mathbf{z} \in \mathbb{R}^N$, the driving force $\mathbf{I} \in \mathbb{R}^N$, the nonlinear vector $\mathbf{N} \in \mathbb{R}^N$ and the matrix
 147 $\mathbf{A} \in \mathbb{R}^{N \times N}$, it is insightful to consider the equilibria of the corresponding autonomous system \mathbf{z}_0 with

parameter	description	value
τ_e	exc. synaptic time constant	5ms
τ_i	inhib. synaptic time constant	20ms
F_0	intra-population conn. weight	2.17
M_0	inter-population conn. weight	3.87
c	connection probability	0.95
N	number of network nodes	200
I_e	constant exc. input	1.1
I_i	constant inhib. input	0.4
D_2	inhib. noise variance	0.2
w_{in}	Poisson input weight	2.1
τ_{in}	synaptic time scale of input	5ms
Δt	numerical integration step	0.5ms

Table 1. Parameter set of model (1).

148 $\dot{z} = 0$ yielding the implicit condition

$$Az_0 = -N(z_0).$$

149 The stability of an equilibrium z_0 is given by the eigenvalue spectrum of the corresponding Jacobian

$$J = A + \nabla N^0$$

150 where $(\nabla N^0)_{ij} = \partial N_i(z)/\partial z_j$ computed at z_0 . The eigenvalues $\{\lambda_k\}$ of J can be written as $\lambda_k = a_k +$
 151 $i2\pi\nu_k$ with the damping a_k and the eigenfrequency ν_k . Asymptotically stable equilibria have $\Re(\lambda_k) < 0$,
 152 e.g. stable foci have $a_k < 0$, $\nu_k \in \mathbb{R}$. Linear response theory tells that noise-driven linear systems, whose
 153 deterministic dynamics exhibit a stable focus, exhibit quasi-cycles with a spectral power peak close to
 154 the eigenfrequency, see e.g. [51, 56, 57]. The smaller the noise intensity, the closer is the spectral peak
 155 frequency to the eigenfrequency. Hence, the eigenfrequency ν_k provides a reasonable estimate of the
 156 quasi-cycle spectral peak.

157 2.4 Numerical simulations

158 The Langevin equations (1) have been integrated over time utilizing the Euler-Maruyama scheme [58].
 159 Table 1 presents the parameters used. In certain cases, the noise variance has been changed over time t
 160 according to

$$D_1(t) = D_{\min} + \frac{D_{\max} - D_{\min}}{T}t \quad (7)$$

161 with the maximum integration time T and the maximum and minimum noise variance values D_{\max} and
 162 D_{\min} , respectively.

163

164 2.5 Numerical spectral data analysis

165 Since prominent oscillations of the network mean activity indicates synchronised activity in the population,
 166 we have computed the power spectrum of the network mean activity $\bar{V}(t) = \sum_{n=1}^N V_n(t)/N$ employing

167 the Bartlett-Welch method with overlap rate 0.8. To gain a power spectrum with frequency resolution Δf ,
 168 the Bartlett-Welch segments were chosen to the length $1/\Delta f$ and the time series had a duration of 5s for
 169 the zero-mean Gaussian noise and 8s for the Poisson noise stimulation.

170 In addition to the power spectrum, the synchronisation between single neuron spike activity and the
 171 dendritic current reflects the degree of coherence in the system. To this end, we have computed the Spike
 172 Field Coherence (SFC) [59]. To estimate the SFC, we have chosen a time window of 5s for zero-mean
 173 Gaussian stimulation and 8s for Poisson stimulation **and computed the spike-triggered average and power**
 174 **spectra in these time windows to compute the SFC for each frequency. Then we have averaged the SFC in**
 175 **the Θ - (4Hz-8Hz), α - (8Hz-12Hz), β - (12Hz-20Hz) and γ - (25Hz-60Hz) frequency band to gain an**
 176 **average SFC in the corresponding band**. This standard measure estimates the coherence between **spikes,**
 177 **that occur if $H[V_n](t) = 1$,** and their corresponding dendritic currents $V_n(t)$ at the same cell averaged
 178 over all cells in the excitatory population. Significant differences of SFC at different noise intensities are
 179 evaluated by an unpaired Welch t-test with $\alpha = 0.05$.

180

181 2.6 Information measures

182 Coherence quantifies the degree of mutual behavior of different elements. Interestingly, recent studies of
 183 biological neural systems have shown that synchronisation and information content are related [60, 61].
 184 For instance, under general anaesthesia asynchronous cortical activity in conscious patients is accompanied
 185 by less stored information and much available information whereas synchronous cortical activity in
 186 unconscious patients exhibits more stored information and less available information [62, 63, 19, 64, 20].
 187 We are curious how much information is stored and available in coherence resonance described in the
 188 present work. The result may indicate a strong link between coherence and information content. To this end,
 189 we compute the amount of stored information in the excitatory population as the predictable information
 190 and the amount of available information as the population's entropy, cf. [64].

191 The predictable information in the excitatory population is computed as the Active Information Storage
 192 AIS [65, 66] utilizing the Gaussian Copula Mutual Information (GCMI) estimation [67]. Assuming a single
 193 time series $V_i(t)$

$$\text{AIS}_i = \text{MI}(V_i(t); \mathbf{V}_{i\Delta}^{(k)}), \quad \mathbf{V}_{i\Delta}^{(k)} = (V_i(t - \Delta), V_i(t - 2\Delta), \dots, V_i(t - k\Delta)), \quad (8)$$

194 where MI is the mutual information [64, 68], k is the embedding dimension and Δ is the embedding delay.
 195 The value AIS_i describes how much the dendritic current $V_i(t)$ in excitatory neuron i is influenced by its
 196 past. To gain an estimate of stored information in the excitatory population, we evaluate the average stored
 197 information in the population and its variance

$$\begin{aligned} \text{AIS} &= \frac{1}{N} \sum_{i=1}^N \text{AIS}_i \\ \sigma_{\text{AIS}}^2 &= \frac{1}{N-1} \sum_{i=1}^N (\text{AIS}_i - \text{AIS})^2. \end{aligned}$$

198 with $N = 200$. Significant AIS differences at different noise intensities are evaluated by an unpaired Welch
 199 t-test with $\alpha = 0.05$.

200 Moreover, we compute the available information in the excitatory cortex of the dendritic current $V_i(t)$ at
201 excitatory neuron i as its entropy H_i utilizing the GCMI estimation. Its population average and variance
202 reads

$$H = \frac{1}{N} \sum_{i=1}^N H_i$$
$$\sigma_H^2 = \frac{1}{N} \sum_{i=1}^N (H_i - H)^2 .$$

203 and entropy differences at different noise intensities are evaluated statistically by an unpaired Welch t-test
204 with $\alpha = 0.05$.

205 In subsequent sections, we have computed AIS and H for embedding dimension $k \in [1; 60]$ and
206 $\Delta \in \{\Delta t, 2\Delta t, 5\Delta t\}$ with $k\Delta = 60$ and find consistent significance test results. Specifically, we have
207 chosen $\Delta = \Delta t$ and $k = 1$ in the shown results.

3 RESULTS

208 The subsequent section shows the derivation of the mean-field equations, before they are applied to describe
209 network dynamics for two types of partial stimulation.

210 3.1 Mean-field description

211 To derive the final equations, we first introduce the idea of a mode projection before deriving the
212 mean-field equations as a projection on the principal mode. The extension to partial stimuli extends the
213 description.

214 Mode decomposition

215 In the model (1), the system activity $\mathbf{V} \in \mathcal{U}$ in space \mathcal{U} may be expanded into a mode basis $\{\Phi_n^e\}$, $n =$
216 $1, \dots, N$, $\Phi_n^e \in \mathbb{C}^N$,

$$\mathbf{V} = \sum_{n=1}^N a_n \Phi_n^e$$

217 with complex mode amplitude $a_n \in \mathbb{C}$ and a biorthogonal basis $\{\Psi_n^e\}$, $\Psi_n^e \in \mathbb{C}^N$ and

$$\Psi_k^{e\dagger} \Phi_n^e = \delta_{kn}, \quad k, n = 1, \dots, N.$$

218 Here, \dagger denotes the transpose complex conjugate. The same holds for \mathbf{W} with the basis $\{\Phi_n^i\}$, $n =$
219 $1, \dots, N$, $\Phi_n^i \in \mathbb{C}^N$,

$$\mathbf{W} = \sum_{n=1}^N b_n \Phi_n^i$$

220 with the complex mode amplitude $b_n \in \mathbb{C}$ and the biorthogonal basis $\{\Psi_n^i\}$, $\Psi_n^i \in \mathbb{C}^N$ and

$$\Psi_k^{i\dagger} \Phi_n^i = \delta_{kn}, \quad k, n = 1, \dots, N.$$

221 Projecting \mathbf{V} , \mathbf{W} onto the respective basis $\{\Psi_k^e\}$ and $\{\Psi_k^i\}$, we obtain amplitude equations

$$\begin{aligned} \tau_e \frac{da_k}{dt} &= -a_k + \Psi_k^{e\dagger} \mathbf{F} \mathbf{S}_1[\mathbf{V}] - \Psi_k^{e\dagger} \mathbf{M} \mathbf{S}_2[\mathbf{W}] \\ &\quad + I^e + \Psi_k^{e\dagger} \boldsymbol{\xi}^e(t) \\ \tau_i \frac{db_k}{dt} &= -b_k + \Psi_k^{i\dagger} \mathbf{M} \mathbf{S}_1[\mathbf{V}] - \Psi_k^{i\dagger} \mathbf{F} \mathbf{S}_2[\mathbf{W}] \\ &\quad + I^i + \Psi_k^{i\dagger} \boldsymbol{\xi}^i(t). \end{aligned}$$

222 Now let us assume that Ψ_k^e, Φ_k^e are eigenvectors of \mathbf{F} with eigenvalue $\lambda_k^e \in \mathbb{C}$

$$\begin{aligned} \mathbf{F} \Phi_k^e &= \lambda_k^e \Phi_k^e \\ \Psi_k^{e\dagger} \mathbf{F} &= \lambda_k^e \Psi_k^{e\dagger} \end{aligned}$$

223 and Ψ_k^i, Φ_k^i are eigenvectors of M with eigenvalue $\lambda_k^i \mathbb{C}$

$$\begin{aligned} M\Phi_k^i &= \lambda_k^i \Phi_k^i \\ \Psi_k^{i\dagger} M &= \lambda_k^i \Psi_k^{i\dagger}. \end{aligned}$$

224 Then

$$\begin{aligned} \lambda_1^e &= F_0, \quad \Phi_1^e = e, \quad \Psi_1^e = e/N \\ \lambda_n^e &\approx 0, \quad n = 2, \dots, N, \end{aligned} \tag{9}$$

225 cf. section 2.1, where we have utilized the bi-orthogonality of the basis. Equivalently,

$$\begin{aligned} \lambda_1^i &= M_0, \quad \Phi_1^i = e, \quad \Psi_1^i = e/N \\ \lambda_n^i &\approx 0, \quad n = 2, \dots, N. \end{aligned}$$

226 We observe that $\Psi_1^{i\dagger} = \Psi_1^{e\dagger}$ and $\Phi_1^e = \Phi_1^i$.

227 The vector space \mathcal{U} can be decomposed into complement subspaces $\mathcal{Z}, \mathcal{Z}_\perp$ with $\mathcal{U} = \mathcal{Z} \oplus \mathcal{Z}_\perp$ and
 228 $\Psi_1^e, \Psi_1^i \in \mathcal{Z}$. Then $\Psi_{k>1}^e, \Psi_{k>1}^i \in \mathcal{Z}_\perp$. Each vector $\Psi_{k>1}^i$ can be described in the basis $\Psi_{k>1}^e$ in \mathcal{Z}_\perp and
 229 one gains

$$\begin{aligned} \Psi_{k>1}^{i\dagger} F &= \sum_{n=2}^N c_n \Psi_n^{e\dagger} F \\ &= \sum_{n=2}^N c_n \lambda_n^e \Psi_n^{e\dagger} \\ &= 0 \end{aligned}$$

230 due to (9) and equivalently

$$\begin{aligned} \Psi_{k>1}^{e\dagger} M &= \sum_{n=2}^N c_n \Psi_n^{i\dagger} M \\ &= \sum_{n=2}^N c_n \lambda_n^i \Psi_n^{i\dagger} \\ &= 0 \end{aligned}$$

231 with some coefficients $c_n \in \mathbb{C}$. This yields

$$\begin{aligned} \tau_e \frac{da_1}{dt} &= -a_1 + \frac{\lambda_1^e}{N} e^t \mathbf{S}_1[\mathbf{V}] - \frac{\lambda_1^i}{N} e^t \mathbf{S}_2[\mathbf{W}] \\ &\quad + I^e + m_e(t) \end{aligned} \quad (10)$$

$$\begin{aligned} \tau_i \frac{db_1}{dt} &= -b_1 + \frac{\lambda_1^i}{N} e^t \mathbf{S}_1[\mathbf{V}] - \frac{\lambda_1^e}{N} e^t \mathbf{S}_2[\mathbf{W}] \\ &\quad + I^i + m_i(t) \end{aligned} \quad (11)$$

$$\tau_e \frac{da_k}{dt} = -a_k + \Psi_k^{e\dagger} \xi^e(t), \quad k = 2, \dots, N \quad (12)$$

$$\tau_i \frac{db_k}{dt} = -b_k + \Psi_k^{i\dagger} \xi^i(t), \quad k = 2, \dots, N \quad (13)$$

232 with $m_{e,i}(t) = e^t \xi^{e,i}(t)/N$.

233 The mean-field equations

234 Equations (12), (13) describe an Ornstein-Uhlenbeck process with solution

$$\begin{aligned} a_k(t) &= \int_{-\infty}^t e^{-(t-\tau)/\tau_e} \Psi_k^{e\dagger} \xi^e(\tau) d\tau \\ b_k(t) &= \int_{-\infty}^t e^{-(t-\tau)/\tau_i} \Psi_k^{i\dagger} \xi^i(\tau) d\tau \end{aligned} \quad (14)$$

235 for $t \rightarrow \infty$. In Eqs. (10), (11) the terms \mathbf{V} , \mathbf{W} can be written as

$$\begin{aligned} \mathbf{V} &= \sum_{n=1}^N a_n(t) \Phi_n^e = a_1 \Phi_1^e + \sum_{n=2}^N a_n(t) \Phi_n^e \\ \mathbf{W} &= \sum_{n=1}^N b_n(t) \Phi_n^i = b_1 \Phi_1^i + \sum_{n=2}^N b_n(t) \Phi_n^i. \end{aligned} \quad (15)$$

236 Inserting expressions in Eqs. (14) into these expressions leads to

$$\begin{aligned} \sum_{n=2}^N a_n(t) \Phi_n^e &= \int_{-\infty}^t e^{-(t-\tau)/\tau_e} \sum_{n=2}^N \Phi_n^e \Psi_n^{e\dagger} \xi(\tau) d\tau. \\ \sum_{n=2}^N b_n(t) \Phi_n^i &= \int_{-\infty}^t e^{-(t-\tau)/\tau_i} \sum_{n=2}^N \Phi_n^i \Psi_n^{i\dagger} \xi(\tau) d\tau. \end{aligned} \quad (16)$$

237 By virtue of the completeness of the basis, it is

$$\begin{aligned} \sum_{n=2}^N \Phi_n^e \Psi_n^{e\dagger} &= \mathbf{I} - \Phi_1^e \Psi_1^{e\dagger} \\ \sum_{n=2}^N \Phi_n^i \Psi_n^{i\dagger} &= \mathbf{I} - \Phi_1^i \Psi_1^{i\dagger} \end{aligned}$$

238 with the unity matrix $\mathbf{I} \in \mathbb{R}^{N \times N}$. Then inserting these identities into (16)

$$\begin{aligned} \sum_{n=2}^N a_n(t) \Phi_n^e &= \int_{-\infty}^t e^{-(t-\tau)/\tau_e} \xi^e(\tau) d\tau \\ &\quad - \int_{-\infty}^t e^{-(t-\tau)/\tau_e} \Phi_1^e m_e(\tau) d\tau \\ \sum_{n=2}^N b_n(t) \Phi_n^i &= \int_{-\infty}^t e^{-(t-\tau)/\tau_i} \xi^i(\tau) d\tau \\ &\quad - \int_{-\infty}^t e^{-(t-\tau)/\tau_i} \Phi_1^i m_i(\tau) d\tau. \end{aligned} \tag{17}$$

239 We define $\eta^{e,i}(t) = \xi^{e,i}(t) - \xi_0^{e,i}$, $e^t \eta^{e,i}(t) = N \rho^{e,i}(t)$ with $\rho^{e,i} \sim \mathcal{N}(0, D_{1,2}/N)$ and temporally
 240 constants $\xi_0^{e,i}$, i.e. $\rho^{e,i}$ are finite size fluctuations with variance $D_{1,2}/N$ and $\rho^{e,i} \rightarrow 0$ for $N \rightarrow \infty$. With
 241 the definitions

$$\mathbf{w}_{e,i}(t) = \int_{-\infty}^t e^{-(t-\tau)/\tau_{e,i}} \eta^{e,i}(\tau) d\tau \tag{18}$$

$$\begin{aligned} \mathbf{s}_{e,i}(t) &= \tau_e (\xi_0^{e,i} - e \bar{\xi}_0^{e,i}) \\ &\quad - e \int_{-\infty}^t e^{-(t-\tau)/\tau_{e,i}} \rho^{e,i}(\tau) d\tau \end{aligned} \tag{19}$$

242 with $\bar{\xi}_0^{e,i} = \sum_{n=1}^N \xi_{0,n}^{e,i}/N$ and inserting Eq. (17) into Eqs. (15)

$$\begin{aligned} \mathbf{V}(t) &= a_1(t) \mathbf{e} + \mathbf{s}_e(t) + \mathbf{w}_e(t) \\ \mathbf{W}(t) &= b_1(t) \mathbf{e} + \mathbf{s}_i(t) + \mathbf{w}_i(t) \end{aligned} \tag{20}$$

243 and the mean-field equations can be written as

$$\begin{aligned} \tau_e \frac{da_1}{dt} &= -a_1 + \frac{F_0}{N} e^t \mathbf{S}_1 [a_1(t) \mathbf{e} + \mathbf{s}_e(t) + \mathbf{w}_e(t)] \\ &\quad - \frac{M_0}{N} e^t \mathbf{S}_2 [b_1(t) \mathbf{e} + \mathbf{s}_i(t) + \mathbf{w}_i(t)] \\ &\quad + I^e + \bar{\xi}_0^e + \rho^e(t) \\ \tau_i \frac{db_1}{dt} &= -b_1 + \frac{M_0}{N} e^t \mathbf{S}_1 [a_1(t) \mathbf{e} + \mathbf{s}_e(t) + \mathbf{w}_e(t)] \\ &\quad - \frac{F_0}{N} e^t \mathbf{S}_2 [b_1(t) \mathbf{e} + \mathbf{s}_i(t) + \mathbf{w}_i(t)] \\ &\quad + I^i + \bar{\xi}_0^i + \rho^i(t) \end{aligned} \tag{21}$$

244 By virtue of the finite-size fluctuations over time $\rho^{e,i}(t)$ the system's mean-field obeys stochastic dynamics.

245 Equations (14) describe an Ornstein-Uhlenbeck process of mode k and thus $\mathbf{w}_{e,i}(t)$ describes a multivari-
 246 ate Ornstein-Uhlenbeck process over time. In addition, $\mathbf{w}_{e,i}(t)$ is stationary over time and, since all modes
 247 k share identical properties, it is stationary over the network. Consequently, the process is ergodic and the
 248 stationary probability density function $p(\mathbf{w}_{e,i})$ of $\mathbf{w}_{e,i}$ can be computed over the network yielding

$$\begin{aligned} \frac{1}{N} \mathbf{e}^t \mathbf{S}_1 [x\mathbf{e} + \mathbf{w}] &= \frac{1}{N} \sum_{n=1}^N S[x + w_n] \\ &\approx \int_{-\infty}^{\infty} S(x + w) p_e(w) dw \\ &= G_1(x), \end{aligned} \quad (22)$$

249 where the approximation is good for large N . Specifically, for Gaussian zero-mean uncorrelated noise ξ_e
 250 with variance D [69]

$$p_e(w) = \frac{1}{\sqrt{2\pi}\sigma} e^{-w^2/2\sigma^2}, \quad \sigma^2 = D/\tau_e.$$

251 Similarly,

$$\begin{aligned} \frac{1}{N} \mathbf{e}^t \mathbf{S}_2 [x\mathbf{e} + \mathbf{w}] &\approx \int_{-\infty}^{\infty} S(x + w) p_i(w) dw \\ &= G_2(x) \end{aligned} \quad (23)$$

252 Moreover, if the mean input is $\xi_0^{e,i} = \alpha^{e,i} \mathbf{e}$ and $N \rightarrow \infty$, then $\mathbf{s}_{e,i} = 0$ and $\rho^{e,i} = 0$ and consequently
 253 the mean-field equation

$$\begin{aligned} \tau_e \frac{da_1}{dt} &= -a_1 + F_0 G_1(a_1) - M_0 G_2(b_1) + I^e + \alpha^e \\ \tau_i \frac{db_1}{dt} &= -b_1 + M_0 G_1(a_1) - F_0 G_2(b_1) + I^i + \alpha^i \end{aligned} \quad (24)$$

254 obeys deterministic dynamics. However, the above formulation depends implicitly on the additive noise
 255 through the convolution of the transfer function.

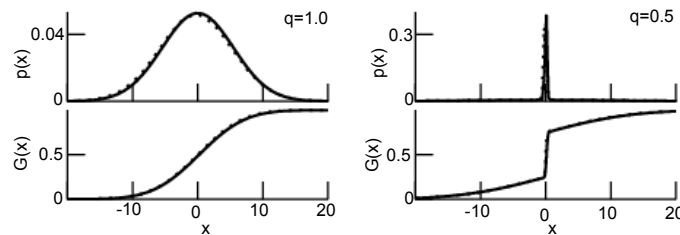


Figure 3. The probability density function p (26) and the resulting transfer function G (27). For $q = 1.0$ $D_1/\tau_e = 0.15$ and for $q = 0.5$ $D_1/\tau_e = 0.5$.

256 Partial stimuli

257 Each noise baseline stimulus at inhibitory neurons $(\xi^i)_n = \xi_n^i$ at network node n is Gaussian distributed
 258 with zero mean and variance D_2 (cf. section 2.1). Then $\bar{\xi}_0^i = 0$, $\mathbf{s}_i(t) = \bar{\rho}^i(t) \sim \mathcal{N}(0, D_2/\tau_i N)$ and,
 259 considering Eq. (18), the corresponding probability density function in Eq. (23) is $p_i(w) = \mathcal{N}(0, D_2/\tau_i)$.
 260 Here $\mathcal{N}(0, \sigma^2)$ denotes a normal distribution with zero mean and variance σ^2 .

261 Additionally, stochastic stimuli driving excitatory neurons in class \mathcal{G}_1 are ergodic (cf. section 2.1). Then
 262 the mean and variance of class \mathcal{G}_1 is

$$\begin{aligned}\bar{\xi}_1^e &= \frac{1}{N_1} \sum_{n \in \mathcal{G}_1} \xi_n^e \\ D_1 &= \frac{1}{N_1} \sum_{n \in \mathcal{G}_1} (\xi_n^e)^2.\end{aligned}\tag{25}$$

263 Using Eq. (18) and Eq. (19) and assuming $N \rightarrow \infty$, then

$$\mathbf{w}^e(t) + \mathbf{s}^e(t) = \int_{-\infty}^t e^{-(t-\tau)/\tau_e} (\boldsymbol{\eta}^e(\tau) + \Delta \boldsymbol{\xi}) d\tau$$

264 whose probability density function $p_e(w)$ is [23]

$$\begin{aligned}p_e(w) &= \sum_{m=1}^2 q_m \mathcal{N}(\bar{\xi}_m^e, D_1^m/\tau_e) \\ &= q \mathcal{N}(\bar{\xi}_1^e, D_1/\tau_e)[w] + (1-q)\delta(w)\end{aligned}\tag{26}$$

265 with $q = N_1/N$, $q_1 = q$, $q_2 = 1 - q$. Here, $\Delta \boldsymbol{\xi} = (1 - q, 1 - q, \dots, -q, -q)\xi_1^e$ with terms $1 - q$ of number
 266 N_1 and assuming that the nodes $n = 1, \dots, N_1$ receive stochastic input. In addition the constant input in
 267 the mean-field equation is $\bar{\xi}_0^e = q\xi_1^e$.

268 Then, utilizing Eqs. (22), (23) and specifying S to a step function (cf. section 2.1), the mean-field transfer
 269 functions in Eq. (24) read

$$\begin{aligned}G_1(a_1) &= \frac{H_0 q}{2} \left[1 - \operatorname{erf} \left(-\frac{a_1 - \xi_1^e}{\sqrt{2D_1/\tau_e}} \right) \right] \\ &\quad + (1 - q)\Theta(a_1) \\ G_2(b_1) &= \frac{1}{2} \left[1 - \operatorname{erf} \left(-\frac{b_1}{\sqrt{2D_2/\tau_i}} \right) \right].\end{aligned}\tag{27}$$

270 Here, $\Theta(\cdot)$ denotes the Heaviside step function. Figure 3 shows examples for p_e and G_1 .

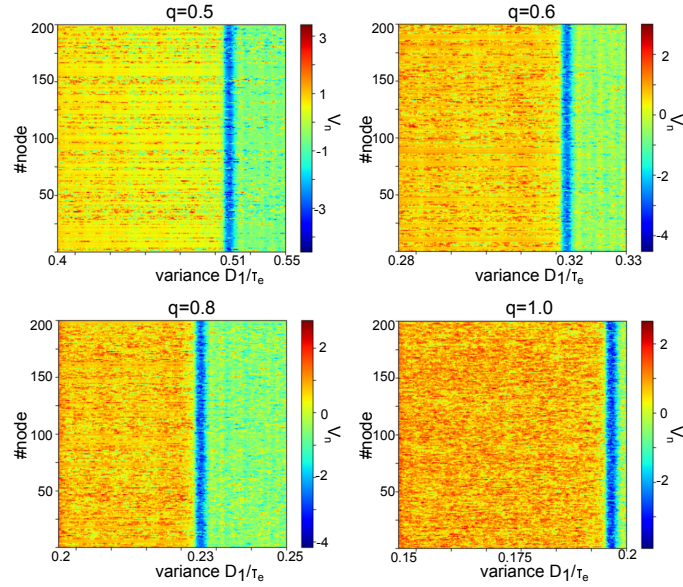


Figure 4. Enhanced zero-mean Gaussian noise induces phase transitions in spatiotemporal dynamics. The panels show the network activity $\mathbf{V}(t)$ according to Eqs. (1) with temporally increasing noise variances D_1/τ_e for different stimulus ratios q .

271 Essentially, the mean-field obeys

$$\begin{aligned}
 \tau_e \frac{da_1}{dt} &= -a_1 + F_0 G_1(a_1) - M_0 G_2(b_1) + I^e \\
 &\quad + \rho^e(t) \\
 \tau_i \frac{db_1}{dt} &= -b_1 + M_0 G_1(a_1) - F_0 G_2(b_1) + I^i \\
 &\quad + \rho^i(t)
 \end{aligned}
 \tag{28}$$

272 utilizing (27).

273 3.2 Zero-mean Gaussian partial stimulation

274 At first, we consider the case of a partial noise stimulation with zero network mean, i.e. $e^t \bar{\xi}^e = 0$ and
 275 $s_e(t) \sim \mathcal{N}(0, D_1/\tau_e N_1)$ and $\xi_1^e = \bar{\xi}_0^e = 0$. Then D_1 parametrizes the noise intensity only. Figure 4 shows
 276 the network evolution of $\mathbf{V}(t)$ for increasing noise intensities, cf. Eqs. (7). Starting from a high activity
 277 state, increasing the noise intensity yields a phase transition of the system to a network state at lower
 278 activity. This occurs for global ($q = 1.0$) and partial stimulation ($q = 0.8, q = 0.6$ and $q = 0.5$). Please
 279 re-call that, for instance, $q = 0.5$ reflects a stimulation where 50% of the network nodes are stimulated.
 280 These stimulated network nodes have been randomly chosen from a uniform distribution.

281 **Figure 5** shows the respective power spectra of the network mean $\mathbf{V}(t)$, which provides insights about the
 282 system's synchronisation at low and high noise intensity. High noise intensity induces strong oscillations
 283 in the γ -frequency band, whereas the low noise intensity states does not - in contrast, this state shows a
 284 decaying low-pass power spectral density that is expected from a non-oscillatory stochastic process.

285 Stronger power spectral density at a given frequency is the signature of a coherent network, as seen in
 286 Fig. 5. Since the neurons in our network model emit spikes and exhibit synaptic input currents, noise-
 287 induced coherence may be visible in the coherence between spiking and synaptic activity as well. In fact,
 288 in Fig. 6(A) one observes a significant strongly enhanced spike-field coherence at high noise intensities for
 289 both global and partial stimulation. Hence, in sum the system exhibits coherence resonance in the sense
 290 that strong noise induces coherent oscillations that are not present at low noise intensities.

291 Coherence resonance is supposed to be linked to information processing in neural systems. Thus
 292 we investigate the relationship between stimulus noise intensity and information in the system across
 293 frequency bands. Figure 6(B) shows how much information is stored in the networks (AIS) and how
 294 much information is available (H). We observe that significantly more information is stored (AIS) and
 295 available (H) at high noise intensities for global stimulation $q = 1.0$, whereas high noise partial stimulation
 296 with $q = 0.8$ diminishes the stored active information and available information significantly. For more
 297 sparse stimulation with $q = 0.6$ the finding in information measures is heterogeneous and no interpretation
 298 consistent with the results for larger q is possible.

299 To understand this noise-induced coherence, we take a closer look at the dynamic topology of the mean-
 300 field equations (28). Their equilibria (cf. section 2.3) for negligible finite-size fluctuations $\rho^{e,i}(t) \ll 1$
 301 are shown in Fig. 7 together with simulated mean-field activity $\bar{V}(t)$ for illustrative purposes. Low noise
 302 intensity induces a bistable regime with a stable node as upper equilibrium and a focus as lower equilibrium.
 303 For global stimulation ($q = 1.0$), this lower focus is unstable at very low noise intensity and stable at
 304 larger noise intensities. Moreover, the lower equilibrium is a stable focus at all noise intensities for partial
 305 ($q < 1.0$) stimulation. The center branch is always a saddle node. For larger noise intensity, the upper
 306 equilibrium branch merges with the center branch via a saddle-node bifurcation and the lower stable focus
 307 is preserved as noise is further increased. This finding remains valid for both global ($q = 1.0$) and partial
 308 ($q < 1.0$) stimulation as shown in Fig. 7 for q ranging within the interval $0.5 \leq q \leq 1.0$. One can see
 309 that for smaller q (i.e. less excitatory neurons are stimulated) the bifurcation point moves to larger noise
 310 intensities. Hence thinning out the stimulation of excitatory neurons increases the noise intensity interval at
 311 which bistability occurs. **Moreover, we point out that the bifurcation points predicted by the mean-field**

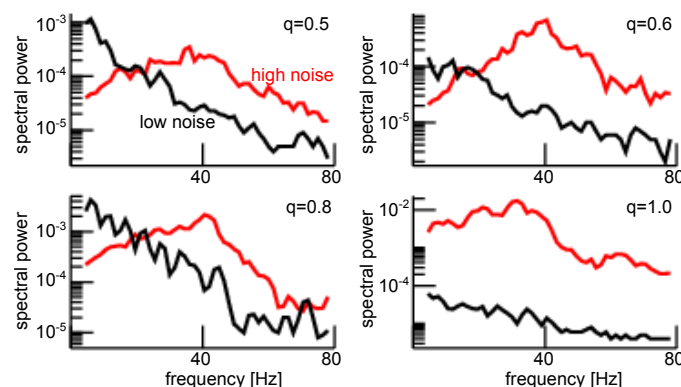


Figure 5. Enhanced noise yields strong power of the global mode $\bar{V}(t)$ in the γ -frequency range. The panels show the power spectra of \bar{V} for the stimulus ratios $q = 0.5$ ($D_1/\tau_e = 0.35$ (black) and $D_1/\tau_e = 0.55$ (red)), $q = 0.6$ ($D_1/\tau_e = 0.25$ (black) and $D_1/\tau_e = 0.33$ (red)), $q = 0.8$ ($D_1/\tau_e = 0.20$ (black) and $D_1/\tau_e = 0.25$ (red)) and $q = 1.0$ ($D_1/\tau_e = 0.15$ (black) and $D_1/\tau_e = 0.20$ (red)). Power spectra at lower noise intensities are computed on the respective upper branch of the bistable system.

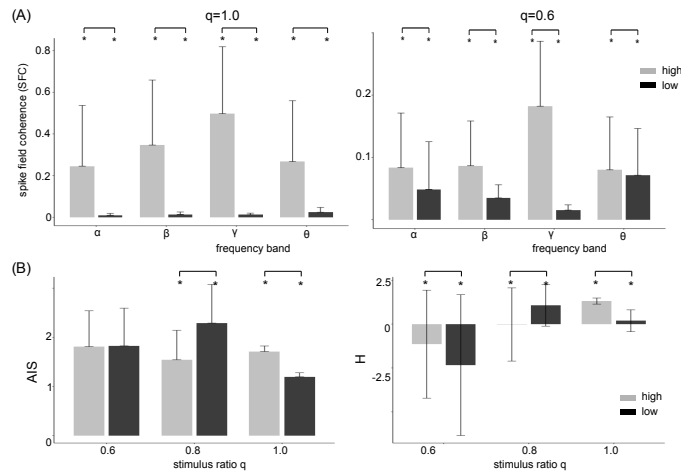


Figure 6. High zero-mean Gaussian noise enhances the Spike-Field Coherence in all frequency bands and affects heterogeneously Active Information Storage (AIS) and differential entropy (H). (A) The differences between high noise intensity (grey-colored) and low noise intensity (black-colored) is significant ($p < 0.001$) for both global and partial stimulation. (B) For global stimulation ($q = 1.0$), high noise intensity induces states of significantly enhanced stored active information (AIS) and available information (H), whereas partial stimulation with $q = 0.8$ diminishes both AIS and H significantly. Results for $q = 0.6$ are not consistent and difficult to interpret. In all panels, vertical bars denote the standard deviation, $p < 0.001$ and parameters are identical to the parameters used in Fig. 5.

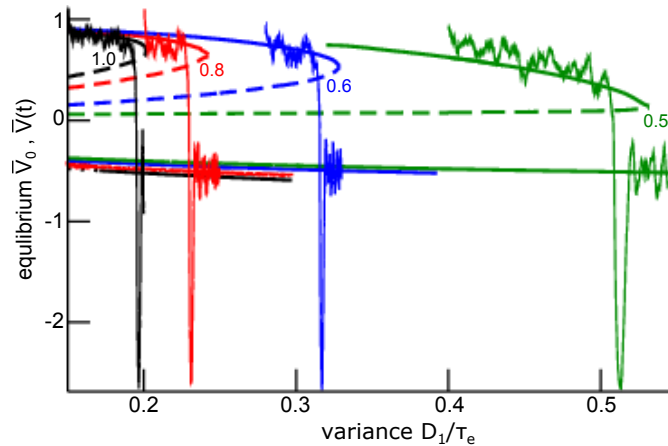


Figure 7. Equilibria and representative time series of the global mode $\bar{V}(t)$ for the zero-mean partial stimulation. There is a bistability and saddle-node bifurcation from a stable node to an stable focus at enhanced noise intensity. The numbers denote the values of the stimulus ratio q . Solid (dashed) lines mark stable (unstable) states. The time series \bar{V} results from the time-varying noise intensity according to Eq. (7.)

312 description and shown in Fig. 7 show very good accordance to the values of D_1/τ_e in Fig. 4, where the
 313 system transitions from the upper to the lower state.

314 The mean-field solution involves finite-size fluctuations that affect the solutions principal oscillation
 315 frequency and magnitude. By construction, these mean-field solutions converge to the network average for
 316 increasing network size N . Figure 8 compares the time series of mean-field solutions and network averages
 317 for increasing network sizes and affirms the convergence and thus the validity of the mean-field description.
 318 It is interesting to note that, besides the mean-field dynamics, the network's dynamical properties change
 319 with increasing N as well. Figure 8 provides the principal oscillation frequencies for both solutions for

320 the given network size: the network speeds up with increasing size and its frequency converges to the
 321 mean-field principal frequency that remains about the same value. However, we point out that the mean-
 322 field solution remains still slightly different even for very large N since it implies the approximation of
 323 negligible connectivity matrix bulk spectra.
 324 Figure 9 affirms this finding by comparing simulation trials of the transitions from the non-oscillatory to
 325 the oscillatory coherent state. We observe that the transition values of D_1/τ_e of the network mean and
 326 the mean-field are closer to each other for larger network size. The mean-field description (28) with (27)
 327 assumes vanishing finite-size fluctuations and these are reduced for larger network size N , i.e. the effective
 328 noise level (the finite-size fluctuations) is reduced and thus deterministic mean-field and stochastic network
 329 activity transition are closer to each other.

330 The frequency range of oscillations observed for steady states located within the lower branch (see
 331 Fig. 7) is a consequence of both network connectivity and neuronal properties - and is further tuned

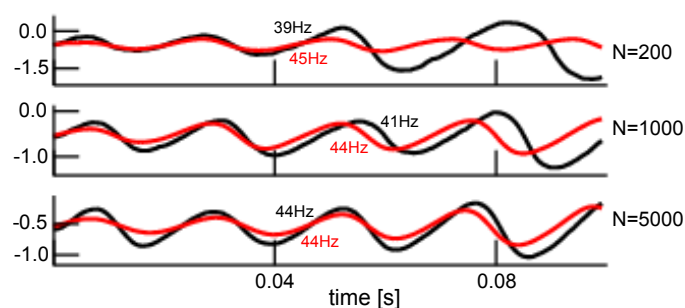


Figure 8. Comparison of network average and mean-field solution for different network sizes. The network average (black) and mean-field solutions (red) resembles more and more the larger the network of size N . This holds for the magnitude and frequency (provided in panels) of both solutions. The initial value of the mean-field activity has been chosen to the initial value of the network average. Simulations consider zero-mean Gaussian simulations with $q = 1$ and $D_1/\tau_e = 0.2$.

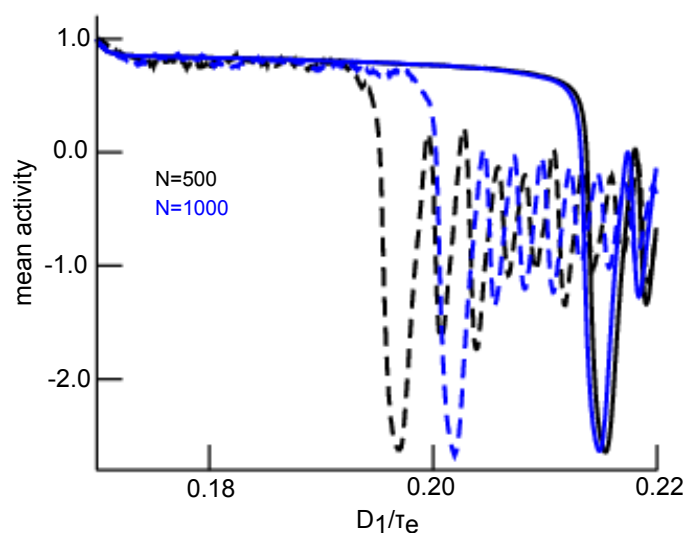


Figure 9. Comparison of transitions in network and mean-field for different network sizes. The network average (dashed line) and mean-field solutions (solid line) resemble more for larger network size N . This is explained by reduced finite-size fluctuations for larger networks. The initial value of the mean-field activity has been chosen to the initial value of the network average. Simulations consider zero-mean Gaussian simulations with $q = 1$.

332 by additive noise. Figure 10 shows the maximum eigenvalue real part for the upper (A) and the lower
 333 branch (B, top opanel) and the eigenfrequency (cf. subsection (2.3)) of the equilibrium at the lower branch
 334 (B, lower panel). We observe that increasing noise intensity decreases slightly the eigenfrequency in the
 335 γ -frequency range and decreases the negative maximum eigenvalue real part. This means that additive
 336 noise increases the damping of the response of the system to perturbations - including noise. This increased
 337 noise-induced damping leads to magnitude changes in quasi-cycle solutions - which is manifested in
 338 the power spectral density distribution. Indeed, the power spectral density distribution widens as noise
 339 intensity increases, leading to the spectra as seen in Fig. 10(C). This broad spectral power distribution is
 340 the signature of suppressed coherence. As a corollary, our analysis demonstrates that coherent band-limited
 341 oscillations emerge for intermediate noise intensities only. This is a known feature of coherence resonance.
 342 For additional illustration, Fig. 1 shows the typical bell-shape of coherence (here spike-field coherence) in
 343 different frequency bands. We observe that the coherence effect is strongest in the γ -frequency range.

344 3.3 Poisson partial stimulation

345 Synaptic receptors respond to afferent Poisson-distributed input spike trains, whose properties differ
 346 substantially from the Gaussian noise processes we considered so far. To generalize our results to more
 347 physiological stimuli statistics, we considered a partial Poisson noise stimulation with dependent mean and
 348 variance. Specifically, afferent spike trains at spike rate r_{in} induce random responses at excitatory synapses
 349 with time constant τ_{in} and synaptic weight w_{in} . Then

$$\begin{aligned} \mathbf{s}^e(t) &= \tau_e \Delta \boldsymbol{\xi} + \bar{\rho}^e(t) \\ \xi_1^e &= w_{\text{in}} r_{\text{in}} \tau_{\text{in}} \\ D_1 &= w_{\text{in}} \xi_0 / 2 \\ \bar{\xi}_0^e &= q \xi_1^e \end{aligned}$$

350 and finite-size fluctuations $\bar{\rho}^e(t) \sim \mathcal{N}(0, D_1/N_1)$. Figure 11(A) illustrates the temporal network activity
 351 for a low and high stimuli firing rates r_{in} . Increasing r_{in} induces a transition from a high-activity to a
 352 low activity state for both global and partial stimulation - similarly as in the Gaussian noise case. The
 353 high-activity state is non-oscillatory while the low-activity state is oscillatory, with frequency found in
 354 the γ -frequency range (Fig. 11(B)). In addition, the low-activity state induced by high Poisson input rate
 355 exhibits a strong spike-field coherence in contrast to the high-activity state (Fig. 11(C)). Moreover, high
 356 stimulation noise increases the stored information and the available information for global stimulation with
 357 $q = 1.0$, cf. Fig. 11(D). Information measures for partial stimulation ($q = 0.6$) are heterogeneous and an
 358 interpretation of results for AIS and H is difficult.

359 These results can be understood by taking a closer look at the dynamic topology of the system. Figure 12
 360 reveals that, for global stimulation ($q = 1.0$), the system has two unstable equilibria and one stable
 361 equilibrium at lower noise intensities. The top branch is a stable node, the center branch a saddle node
 362 and the lower branch an unstable focus. There is a very small noise intensity interval at which the top and
 363 bottom branch are both stable. Increasing the Poisson stimuli firing rate leads to a sudden suppression
 364 of high-activity equilibria through a saddle-node bifurcation. Consequently, the transition observed in
 365 Fig. 11(A) is a jump from the stable node on the top bifurcation branch to the stable focus on the bottom
 366 branch similar to the effect shown in Fig. 4. For partial stimulation ($q = 0.6$), the lower branch exhibits

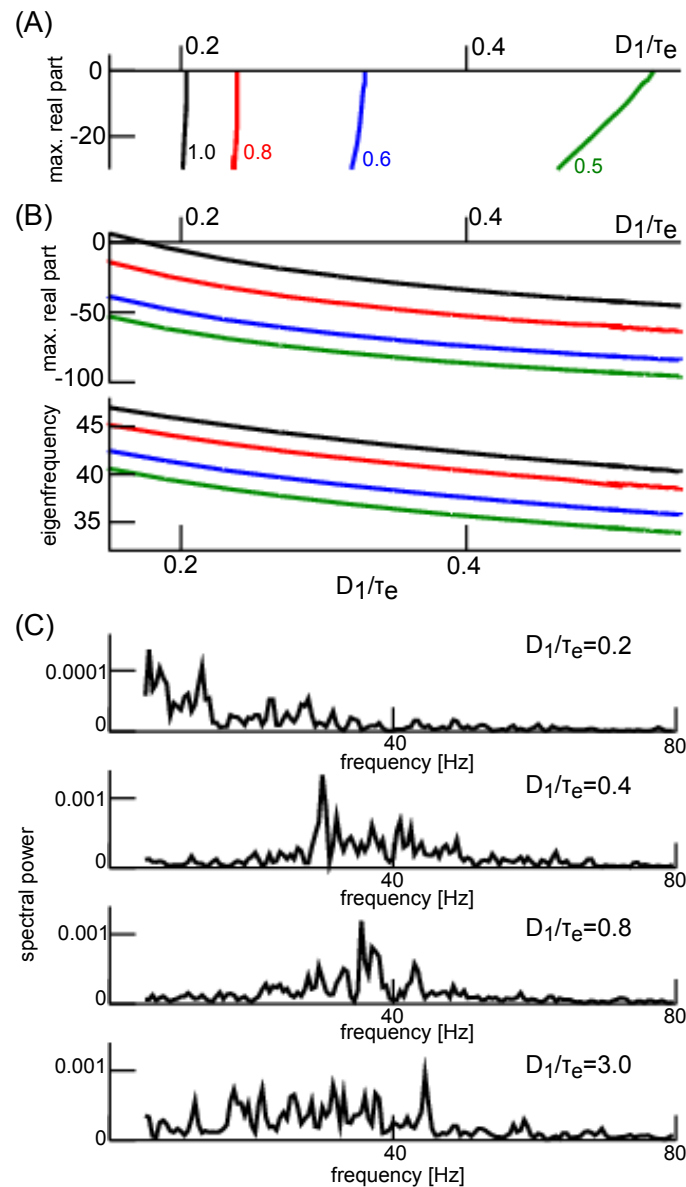


Figure 10. Eigenvalues at the top and bottom branch in Fig. 7 and corresponding power spectra. (A) maximum eigenvalue of equilibria on the top branch in Fig. 7. (B) maximum real part r of the eigenvalue $r + i2\pi\nu$ (top panel) and the corresponding eigenfrequency ν . The numbers denote the values of the stimulus ratio q in all panels. (C) Power spectra of $V(t)$ about the lower branch for $q = 0.6$ for different noise intensities D_1/τ_e .

367 a stable focus for much lower input firing rates. The saddle-node bifurcation is delayed, leading to an
 368 increased noise intensity interval of bistability. Hence, the system exhibits coherence resonance for Poisson
 369 noise as well.

4 DISCUSSION

370 **This study** presents a rigorous derivation of mean-field equations for two nonlinearly coupled non-sparse
 371 Erdős-Rényi networks(ERN) that are stimulated by additive noise. This mean field representation is made
 372 possible through spectral separation: the eigenspectrum of ERN networks exhibits a large spectral gap
 373 between the eigenvalue with largest real part and the rest of the spectrum. We show that the projection of
 374 the network dynamics onto the leading eigenmode represents the mean-field. Its dynamics are shaped by

375 eigenmodes located in the complement subspace spanned by non-leading eigenmodes. In our model, the
376 subspace dynamics are governed and influenced by additive noise statistics and they obey an Ornstein-
377 Uhlenbeck process.

378 We extended the mean-field derivation to various types of additive noise, such as global and partial noise
379 stimuli (i.e. when only a fraction of the excitatory neurons are stimulated) and for both zero-mean Gaussian
380 and **Poisson-like** noise. Collectively, our analysis shows that additive noise induces a phase transition from
381 a non-oscillatory state to an oscillatory coherent state. Such noise-induced coherence is known as coherence
382 resonance (CR). This phase transition has been shown to occur not only for Gaussian zero-mean noise but
383 also for **Poisson-like** noise. To the best of our knowledge, CR has not been found yet for such **Poisson-like**
384 noise. The general underlying mechanism is a noise-induced multiplicative impact of additive stimulation
385 via the nonlinear coupling of different modes. This multiplicative effect modifies the net transfer function
386 of the network and thus enlarges its dynamical repertoire. This resembles the impact of additive noise in
387 stochastic bifurcations [51, 52, 70, 71]

388 Embedding into literature

389 Our results build on previous studies from the authors [23, 54, 55] to provide a rigorous derivation of the
390 mean-field description, whereas previous work have motivated heuristically the mean-field reduction and,
391 e.g., failed to show in detail whether the mean-field equation is the only solution for any given additive
392 stimuli. Several other previous studies have presented mean-field descriptions in stochastically driven
393 systems. For instance, Bressloff et al. [28] have derived rigorously mean-field equations for stochastic
394 neural fields considering, inter alia, finite-element fluctuations by utilizing a Master equation and van
395 Kampen's volume expansion approach. We note here that we also took into account finite-size fluctuations
396 resulting from a non-negligible variance of statistical mean values. Moreover, [28] do not specify the
397 network type and results in a rather opaque description, whereas we assume an ERN and thus exploit its
398 unique eigenspectrum structure. This yields directly to a mean-field description, whose dependence of
399 stochastic forces is obvious and avoids its implicit closure problem known from mean-field theories [43].
400 This is possible since the ERN considered share many properties with Ising models, that are known to
401 permit an analytically treatable solution of the closure problem, see e.g. [72].

402 Moreover, several technical analysis steps in the present work have been applied in previous studies in
403 a similar context. In a work on stochastic neural mean-field theory, Faugeras and colleagues [27] have
404 assumed that the system activity fluctuations obey a normal probability distribution and have derived an
405 effective nonlinear interaction in their Proposition 2.1 similar to our Eq. (22). Further, the authors have
406 shown how the fluctuation correlation function, i.e. the system activity's second moment, determine the
407 mean-field dynamics. This is in line with our result (22) showing how the mean and variance of the additive
408 noise tunes the system's stability. However, the authors have not considered in detail the random nature
409 of the system connectivity, whereas we have worked out the interaction of external stimulation and the
410 ERN. This interaction yields directly the mean-field and its dependence of the external stimulus that is
411 not present in [27]. Moreover, the present work also shows how the mean-field fluctuations affect the
412 mean-field dynamics by deriving the fluctuation's probability density function that describes all higher
413 moments.

414 Noise-induced synchronization has been found recently in a system of stochastically-driven linearly
415 coupled FitzHugh-Nagumo neurons by Touboul and colleagues [73]. The authors have found a minimum

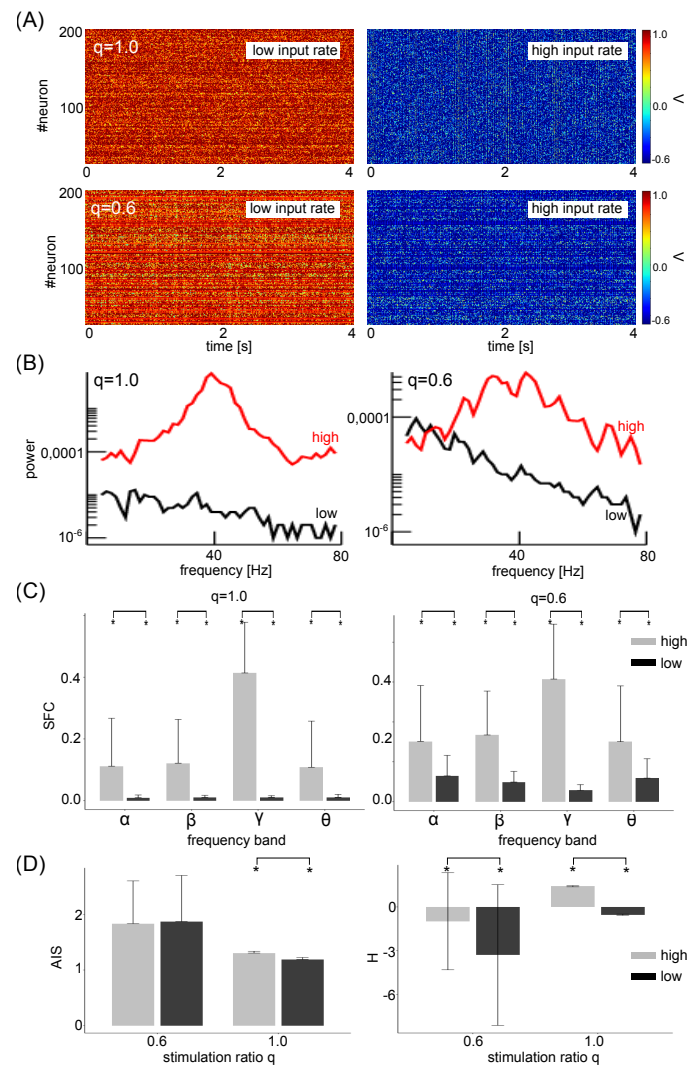


Figure 11. Poisson noise induces transitions from a non-oscillatory to an oscillatory state for both global and partial stimulation. (A) Network activity $V_n(t)$ for low input firing rate ($r = 0.04$ for $q = 1.0$ and $r = 0.09$ for $q = 0.6$) and high input firing rate ($r = 0.14$ for $q = 1.0$ and $r = 0.19$ for $q = 0.6$). For the low (high) input rate the system evolves about an upper (lower) state. (B) Power spectra of the network mean $\bar{V}(t)$ showing γ -activity for the large input rate. (C) The high input firing rate (grey-colored) induces a state of large spike-field coherence compared to the state for low input firing rate (black-colored) for both global and partial stimulation ($p < 0.01$). (D) For global stimulation ($q = 1.0$), high input firing induces a state of significantly enhanced stored active information (AIS) and available information (H). This is not consistent to results for partial stimulation ($q = 0.6$). Here is $p < 0.01$.

416 ratio of activated neurons that are necessary to induce global oscillatory synchronization, i.e. CR in the
 417 sense presented in our work. This question has been considered in the present work as well by asking how
 418 the mean-field dynamics, and thus how noise-induced synchronization, changes when modifying the ratio
 419 of stimulated excitatory neurons q while retaining the stimulation of inhibitory neurons. We find that global
 420 stimulation, i.e. stimulation of all excitatory neurons, yields a finite critical noise intensity below which the
 421 system is bistable and exhibits CR. Partial stimulation shifts this critical noise intensity to larger values and
 422 enlarges the bistability parameter space - and thus promotes CR.

423 Several previous studies of mean-field dynamics in neural systems have applied the master equation
 424 formalism [74, 75, 76]. This works nicely in completely irregular networks and the asynchronous activity

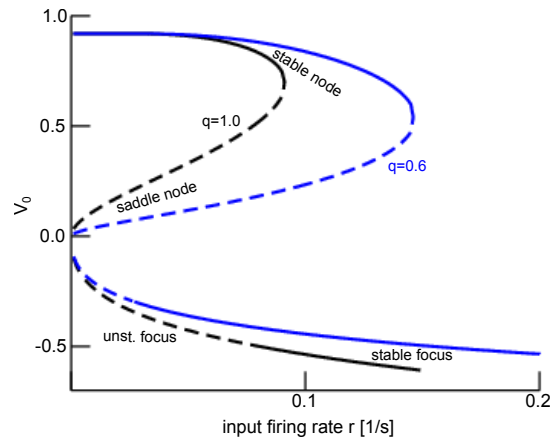


Figure 12. Equilibria of the mean-field $\bar{V}(t)$ for the Poisson partial stimulation. For global stimulation $q = 1.0$, the system is always monostable with three equilibria at low input firing rates and a single equilibrium at large input firing rates. Increasing the input firing rate from low to large firing rates, the system jumps from the upper stationary state (stable node) to a stable focus on the lower stationary state via a saddle-node bifurcation. For partial stimulation $q = 0.6$, the system is monostable with three equilibria at low input firing rates. For larger input rates, the system is bistable and passes a saddle-node bifurcation inducing a transition from a stable node to an stable focus at enhanced input firing rate r . Solid (dashed) lines mark stable (unstable) states, black- and blue-colored lines denote equilibria for global and partial stimulation, respectively. The bifurcation diagram of the mean-field $\bar{W}(t)$ is equivalent.

425 regime and has been applied successfully to neural populations considering biological neuron models [77,
 426 78, 79, 80]. However, the analysis of more regular networks will be very difficult to develop with the
 427 Master equation since the implicit integration over system states would be more complex. Conversely, our
 428 presented approach may consider regular structures by a corresponding matrix eigenvalue decomposition.

429 At last, we mention the relation to the Master stability function [81, 82]. This function describes the
 430 stability of identical synchronisation of complex networks in a synchronisation manifold and this manifold
 431 corresponds to the mean-field in our study. Although the Master stability function has been proven to be
 432 powerful, to the best of our knowledge it does not allow to reveal coherence resonance as the current work.

433 Limits and outlook

434 The present work proposes to describe mean-field dynamics in a topological network by projection
 435 onto the networks eigenmodes. This works well for non-sparse random ERN with large connectivity
 436 probability. This network does not exhibit a spatial structure. However, less connected ERN networks show
 437 different dynamics, cf. the Appendix. Moreover, biological networks are not purely random but may exhibit
 438 distance-dependent synaptic weights [83] or spatial clusters [84]. Our specific analysis applies for networks
 439 with a large spectral gap in their eigenspectra and it might fail for biological networks with smaller spectral
 440 gaps (as shown in the Appendix). Future work will attempt to utilize the presented approach to derive
 441 mean-field dynamics for heterogeneous networks that exhibit a smaller spectral gap, such as scale-free
 442 networks [84].

443 Moreover, the single neuron model in the present work assumes a simple static threshold firing dynamics
 444 (McCullough-Pitts neuron) while neglecting somatic dynamics as described by Hodgkin-Huxley type
 445 models or the widely-used FitzHugh-Nagumo model [11, 73]. Future work will aim at reinforcing the

446 biological relevance of neurons coupled through ERN. This will be possible by extending the trivial transfer
447 function from a step function to sigmoidal shapes for type I or type II neurons [85, 86, 76]

448 Our results show that noise-induced CR emerges in the γ -frequency range. This frequency band is
449 thought to play an important role in visual information processing [13, 14, 15, 16, 17]. Experimental
450 studies have shown that the degree of this γ -synchronization in primary cortical areas may be modulated
451 by attention [87, 88, 59, 89]. Since attention is known to affect the ARAS activity [90] and specifically the
452 brain stem as part of the ARAS [91] and ARAS, in turn, provides input to the cortex [92]. We conclude
453 that it is possible that attention modulates the cortical input activity, i.e. the Poisson firing rate in our model.
454 In this picture, attention-modulated enhanced ARAS activity induces γ -coherence and may enhance
455 stored information [93], as shown in Figs. 6 and 11. Future more detailed brain models including the
456 cortico-thalamic feedback and cortical interactions [57, 21] will provide further evidence whether coherence
457 resonance is present in visual processing.

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