

A DISTRIBUTION OF SPIKE TRANSMISSION DELAYS AFFECTS THE STABILITY OF INTERACTING SPIKING NEURONS

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ABSTRACT.

We summarize the approach leading to the dynamical mean-field equation for the spike emission rate $\nu(t)$ of an interacting population of Integrate-and-Fire (IF) neurons derived in [9]. Building on the results concerning the stability conditions and finite-size effects, we investigate how such properties are affected by a non-trivial distribution of spike transmission delays. The main findings are: i) the stability of the collective asynchronous states is improved by widening the distribution of delays; ii) high-frequency components of the power spectrum of the collective activity are damped; iii) Details of the stability and spectral properties are strongly affected by the shape of the delay distribution. We present quantitative predictions from the theory and we demonstrate a very good agreement with simulations.

Introduction In [9] we introduced a method to derive (*via* a population density approach, [6, 10]) the equations which govern the time evolution of the population activity of an interacting ensemble of IF neurons in the *extended* mean field approximation (*i.e.*, taking into account the instantaneous fluctuation of the afferent current). The general approach is also amenable to an approximate treatment, by which we could characterize the stability properties and the transient response of the activity $\nu(t)$ of the neural population in a specific context, elucidating a close and interesting relationship between the time course of $\nu(t)$ and the “transfer function” characterizing the static mean field properties of the system. A proper treatment of finite-size effects allowed us to study the power spectrum of $\nu(t)$. The synaptic transmission delays turn out to play a major role in the above analysis. In the present work we remove a somewhat unnatural constraint assumed in [9], that the synaptic delays are the same for all neurons, and we show how a *distribution* of synaptic delays can be easily embedded in the analysis. Extending preliminary results presented in [8] we discuss how the delay distribution affects the stability conditions and the spectral properties of the system.

Dynamic equations for the population activity In the diffusion approximation, the sub-threshold dynamics of the membrane depolarization V of a general class of IF neurons is given by [13] $\dot{V} = f(V) + \mu(V, t) + \sigma(V, t)\Gamma(t)$, where the afferent current is described as a Gaussian white noise with mean $\mu(V, t)$ and variance $\sigma^2(V, t)$ and $f(V)$ is a leakage term.

In the *extended* mean field approach [3, 2] all neurons in a homogeneous population are driven by stochastic currents with the same mean and variance, both depending on the recurrent and external (ν and ν_{ext} respectively) emission rates, equal for all neurons: $\mu = \mu(V, \nu, \nu_{ext})$ and $\sigma^2 = \sigma^2(V, \nu, \nu_{ext})$. In this approximation, the set of evolving V s is

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seen as a sample of independent realizations drawn from the p.d.f $p(v, t)$, governed by the Fokker-Planck equation:

$$(1) \quad \partial_t p(v, t) = L p(v, t) = \left[-\partial_v [f(v) + \mu(v, t)] + \frac{1}{2} \partial_v^2 \sigma^2(v, t) \right] p(v, t),$$

complemented by boundary conditions accounting for the realizations disappearing at the spike emission threshold θ and re-appearing at the reset potential H [12, 1, 6, 4, 5]. Since L depends on μ and σ^2 , it is an implicit function of the emission rate ν . The latter in turn expresses the flux of realizations crossing θ , or the fraction of neurons emitting spikes per unit time:

$$(2) \quad \nu(t) = -\frac{1}{2} \sigma^2(v, t) \partial_v p(v, t)|_{v=\theta}.$$

It is convenient to expand $p(v, t)$ in Eqs. (1) and (2) onto the complete set of eigenfunctions ϕ_n of L : $p(v, t) = \sum_n a_n(t) \phi_n(v, t)$. In stationary conditions ν is the inverse of the mean inter-spike interval, and it also equals the single neuron transfer function $\Phi(\mu, \sigma^2)$, given by (2) with $p(v, t) = \phi_0(v)$, the eigenfunction of L with zero eigenvalue, stationary solution of Eq. (1). The time evolution of $p(v, t)$ is then described in terms of dynamical equations for the $a_n(t)$ (see e.g. [11, 6]); taking into account Eq. (2), allows us to write the equations governing the time evolution of $\nu(t)$ (the “emission rate equation”) as [9]:

$$(3) \quad \begin{cases} \dot{\bar{a}}(t) &= [\Lambda(t - \delta) + \mathbf{C}(t - \delta) \nu(t - \delta)] \bar{a}(t) + \bar{c}(t - \delta) \nu(t - \delta) \\ \nu(t) &= \Phi(t - \delta) + \bar{f}(t - \delta) \cdot \bar{a}(t) \end{cases},$$

where f_n is the contribution to the flux through θ due to the mode ϕ_n ($n \neq 0$); $c_n = \langle \partial_v \psi_n | \phi_0 \rangle$, $C_{nm} = \langle \partial_v \psi_n | \phi_m \rangle$; ψ_n are the eigenfunctions of the adjoint operator L^+ and $\langle \cdot | \cdot \rangle$ is a suitable inner product. C_{nm} and c_n are coupling terms, in that for uncoupled neurons μ and σ do not depend on the recurrent frequency ν , and $\partial_v \psi_n$ vanishes. Λ is the diagonal matrix of the common eigenvalues of L and L^+ . In Eq. (3) a single allowed synaptic delay δ appears. However, taking into account a *distribution* of delays is relevant both in order to relax a somewhat implausible assumption and because it might provide an effective treatment of non-instantaneous synaptic currents provoked by each spike (see [4]).

Going from a single δ to a distribution $\rho(\delta)$ amounts to substitute the convolution $\int \nu(t - \delta) \rho(\delta) d\delta$ for $\nu(t - \delta)$. We will show in the following the implications of a non-trivial $\rho(\delta)$.

Stability and response times The system (3) has fixed points ν_0 given by the self-consistency equation [3, 2] $\bar{a} = 0$ and $\nu_0 = \Phi(\nu_0)$; we assess their stability using a local analysis Eq. (3). To this end we study the poles of the Laplace transform $\nu(s)$ of the linearized form of $\nu(t)$ in Eq. (3). The real and imaginary parts of these poles describe the characteristic times and the oscillatory properties of the collective activity $\nu(t)$, respectively.

Such poles can be grouped in two classes. The first is related to the transmission delays (“transmission poles”), appearing only in coupled networks, and for a single fixed delay δ they are approximately given by:

$$(4) \quad s_n^{(t)} \simeq \frac{1}{\delta} \ln |\Phi'| + i \frac{n\pi}{\delta},$$

where n is any odd (even) integer for inhibitory (excitatory) populations and $\Phi' = \partial_\nu \Phi|_{\nu=\nu_0}$. The fixed point becomes unstable when $\text{Re}(s_n^{(t)}) > 0$, which happens exactly when

$$(5) \quad \Phi'(\nu_0) > 1,$$

for an excitatory population. A sufficient condition for a weakly coupled inhibitory population to be stable is $\Phi'(\nu_0) > -1$. The “diffusion poles” $\{s_n^{(d)}\}$ have negative real parts and do not contribute to the stability conditions. Far from instability the longest $-1/\text{Re}(s_n^{(d)})$ sets the time scale of the transient of the network relaxation to the fixed point. For weak coupling and for a single fixed delay δ , in a regime of low noise in the afferent current (*drift-dominated* – supra-threshold regime), $\{s_n^{(d)}\}$ are a perturbation of the eigenvalues λ_n of L , and the longest time scale is related to:

$$(6) \quad s_1^{(d)} \simeq \lambda_1 \left(1 + \frac{f_1 c_1}{1 - \Phi' + \lambda_1 \delta} \right).$$

It is worth noting how, despite the fact that there is no obvious *a priori* relation between the single neuron properties and characteristic times, and the dynamics of the collective activity, the single neuron transfer function Φ emerges in a leading role in determining both the stability of the system, and the response times.

In the case of a non-trivial delay distribution $\rho(\delta)$ it turns out that only the transmission poles are significantly affected by the width Δ and the shape of $\rho(\delta)$ as we show in the following.

Figure 1, left, shows for different values of Δ the distribution of diffusion and transmission poles (only the first three pairs) in the complex plane of the Laplace variable, for an inhibitory population of linear (constant leakage) IF neurons (‘LIF’) [5], in a drift-dominated regime.

It is clearly seen in the figure that the diffusion poles (diamonds) are virtually unaffected by the changes in Δ . The parameters are chosen in such a way that in the $\Delta = 0$ ms case the asynchronous, constant ν state of the network is unstable, and the neural population is easily driven to a global oscillatory state. In this case the real part of the first transmission pole is positive.

It is seen that, starting from the unstable state (the darkest, rightmost circles for $\Delta = 0$), increasing Δ makes the network more stable (the real part of the transmission poles becomes more and more negative).

Another noteworthy feature emerges from a comparison of the real parts of the transmission and diffusion poles. A careful inspection of the figure reveals that close to the stability boundary (exemplified by the case $\Delta = 1$ ms) the “memory” of the system is dominated by the real part of the first transmission poles, while when the latter are far from the imaginary axis (the cases $\Delta \geq 2$ ms) the dominating time scale is determined by the real part of the first diffusion poles.

The non-monotonic dependence on Δ of the real parts of the transmission poles is immaterial in this case, since it affects virtually unobservable time scales. We will see in the following that this feature strongly depends on the shape of $\rho(\delta)$. A non-trivial dependence on Δ of the imaginary part of the transmission poles is also observed in the figure.

The scenario just described, predicted by the linearized emission rate equation, is checked through simulations in the right panel of Fig. 1. The simulated neural populations are composed by $N = 5000$ inhibitory LIF neurons. The stimulation protocol is the same for all the reported plots: The initially quiescent population receives an external stepwise excitatory (noisy) current at $t = 50$ ms which remains constant throughout the simulation. The parameters are such that the asymptotic emission rate of the asynchronous state is $\nu = 20$ Hz. The plots show the average over 10 runs and shaded strips represent standard deviations. Each plot corresponds to a value of Δ marked in the left panel.

In the topmost unstable case ($\Delta = 0$ ms) after a transient the system persists in a global oscillatory state. The frequency of the oscillation is found to be very close to $\text{Im} s_1^{(t)}/2\pi$ as

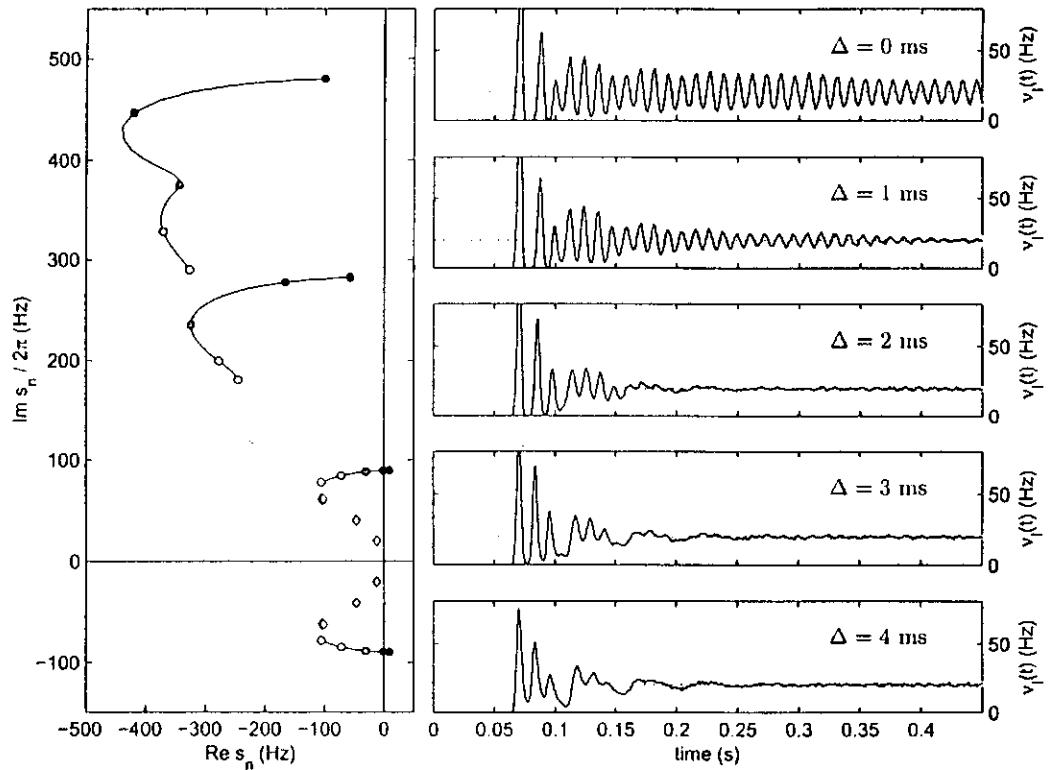


Figure 1: Poles distribution and sample transient responses for a recurrent inhibitory population of LIF neurons in a drift-dominated regime with different widths Δ of the uniform distribution of delays, and the same average delay $\bar{\delta} = 5$ ms. Left: distribution of poles in the complex plane of the Laplace variable. Diamonds: first 3 conjugate pairs of diffusion poles; Circles: first 3 conjugate pairs of transmission poles. The darker the marker, the smaller $\Delta \in \{0, 1, 2, 3, 4\}$ ms. Right: Sample transient responses for the Δ values corresponding to the markers in the left panel (top to down \leftrightarrow dark to light). Dotted lines indicate for reference the first-order stationary state of the collective activity ($\nu_0 = 20$ Hz).

predicted by the theory for this Hopf bifurcation.

As Δ increases the system asymptotically approaches an asynchronous state of constant ν , losing memory of its transient response in an increasingly short time: For short Δ this is brought about by the decrease of $-1/\text{Re } s_1^{(t)}$, while for longer Δ the diffusion poles take over and their real part, independent on Δ , dominates the characteristic time of the transient response.

The latency of $\nu(t)$ is determined by the peculiar initial conditions and equals the time needed for the depolarization of each neuron, initially at rest, to reach the firing threshold when driven only by external current (neurons of course begin to interact after firing).

Spectral properties The (stationary and transient) spectral content of $\nu(t)$ is embodied in the power spectrum $P(\omega)$, a proper treatment of which requires taking into account the effects of the finite number N of neurons in the population under consideration. Besides

incoherent fluctuations, *e.g.* due to quenched randomness in the neurons' connectivity and/or to external input (which are taken into account in the variance σ^2 of the afferent current entering the Fokker-Planck equation) two additional finite-size effects contribute: 1) For finite N , the number of spikes emitted per unit time is well described by a Poisson process with mean and variance $N\nu(t)$, such that the fraction ν_N of firing neurons per unit time is approximated by $\nu_N(t) = \nu(t) + \sqrt{\nu(t)/N}\Gamma(t)$, with Γ a memoryless, white noise [4]. In the mean field treatment, the fluctuating ν_N enters the infinitesimal mean and variance of the afferent current, so that μ and σ^2 are now stochastic variables ($\mu \rightarrow \mu_N$ and $\sigma^2 \rightarrow \sigma_N^2$), thereby making the Fokker-Planck operator itself N -dependent and stochastic (L_N). 2) While the above transition $L \rightarrow L_N$ still describes the time evolution of an *infinite* number of realizations, though driven by N -dependent fluctuating currents, the finite size of the neurons sample has to be explicitly taken into account in the boundary condition expressing the conservation of the total number of realizations crossing the threshold and re-appearing at the reset potential. The latter effect was not considered in previous treatments of finite-size effects. The combined finite- N effects result in the following modified FP equation [9]

$$(7) \quad \partial_t p(v, t) = L_N p(v, t) + \delta(v - H) \sqrt{\frac{\nu(t)}{N}} \Gamma(t)$$

In the framework of the local analysis, and for a non-trivial delay distribution, the resulting expression for the power spectral density of the population activity is:

$$(8) \quad P(\omega) = \frac{\left| 1 + \vec{f} \cdot (i\omega \mathbf{I} - \mathbf{\Lambda})^{-1} \vec{\psi} \right|^2}{\left| 1 - \Phi' \rho(i\omega) - i \vec{f} \cdot (i\omega \mathbf{I} - \mathbf{\Lambda})^{-1} \vec{c} \omega \rho(i\omega) \right|^2} \frac{\nu_0}{N}.$$

$\rho(i\omega)$ is the Fourier transform of the delays distribution centered around the mean $\bar{\delta}$; the elements of $\vec{\psi}$ are the eigenfunctions of L^+ , evaluated at H , and the ψ -dependent term accounts for the finite- N effects on the boundary. It is found that: 1) the asymptotic $P(\omega)$ oscillates around the white-noise flat spectrum ν_0/N ; 2) $P(\omega)$ has resonant peaks centered in the imaginary parts of the transmission poles $s_n^{(t)}$, which are determined by the transmission delays and are modulated by the width and the shape of $\rho(\delta)$; they disappear for uncoupled neurons, since in that case $\Phi' = 0 = \vec{c}$; 3) the numerator of Eq. (8) modulates the spectrum and introduces peaks which, at least for not too large couplings, are located at the imaginary part of the eigenvalues of L (which are multiples of ν_0). It turns out that, as conjectured in [9], the latter, low- ω component of the power spectrum appears only in drift-dominated regimes and it is present even for uncoupled neurons.

Figure 2 illustrates how the power spectrum of the collective activity is affected by the width Δ of a uniform $\rho(\delta)$ for an inhibitory population of LIF neurons: both theoretical predictions and simulation results are reported. From top to bottom ($\Delta \in 0, 1, 2, 3$ ms) we move from an unstable situation to a stable one.

"due to higher transmission poles" into "due to higher order transmission poles"

Starting from the latter the following feature are visible: i) the first peak at 20 Hz and the secondary, barely recognizable, 40 Hz peak are the ones corresponding to the first two eigenvalues of L (whose imaginary parts equal ν_0 and $2\nu_0$); ii) the asymptotic value of $P(\omega)$ is ν_0/N as predicted by the theory; iii) a prominent "transmission" peak very close to the predicted value $\omega/2\pi \simeq 1/2\bar{\delta}$ is clearly visible in the plot of the bottom, where one can also recognize smaller peaks due to higher order transmission poles. As we move towards the stability boundary (bottom to top), the spectral peaks associated with the transmission poles keep their position and get sharpened, consistently with the predicted dependence on Δ of the imaginary and real part of the poles (see Fig. 1).

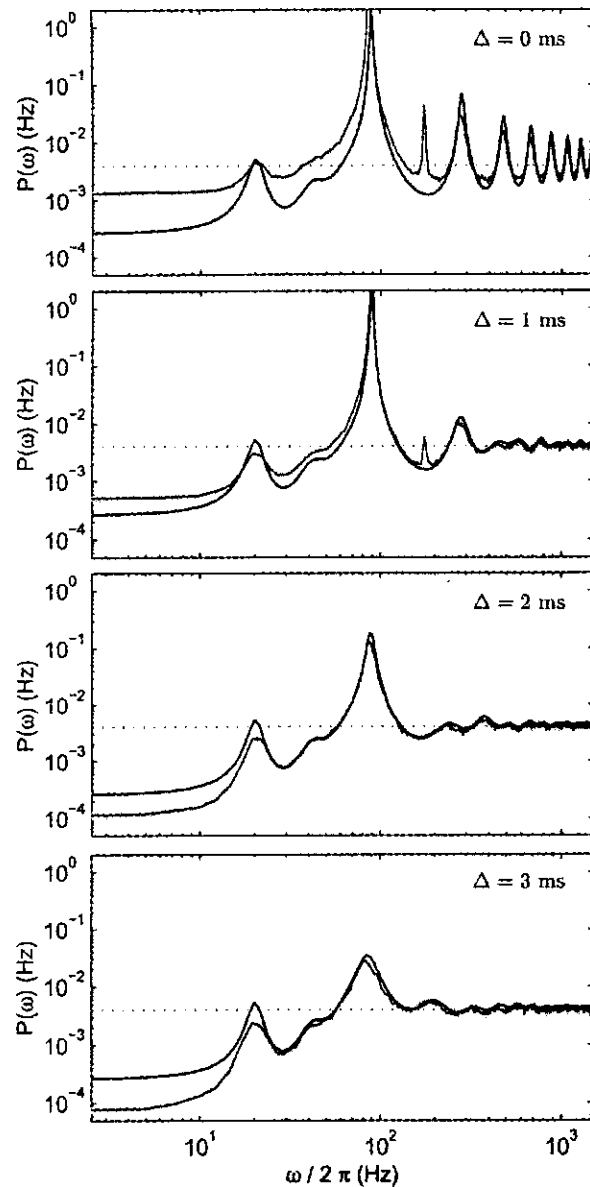


Figure 2: Power spectrum of the collective activity of an inhibitory population of LIF neurons for a uniform $\rho(\delta)$ and half-width $\Delta \in 0, 1, 2, 3$ ms, theory (thick lines) *versus* simulation (thin lines; averaged over 10 samples, shaded strips are the error estimates). Dotted lines: power spectrum of a white noise with variance $\nu_0/N = 20\text{Hz}/5000 = 0.004\text{Hz}$.

Upon crossing the stability boundary ($\Delta = 1, 0$ ms), a further unpredicted peak appears, which is signalling the Hopf bifurcation describing the transition from the stable asynchronous state to the global oscillatory state, and bringing the system out of the range of validity of the linearized theory.

Apart from a discrepancy in the low- ω region (see [9] for a brief discussion of its possible origin), an excellent agreement between the linearized theory (when applicable) and the simulations is apparent from the figure.

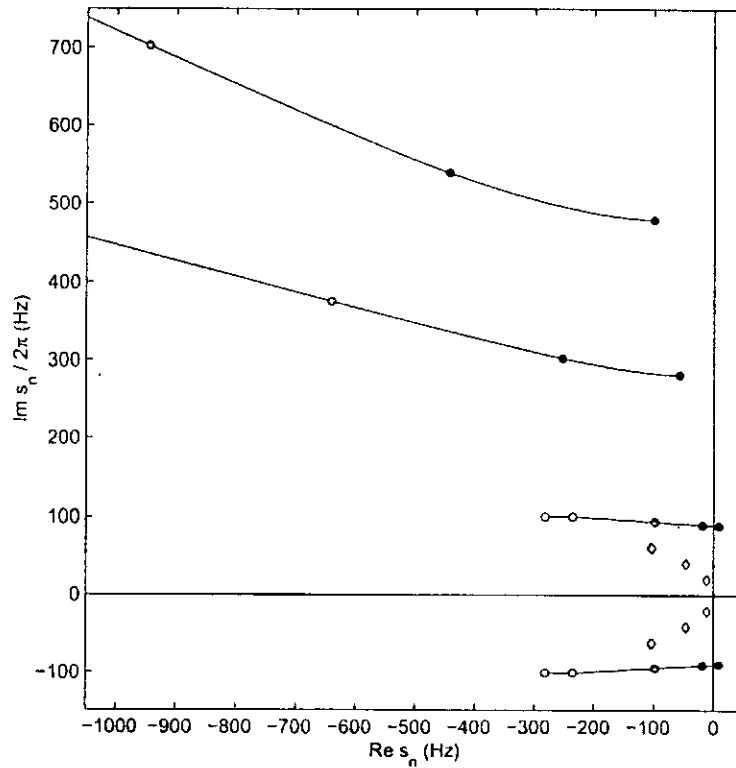


Figure 3: Poles distribution for a recurrent inhibitory population of LIF neurons in a drift-dominated regime with different widths Δ of an exponential distribution of delays. The distribution of poles in the complex plane of the Laplace variable is illustrated as in Fig. 1.

A more realistic $\rho(\delta)$ The choice of a uniform distribution of delays is arbitrary and one might wonder to what extent the results illustrated above depend on the shape of $\rho(\delta)$. We therefore repeated the analysis for a different delay distribution, more plausible from the biological point of view. In fact it is not unreasonable to assume that i) very few spikes come to a neuron under consideration with too short a delay (there is a lower bound on the distance between neurons), ii) beyond a threshold minimal distance the number of synaptic contacts should be a decreasing function of the distance between target neurons and the ones firing on it, so a $\rho(\delta)$ is assumed to be a decreasing function of δ (see [7] for a discussion of the intra-cortical connectivity).

Figure 3 illustrates the pattern of singularities of the Laplace transform of ν for an exponential distribution of delays

$$\rho(\delta) = e^{-\frac{\delta - \bar{\delta} + \Delta}{\Delta}} \Theta(\delta - \bar{\delta} + \Delta),$$

where $\bar{\delta}$ and Δ are respectively the mean and the standard deviation of $\rho(\delta)$, and $\Theta(t)$ is the Heaviside function.

As for the stability properties, the emerging scenario is not qualitatively changed with respect to the uniform $\rho(\delta)$. However, the real part of the transmission poles is now a monotonically decreasing function of Δ , and it is more sensitive to changes in Δ , thus emphasizing the impact of Δ on the stability properties of the system. Other differences

emerging in comparing Fig. 1 and Fig. 3 are of minor phenomenological relevance.

The spectral analysis for the exponential $\rho(\delta)$ (not shown) retains the qualitative features emerging in the case of a uniform distribution of delays. Consistently with the greater sensitivity to Δ mentioned above the high frequency damping is more effective in this case.

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