Mechanisms that modulate the transfer of spiking correlations

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Abstract

Correlations between neuronal spike trains impact network dynamics and population coding. Overlapping afferent populations and correlations between presynaptic spike trains introduce correlations between the inputs to downstream cells. To understand network activity and population coding, it is therefore important to understand how these input correlations are transferred to output correlations. Recent studies have addressed this question in the limit of many inputs with infinitesimal postsynaptic response amplitudes, where the total input can be approximated by Gaussian noise. In contrast, we address the problem of correlation transfer by representing input spike trains as point processes with each input spike eliciting a finite postsynaptic response. This approach allows us to naturally model synaptic noise and recurrent coupling, and to treat excitatory and inhibitory inputs separately. We derive several new results that provide intuitive insights into the fundamental mechanisms that modulate the transfer of spiking correlations.

1 Introduction

The amount of information carried by neuronal populations can be strongly modulated by correlations in neuronal activity (Zohary et al., 1994; Sompolinsky et al., 2001; Averbeck et al., 2006), and the structure of correlations can encode information about a stimulus (Vaadia et al., 1995; Dan et al., 1998; Maynard et al., 1999; Shlens et al., 2006; Biederlack et al., 2006; Temereanca et al., 2008). An understanding of how correlated variability is propagated is therefore central to understanding coding in neural tissue.

Synaptic divergence introduces correlated variability between the activity of nearby cells (Shadlen and Newsome, 1998) and synaptic convergence downstream can dramatically amplify correlations (Renart et al., 2010; Rosenbaum et al., 2010). In the absence of mechanisms to modulate these correlations, highly correlated activity can develop in deeper layers (Reyes, 2003). However, correlations measured *in vivo* tend to be small (Ecker et al., 2010; Renart et al., 2010). Some recent studies show that correlations can be modulated by network effects (Hertz, 2010; Renart et al., 2010). Here, we

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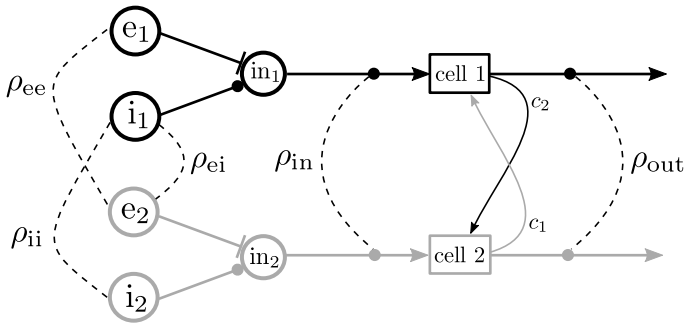

Figure 1: **An abstract representation of the input-output model.** Two cells each receive correlated excitatory, $\mathrm{e}_j(t)$, and inhibitory, $\mathrm{i}_j(t)$, inputs. These are combined to obtain the total input currents, $\mathrm{in}_j(t)$, which drive two integrate-and-fire neurons, IF_j, to produce output spike trains, $\mathrm{out}_j(t)$. In some of our analysis, we consider coupling between the cells. We seek to understand how the correlation, ρ_{out}, between the output spike trains is related to the statistics of the inputs and the dynamical properties of the neurons.

examine how stochastic dynamics at the cellular level modulate correlations between the outputs of two cells (See Fig. 1).

Earlier analytical approaches to this problem relied on the assumption that inputs can be modeled by correlated Gaussian noise (Moreno-Bote and Parga, 2010, 2006; de la Rocha et al., 2007; Ostojić et al., 2009; Vilela and Lindner, 2009). Such models are obtained in the limit of a large number of inputs, each of vanishing strength (Renart et al., 2004) and may not fully capture the statistical properties of the neurons' responses (Helias et al., 2010).

In the models we consider, input are represented by point processes with each input spike having a finite impact on the membrane potential of a cell. This approach allows us to examine the effects of synaptic failure, random synaptic amplitudes, and recurrent coupling between cells, while maintaining a more direct connection to physiology. Moreover, excitatory and inhibitory inputs can be treated separately (See Fig. 1). We find that the effects of synaptic noise and excitatory–to–inhibitory correlations, which are often ignored when inputs are modeled as Gaussian noise, can greatly reduce output correlations. Such reductions in correlation from input to output may partly explain small correlations sometimes observed *in vivo* (Ecker et al., 2010; Renart et al., 2010).

To obtain a clearer understanding of how the internal dynamics of spiking neurons affect correlations, we study correlation transfer for random walk neuronal models. These models are drastic simplifications of detailed neuron models. However, they have the advantage of being mathematically tractable, while capturing essential features of the response of spiking cells (Fusi and Mattia, 1999; Salinas and Sejnowski, 2000; Rauch et al., 2003). Due to the simplicity of these models, our results can be understood using intuitive and mechanistic explanations. We verify that physiologically more realistic models behave similarly by comparing our analytical results with simulations.

2 Methods

2.1 Spike Trains and Correlations

Spike trains are represented as stationary point processes, $a(t) = \sum_i \delta(t - t_i)$ where $\delta(t)$ denotes the Dirac function and $\{t_i\}_{i=1}^{\infty}$ is the set of spike times (Cox and Isham, 1980; Daley and Vere-Jones, 2003). The process $N_a(t) = \int_0^t a(s)ds$ counts the number of spikes in the interval $[0, t]$ and $N_a(t_1, t_2) = \int_{t_1}^{t_2} a(s)ds$ counts the number of spikes in $[t_1, t_2]$. Stationarity implies constant firing rates $r_a = E[a(t)] = E[N_a(t)]/t$, where $E[\cdot]$ denotes expected value. The second order statistics are quantified using the covariance and variance of spike counts

$$\gamma_{ab}(t) = \mathrm{cov}(N_a(t), N_b(t)) \quad \text{and} \quad \sigma_a^2(t) = \gamma_{aa}(t)$$

which are related to the cross-covariance functions by

$$\gamma_{ab}(t) = \int_{-t}^{t} R_{ab}(s)(t - |s|)ds. \tag{1}$$

where $R_{ab}(\tau) = \mathrm{cov}(a(t), b(t + \tau)) = r_b(H_{ab}(\tau) - r_a)$ is the cross-covariance function, and

$$H_{ab}(\tau) = \lim_{\delta \to 0} \Pr(N_b(\tau, \tau + \delta) > 0 \big| \, N_a(0, \delta) > 0)/\delta$$

is the conditional firing rate (Cox and Lewis, 1972; Brody, 1999). The correlation coefficient is then given by $\rho_{ab}(t) = \gamma_{ab}(t)/\left(\sigma_a(t)\sigma_b(t)\right)$. We mostly focus on the *asymptotic* statistics

$$\gamma_{ab} = \lim_{t \to \infty} \gamma_{ab}(t)/t, \quad \sigma_a^2 = \gamma_{aa}, \quad \text{and} \quad \rho_{ab} = \gamma_{ab}/\left(\sigma_a\sigma_b\right),$$

which can alternately be defined using the relation $\gamma_{ab} = \int_{-\infty}^{\infty} R_{ab}(s)ds$.

The Fano factor, $F_a = \sigma_a^2/r_a$, is a measure of the variability, or randomness, in a spike train. For renewal spike trains, $F_a = \mathrm{CV}_a^2$ where CV_a is the coefficient of variation (CV) of the interspike interval distribution (Cox, 1962; Nawrot et al., 2008).

A measure of *exact synchrony* between spike trains a and b is

$$S_{ab} = r_{a \cdot b}/\sqrt{r_a r_b} = \lim_{t \to 0} \rho_{ab}(t)$$

where $r_{a \cdot b} = \lim_{\delta \to 0} \int_{-\delta}^{\delta} R_{ab}(s)ds$ is the rate of spikes occurring at precisely the same time. Precisely synchronous spikes can occur in integrate-and-fire models with finite, instantaneous synaptic responses whenever two neurons receive exactly synchronous excitatory input spikes. Although exactly synchronous spikes are a mathematical idealization, S_{ab} models the proportion of output spike pairs caused by a shared excitatory input in physiological settings.

2.2 The leaky integrate-and-fire (LIF) model

Throughout the text we compare analytical results to simulations of a current based leaky integrate-and-fire model, hereafter referred to as the LIF. The membrane potential of the LIF is described by (Tuckwell, 1988; Burkitt, 2006)

$$dV = -\frac{1}{\tau_m}(V - V_{\rm rest})dt + \overline{d_{\rm e}}{\rm e}(t)dt - \overline{d_{\rm i}}{\rm i}(t)dt. \tag{2}$$

where ${\rm e}(t)$ and ${\rm i}(t)$ are the excitatory and inhibitory input spike trains respectively and $\tau_m > 0$ is the membrane time constant (Burkitt, 2006). When $V(t)$ reaches threshold, θ, an output spike is produced, and $V(t)$ is set to $V_{\rm reset}$. To simulate an inhibitory reversal potential, a lower barrier on the membrane potential is imposed at $\beta \leq V_{\rm reset}$. For simplicity we assume that $\overline{d_{\rm e}} = \overline{d_{\rm i}}$. This assumption is relaxed in a later section and in the Appendix by allowing variable synaptic responses. Voltage is measured in units of the postsynaptic amplitude so that $\overline{d_{\rm e}} = \overline{d_{\rm i}} = 1$. Due to our choice of units, the leak current when the membrane potential is at $V = v$ is given by v/τ_m. The maximum leak current is therefore θ/τ_m. We set $V_{\rm reset} = V_{\rm rest} = 0$ and in simulations we use $\theta = 30$, and $\beta = -2$.

The output spike train, ${\rm out}(t)$, is a point process consisting of times at which the membrane potential, $V(t)$, reaches threshold. In all examples considered, $V(t)$ is an ergodic process (Stratonovich, 1963), and in all analysis, $V(0)$ is assumed to be drawn from the stationary distribution of $V(t)$ so that the output spike train is stationary.

2.3 The statistics of the input spike trains

We consider two cells, $j = 1, 2$, each of which receives excitatory and inhibitory input spike trains, ${\rm e}_j(t)$ and ${\rm i}_j(t)$. Although more general results are derived in the Appendix, several assumptions are made in the text to keep the presentation less burdensome. In particular, we assume that the inputs to the two cells are statistically identical: $r_{\rm e_1} = r_{\rm e_2} = r_{\rm e}$, $r_{\rm i_1} = r_{\rm i_2} = r_{\rm i}$, $\sigma^2_{\rm e_1} = \sigma^2_{\rm e_2} = \sigma^2_{\rm e}$, $\sigma^2_{\rm i_1} = \sigma^2_{\rm i_2} = \sigma^2_{\rm i}$, $\gamma_{\rm e_1 i_2} = \gamma_{\rm i_1 e_2} = \gamma_{\rm ei}$, and $\gamma_{\rm e_1 i_1} = \gamma_{\rm e_2 i_2} = 0$. The assumption that $\gamma_{\rm e_1 i_1} = \gamma_{\rm e_2 i_2} = 0$ may not hold in general (Okun and Lampl, 2008). However, correlations between the inputs to a cell simply change the input variances, $\sigma^2_{\rm in}$, and their effect has been studied (Salinas and Sejnowski, 2000, 2002). To incorporate such correlations in our model, one substitutes $\sigma^2_{\rm in} = \sigma^2_{\rm e} + \sigma^2_{\rm i} - 2\gamma_{{\rm e}_j{\rm i}_j}$.

We denote the excitation-to-inhibition ratio by $q = r_{\rm e}/r_{\rm i}$. Due to our choice of units, the excitatory and inhibitory input currents to cell j are the point processes, ${\rm e}_j(t)$ and $-{\rm i}_j(t)$, with mean values $r_{\rm e}$ and $r_{\rm i}$ respectively. The total input current is given by ${\rm in}_j(t) = {\rm e}_j(t) - {\rm i}_j(t)$ with mean $\mu_{\rm in} = E[{\rm in}_j(t)] = r_{\rm e} - r_{\rm i}$, variance $\sigma^2_{\rm in} = \sigma^2_{\rm e} + \sigma^2_{\rm i}$, and correlation

$$\rho_{\rm in} = \rho_{{\rm in}_1{\rm in}_2} = \frac{\rho_{\rm ee}\sigma^2_{\rm e} + \rho_{\rm ii}\sigma^2_{\rm i} - 2\rho_{\rm ei}\sigma_{\rm e}\sigma_{\rm i}}{\sigma^2_{\rm e} + \sigma^2_{\rm i}} \tag{3}$$

where $\rho_{\rm ee} = \rho_{\rm e_1 e_2}$, $\rho_{\rm ii} = \rho_{\rm i_1 i_2}$, and $\rho_{\rm ei} = \rho_{\rm e_1 i_2} = \rho_{\rm e_2 i_1}$ are the excitatory-to-excitatory, inhibitory-to-inhibitory, and excitatory-to-inhibitory correlations (Shadlen and Newsome, 1998; Salinas and Sejnowski, 2000). To generate pairs and quadruples of correlated spike trains for simulations, we used the algorithms outlined in Appendix A.

2.4 The statistics of the output spike trains

In addition to symmetry between the input statistics, we also assume that the two neurons are dynamically identical (this is also relaxed in the Appendix). Hence the output spike trains, $\mathrm{out}_j(t)$, are statistically identical. Define the rate, variance, covariance, Fano factor, synchrony, and correlation of the two output spike trains as $r_{\mathrm{out}} = r_{\mathrm{out}_j}$, $\sigma^2_{\mathrm{out}} = \sigma^2_{\mathrm{out}_j}$, $\gamma_{\mathrm{out}} = \gamma_{\mathrm{out}_1\mathrm{out}_2}$, $F_{\mathrm{out}} = \sigma^2_{\mathrm{out}}/r_{\mathrm{out}}$, $S_{\mathrm{out}} = S_{\mathrm{out}_1\mathrm{out}_2}$ and $\rho_{\mathrm{out}} = \rho_{\mathrm{out}_1\mathrm{out}_2} = \gamma_{\mathrm{out}}/\sigma^2_{\mathrm{out}}$, respectively. When the outputs are renewal processes, the output coefficient of variation is given by $\mathrm{CV}_{\mathrm{out}} = \sqrt{F_{\mathrm{out}}}$.

In Appendix C, we use the renewal properties of the output spike trains, and the Markov properties of the membrane potentials for the LIF and dLIF models with Poisson inputs to derive the following expression for the output correlation,

$$\rho_{\mathrm{out}} = \frac{\mathrm{CV}^2_{\mathrm{out}} + 1}{\mathrm{CV}^2_{\mathrm{out}}} \left(\frac{E[\tau_1] - E[\tau_1 \,|\, V_2 \nearrow \theta]}{E[\tau_1]} \right) + \frac{S_{\mathrm{out}}}{\mathrm{CV}^2_{\mathrm{out}}}. \tag{4}$$

Here $E[\tau_1 \,|\, V_2 \nearrow \theta]$ is the expected time until the next spike in neuron 1 given that neuron 2 has just spiked, and $E[\tau_1] = \left(\mathrm{CV}^2 + 1\right) / \left(2r_{\mathrm{out}}\right)$ is the expected time until the next spike in neuron 1 starting from an arbitrary initial time (referred to in Cox (1962) as the *expected recurrence time*). This expression is exact for uncoupled integrate-and-fire models, instantaneous synapses, and white inputs. It is approximately valid for coupled models receiving non-white inputs or non-instantaneous synapses in the fluctuation dominated regime. We are unaware of a similar expression in the literature, and we use it in Sec. 4 to analyze the transfer of correlations in the fluctuation dominated regime. The expression is also useful for calculating asymptotic correlations in simulated data. We found it to be much faster and more accurate than standard methods for calculating the correlation between two LIF neurons, see the discussion in Appendix C.

2.5 The drift dominated regime and the perfect integrate-and-fire (PIF) model

In drift dominated regimes, where the excitatory current dominates the inhibitory and leak currents ($r_{\mathrm{e}} \gg r_{\mathrm{i}} + \theta/\tau_m$), the lower reflecting barrier at β is visited rarely. In addition, the dynamics of the neuron are dominated by the input and a good approximation is obtained by ignoring the leak current (see Fig. 2C). We therefore approximate the LIF in drift dominated regimes by the analytically tractable perfect integrate-and-fire (PIF) model, which is obtained by setting $1/\tau_m = 0$ in Eq. (2) and ignoring the lower boundary at β (Gerstein and Mandelbrot, 1964; Knight, 1972).

Note that the PIF model is a good approximation when the membrane time constant is slow compared to inputs and the cells *integrate* their inputs. It is not a good approximation when the membrane time constant is fast and the membrane potentials *track* their inputs, as occurs in conductance based models in high conductance states.

The output statistics of the PIF can be obtained analytically. When $\mu_{\mathrm{in}} = r_{\mathrm{e}} - r_{\mathrm{i}} > 0$, the output rate is $r_{\mathrm{out}} = \mu_{\mathrm{in}}/\theta$, and when $\mu_{\mathrm{in}} \leq 0$, the output rate is zero. Thus the rate transfer function of the PIF is threshold-linear. Hereafter we assume that $\mu_{\mathrm{in}} > 0$ when considering the PIF model. The total variance of the output spike trains and Fano factor are $\sigma^2_{\mathrm{out}} = (\sigma^2_{\mathrm{e}} + \sigma^2_{\mathrm{i}})/\theta^2$, and $F_{\mathrm{out}} = (\sigma^2_{\mathrm{e}} + \sigma^2_{\mathrm{i}})/[\theta(r_{\mathrm{e}} - r_{\mathrm{i}})]$ respectively (see Appendix B). For Poisson inputs this yields

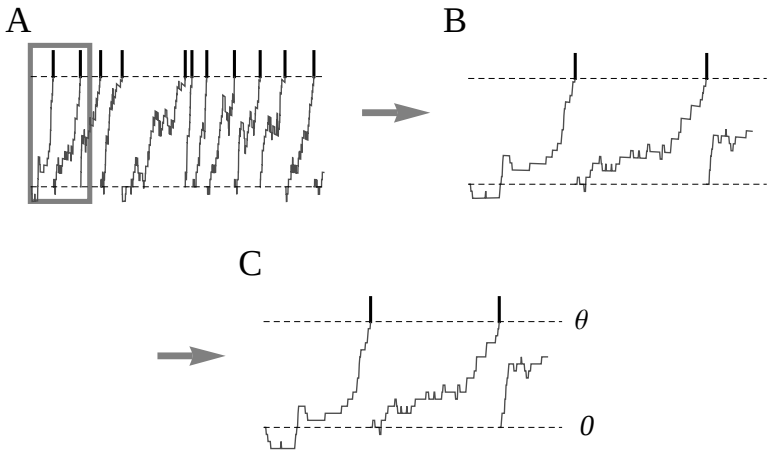

Figure 2: **An LIF in the drift dominated regime can be approximated with a PIF. (A)** The typical behavior of an LIF in the drift dominated regime ($r_{\mathrm{e}} = 2.5$KHz, $r_{\mathrm{i}} = 1$KHz, $\tau_m = 20$ms and $\theta = 15$). **(B)** Same as (A), except time was rescaled using the fast input timescale, so that (B) represents the region inside the gray box in (A). **(C)** The response of a PIF driven by the same input.

$\mathrm{CV}_{\mathrm{out}} = \sqrt{(q+1)/[\theta(q-1)]}$ where $q = r_{\mathrm{e}}/r_{\mathrm{i}} > 1$ measures the excitation-to-inhibition balance. In Fig. 3 the output rate and CV are plotted as dashed lines.

2.6 The discrete LIF (dLIF) model

Outside of the drift dominated regime, spiking is increasingly due to fluctuations of the membrane potential around its mean value (Salinas and Sejnowski, 2000; Ringach and Malone, 2007). In such regimes, the PIF no longer provides a good approximation and we instead use the analytically tractable discrete LIF (dLIF) model which is defined by

$$dV = \mathrm{e}(t)dt - \mathrm{i}(t)dt - I_L(t)dt, \tag{5}$$

with a threshold at θ, reset at 0, and a reflecting lower boundary at $\beta \leq 0$. Here, $I_L(t) = \sum_i \delta(t - t_i)$ is a Poisson point process with rate $\bar{I}_L$ that models a leak current. Using Poisson jumps to model leak may at first seem unnatural. However, the dLIF can be thought of as a noisy integrate–and–fire model with constant leak (Fusi and Mattia, 1999). We use the dLIF because it is analytically tractable and captures the fundamental properties of correlation transfer in more realistic leaky models.

In parameter regimes where the input currents dominate the leak current, the dLIF provides a good quantitative approximation to the LIF. Outside of such regimes, it captures the qualitative dependence of the spiking statistics on parameters. We emphasize that the purpose of the model is not to quantitatively approximate the LIF (which is itself a simplified model). Instead the dLIF serves as an analytically tractable leaky model that can be used to understand the principal mechanisms that shape correlation transfer.

When $\mathrm{e}(t)$ and $\mathrm{i}(t)$ are Poisson, the membrane potential $V(t)$ for the dLIF model is a continuous-time Markov process on a discrete state space and we can compute the univariate and bivariate spiking statistics exactly (see Appendix D). It is hereafter assumed that $\mathrm{e}(t)$ and $\mathrm{i}(t)$ are Poisson when referring to the dLIF model. The stationary firing rate and CV for the dLIF are derived in closed form in Appendix D,

$$r_{\mathrm{out}} = \frac{(\hat{q}-1)^2}{\hat{q}\left((\hat{q}^{-\theta}-1)\,\hat{q}^{\beta}+\theta(\hat{q}-1)\right)}\, r_{\mathrm{e}}, \tag{6}$$

and

$$\mathrm{CV}^2_{\mathrm{out}} = \frac{4(\hat{q}(\theta-\beta+1)-\theta+\beta)\hat{q}^{\theta+\beta}+\hat{q}^{2\beta}+\hat{q}^{2\theta}\left(-\left(\hat{q}^{2\beta}-4(\hat{q}(\beta-1)-\beta)\hat{q}^{\beta}-\hat{q}^2\theta+\theta\right)\right)}{\left(\hat{q}^{\beta}-\hat{q}^{\theta}\left(\hat{q}^{\beta}-\hat{q}\theta+\theta\right)\right)^2}, \tag{7}$$

where $\hat{q} = r_{\mathrm{e}}/(r_{\mathrm{i}}+\bar{I}_L)$. Throughout the text, we take $\beta = -2$ and $\theta = 30$. Fig. 3 shows that the input-to-output rate curve has the threshold-linear shape that is typical of integrate-and-fire neurons. In the fluctuation dominated regime, $\mathrm{CV}_{\mathrm{out}} \approx 1$, while in the drift dominated regime, $\mathrm{CV}_{\mathrm{out}}$ is decreased.

We were unable to derive closed form expressions for the bivariate and the time-dependent univariate spiking statistics. However, since the membrane potentials are a Markov process on a discrete state space, their exact time dependent distributions can be found by exponentiating their infinitesimal generator matrix and the stationary distribution is given by the dominant eigenspace of the generator. These methods are discussed in detail in Appendix D, and a suite of Matlab programs that implement these methods can be found at `http://www.mathworks.com/matlabcentral/fileexchange/28686`

2.7 The memory timescale of the dLIF model

The membrane potential of the dLIF model with Poisson inputs is an ergodic Markov process. The infinitesimal generator for such a process has exactly one zero left eigenvalue and the remaining left eigenvalues have negative real part (Karlin and Taylor, 1975). The non-zero left eigenvalue with real part nearest to zero, λ_1, determines the timescale at which the membrane potential relaxes to its stationary distribution. In particular, defining $\tau_{\mathrm{mem}} = -1/\mathrm{Re}(\lambda_1)$, the distribution relaxes to its steady state exponentially like $e^{-t/\tau_{\mathrm{mem}}}$. We use this result in Secs. 4 and 7 to analyze the asymptotic correlation and to estimate the tail of the cross-covariance function for the dLIF model. See Appendix D for a more complete discussion.

3 Correlations are nearly preserved in drift dominated regimes

When excitation is stronger than inhibition and leak, the membrane dynamics of a leaky model can be approximated by the PIF (see the Methods and Fig. 2). Input to the model neurons, $\mathrm{in}_j(t) = \mathrm{e}_j(t) - \mathrm{i}_j(t), j = 1, 2$, is a sum of excitatory, $\mathrm{e}_j(t)$, and inhibitory, $\mathrm{i}_j(t)$, components. When

$\mathrm{e}_j(t)$, $\mathrm{i}_j(t)$, $\mathrm{out}_j(t)$	Spike trains: excitatory input, inhibitory input, and output for neuron $j = 1, 2$.
$\mathrm{in}_1(t)$, $\mathrm{in}_2(t)$	Total input: $\mathrm{in}_j(t) = \mathrm{e}_j(t) - \mathrm{i}_j(t)$.
$N_{\mathrm{e}_j}(t)$, $N_{\mathrm{i}_j}(t)$, $N_{\mathrm{in}_j}(t)$, $N_{\mathrm{out}_j}(t)$	Spike counts: $N_X(t) = \int_0^t X(s)ds$.
r_{e}, r_{i}, r_{out}	Firing rates: excitatory, inhibitory, and output.
q	Excitation-to-inhibition balance: $q = \frac{r_{\mathrm{e}}}{r_{\mathrm{i}}}$.
σ^2_{e}, σ^2_{i}, σ^2_{in}, σ^2_{out}	Asymptotic spike count variances.
F_{e}, F_{i}, F_{out}	Asymptotic Fano factors: $F_x = \frac{\sigma_x^2}{r_x}$.
$\mathrm{CV}_{\mathrm{out}}$	Output coefficient of variation.
γ_{ee}, γ_{ii}, γ_{ei}, γ_{in}, γ_{out}	Asymptotic spike count covariances: excitatory-to-excitatory, inhibitory-to-inhibitory, excitatory-to-inhibitory, total input, and output-to-output.
ρ_{ee}, ρ_{ii}, ρ_{ei}, ρ_{in}, ρ_{out}	Asymptotic spike count correlations: $\rho_{xy} = \gamma_{xy}/(\sigma_x\sigma_y)$.
S_{out}	Output synchrony, $S_{\mathrm{out}} = \frac{r_{\mathrm{synch}}}{r_{\mathrm{out}}}$ where r_{synch} is the rate of synchronous output spikes.
θ, β	Threshold and lower boundary for IF models
τ_m	Membrane time constant for the LIF model.
$\bar{I}_L$	Mean leak current for the dLIF model
τ_{mem}	Memory timescale for the dLIF model.

Table 1: **Notation for parameters and spike train statistics.** We assume symmetry between cells (e.g., $r_{\mathrm{e}_1} = r_{\mathrm{e}_2} = r_{\mathrm{e}}$) throughout the text, except in the Appendix.

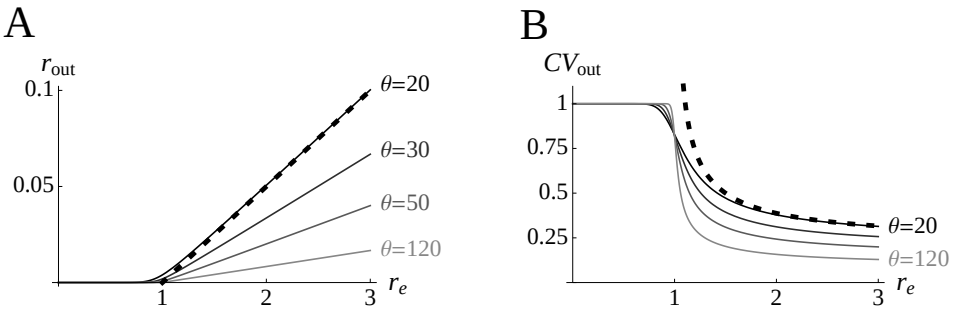

Figure 3: **The univariate spiking statistics for the dLIF. (A)** The output firing rate and **(B)** the output CV as functions of the excitatory input rate, r_{e}, for $\beta = -2$ and for $\theta = 20$, 30, 50, and 120. The dashed lines show the output statistics of the PIF when $\theta = 20$ and $r_{\mathrm{i}} = 1$. Both r_{e} and r_{out} are in units of $r_{\mathrm{i}} + \bar{I}_L$ (equivalently, $r_{\mathrm{i}} + \bar{I}_L = 1$ is fixed) so that $\hat{q} = r_{\mathrm{e}}$.

$r_\mathrm{e} - r_\mathrm{i} > 0$, the output firing rate of the PIF is positive, with spike count

$$\begin{aligned} N_{\mathrm{out}_j}(t) &= \frac{N_{\mathrm{e}_j}(t) - N_{\mathrm{i}_j}(t) - V_j(t) + V_j(0)}{\theta} \\ &= \frac{1}{\theta} N_{\mathrm{in}_j}(t) + \mathcal{O}(1), \qquad j = 1, 2 \end{aligned} \tag{8}$$

where $\mathcal{O}(1)$ represents terms bounded in time. Thus for large t, the input and output spike counts are linearly related.

This implies that $\sigma^2_\mathrm{out} = \sigma^2_\mathrm{in}/\theta^2$ and $\gamma_\mathrm{out} = \gamma_\mathrm{in}/\theta^2$. The covariance and variance are scaled by the same factor, and therefore the correlation coefficient is unchanged by a layer of PIFs,

$$\rho_\mathrm{out} = \rho_\mathrm{in} = \frac{\rho_\mathrm{ee}\sigma^2_\mathrm{e} + \rho_\mathrm{ii}\sigma^2_\mathrm{i} - 2\rho_\mathrm{ei}\sigma_\mathrm{e}\sigma_\mathrm{i}}{\sigma^2_\mathrm{e} + \sigma^2_\mathrm{i}}. \tag{9}$$

The fact that $\rho_\mathrm{out} = \rho_\mathrm{in}$ is valid for a pair of PIFs with arbitrary stationary inputs with positive mean (see Appendix B for a detailed proof). Thus the result does not depend on the assumption of instantaneous postsynaptic potentials and remains true when inputs are modeled as continuous (e.g., white) noise (Vilela and Lindner, 2009). It follows that a threshold mechanism or a threshold-linear f-I curve alone is not enough to reduce correlations.

We conclude that a pair of LIFs in the drift dominated regime nearly preserve correlations. This conclusion is consistent with previous observations for LIF models driven by correlated, positively biased white noise (de la Rocha et al., 2007; Shea-Brown et al., 2008; Tchumatchenko et al., 2008; Vilela and Lindner, 2009) and is verified for the LIF with discrete post-synaptic potentials in Fig. 4. In the drift dominated regime, output correlations for a pair of LIFs approximately match the theoretical values obtained for PIFs. Outside of this regime, the LIF output correlations are reduced in magnitude. We investigate this reduction of correlations next.

4 Correlations are reduced in fluctuation dominated regimes

When input to the cells is weaker and firing rates lower, correlations are reduced in the output (Stroeve and Gielen, 2001; de la Rocha et al., 2007; Shea-Brown et al., 2008; Tchumatchenko et al., 2008). In this section, we provide a mechanistic explanation of this reduction in correlations, which can be observed in the LIF simulations in Fig. 4. Although our explanation applies to a wide class of neuron models, we illustrate the results with the dLIF model described in the Methods. This model is simple enough that the output correlation and other quantities of interest can be computed exactly, yet it captures the overall features of correlation transfer in both the drift and fluctuation dominated regimes.

The fact that the PIF preserves correlations relies on an asymptotically linear and deterministic relation between the input and output spike counts, *cf.* Eq. (8). The same relation holds approximately for the LIF in drift dominated regimes since leak has a small effect, and the lower boundary of the membrane voltage is visited rarely (see Fig. 2).

However, in the fluctuation dominated regime where spiking is caused by random fluctuations of the membrane potential, the output spike count over large windows depends on the timing of

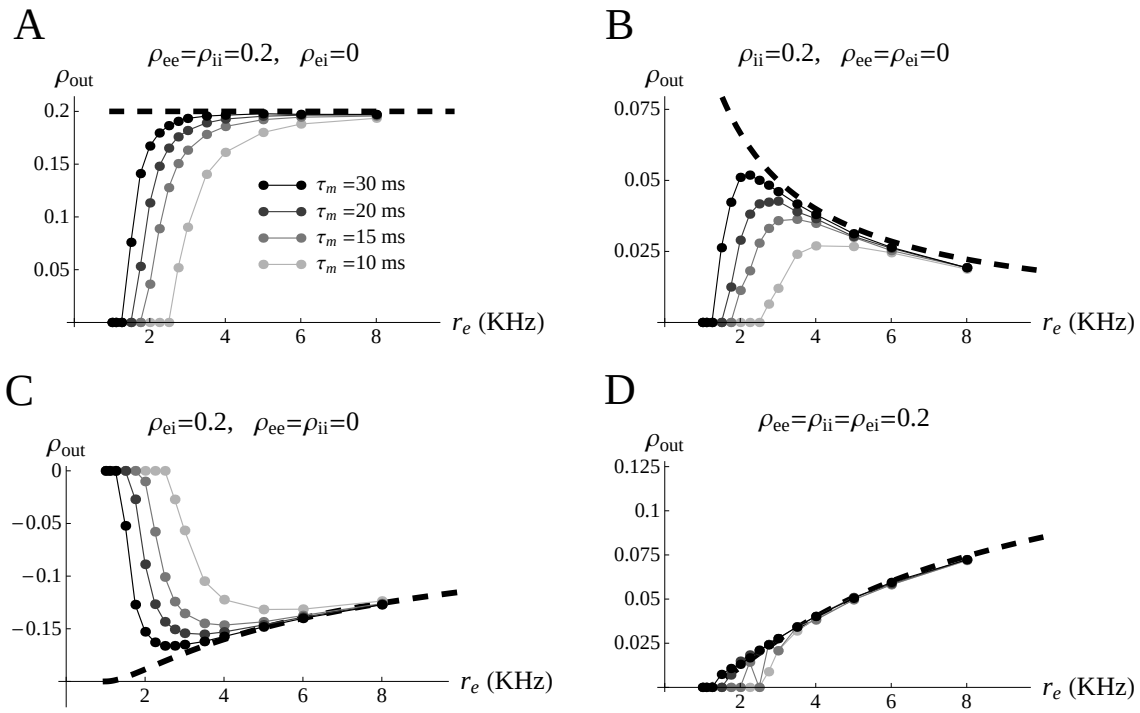

Figure 4: **Correlation transfer for the LIF model.** In each panel a set of input correlations, $\rho_{\rm ee}$, $\rho_{\rm ii}$ and $\rho_{\rm ei}$ is fixed. Output correlations, $\rho_{\rm out}$, are shown as functions of the excitatory input rate when $r_{\rm i} = 1$KHz. Thick dashed lines represent the output correlation for the PIF, *cf.* Eq. (9). Thin solid lines represent output correlation from simulations of the LIF with correlated Poisson inputs. Along each dashed line the membrane time constant is held fixed and is larger for darker lines (see legend). As the rate of excitation increases relative to inhibition and relative to leak, the LIF is better approximated by the PIF. The output rates for the LIF varied from $< 10^{-3}$Hz to 216Hz. The PIF and LIF agree well (equivalently, correlations are nearly preserved) for moderate firing rates, e.g. $|\rho_{\rm LIF} - \rho_{\rm PIF}| \leq 0.1\rho_{\rm PIF}$ when $r_{\rm out} \geq 40$Hz and $\tau_m = 20$ms in (A). Correlation parameters are **(A)** $\rho_{\rm ee} = \rho_{\rm ii} = 0.2$ and $\rho_{\rm ei} = 0$. **(B)** $\rho_{\rm ii} = 0.2$ and $\rho_{\rm ee} = \rho_{\rm ei} = 0$. **(C)** $\rho_{\rm ei} = 0.2$ and $\rho_{\rm ee} = \rho_{\rm ii} = 0$. **(D)** $\rho_{\rm ee} = \rho_{\rm ii} = \rho_{\rm ei} = 0.2$. Here, and in all subsequent figures, sample points from simulations are marked with dots and error bars are not drawn when the standard errors are smaller than the diameter of the dots. Otherwise, error bars have radius of one standard error.

input spikes, instead of the input spike count alone. As a result, the relationship between input and output spike counts is stochastic and non-linear so that Eq. (8) is no longer valid. To understand correlation transfer in the fluctuation dominated regime, we instead consider the following equation for the output correlation derived in Appendix C,

$$\rho_{\text{out}} = \frac{\text{CV}_{\text{out}}^2 + 1}{\text{CV}_{\text{out}}^2} \left(\frac{E[\tau_1] - E[\tau_1 \,|\, V_2 \nearrow \theta]}{E[\tau_1]} \right) + \frac{S_{\text{out}}}{\text{CV}_{\text{out}}^2}.$$

Here $E[\tau_1 \,|\, V_2 \nearrow \theta]$ is the expected time until the next spike in neuron 1 given that neuron 2 has just spiked, and $E[\tau_1] = \left(\text{CV}^2 + 1\right) / \left(2r_{\text{out}}\right)$ is the expected time until the next spike in neuron 1 starting from an arbitrary initial time (referred to in Cox (1962) as the *expected recurrence time*).

When the excitatory inputs are correlated synchronously ($S_{\text{e}_1\text{e}_2} > 0$), there is a non-zero probability of an exactly synchronous spike in neuron 1 and neuron 2. This leads to positive values of S_{out} and thereby increases ρ_{out}. However, in the fluctuation dominated regime, S_{out} is small and can be ignored (see Fig. 6 inset). Also, in this regime firing is approximately Poissonian so that $\text{CV}_{\text{out}}^2 \approx 1$ (see Fig. 3B) and $(\text{CV}_{\text{out}}^2 + 1)/\text{CV}_{\text{out}}^2 \approx 2$. Therefore, in the fluctuation dominated regime, changes in ρ_{out} are dominated by the "memory",

$$M = \frac{E[\tau_1] - E[\tau_1 \,|\, V_2 \nearrow \theta]}{E[\tau_1]}.$$

which quantifies the relative impact of a spike in neuron 2 on the time until the next spike in neuron 1. In particular, $\rho_{\text{out}} \approx 2M$ in the fluctuation dominated regime.

When V_1 and V_2 are independent, $E[\tau_1 \,|\, V_2 \nearrow \theta] = E[\tau_1]$ and $S_{\text{out}} = 0$ so that $\rho_{\text{out}} = 0$. When V_1 and V_2 are positively correlated, conditioning on V_2 being at threshold increases the probability that V_1 is near threshold. This decreases the expected time for V_1 to reach threshold, yielding $E[\tau_1 \,|\, V_2 \nearrow \theta] \leq E[\tau_1]$, and a positive value of M. A positive value of M implies a positive value of ρ_{out} since $S_{\text{out}} \geq 0$. Similarly, when V_1 and V_2 are negatively correlated, the expected time until V_1 reaches threshold is *lengthened* by conditioning on V_2 being at threshold. Therefore $E[\tau_1 \,|\, V_2 \nearrow \theta] \geq E[\tau_1]$, resulting in negative output correlations when S_{out} is sufficiently small.

When excitation is weak in relation to inhibition and leak, firing is due to rare excursions of the membrane potential across threshold (Paninski, 2006; Ringach and Malone, 2007). The stationary distribution of the membrane potentials is concentrated near rest, but conditioning on a spike in neuron 2 pushes the distribution of V_1 closer to threshold. The distribution of V_1 then relaxes back to its stationary distribution. The timescale of this relaxation is given by the memory timescale, τ_{mem} (see Methods). In Fig. 5A, we show that the memory timescale is much faster than the spiking timescale ($\tau_{\text{mem}} \ll E[\tau_1]$) in the fluctuation dominated regime. This is due to the fact that the spiking dynamics are much slower than the subthreshold dynamics in this regime. The result of this effect is illustrated in Fig. 5B: The distribution of V_1 settles to its stationary state long before the next spike. Neuron 1 effectively *forgets* the effects of the spike in neuron 2 before it has a chance to spike (Knight, 1972). Therefore a spike in cell 2 has a small impact on the waiting time to the next spike in cell 1 and the output spike trains are nearly independent. As a result, $E[\tau_1] \approx E[\tau_1 \,|\, V_2 \nearrow \theta]$ (the arrows in Fig. 5B are close together) so that $M \approx 0$, and therefore $\rho_{\text{out}} \approx 0$.

As r_{e} increases towards the drift dominated regime, conditioning on V_2 being at threshold has

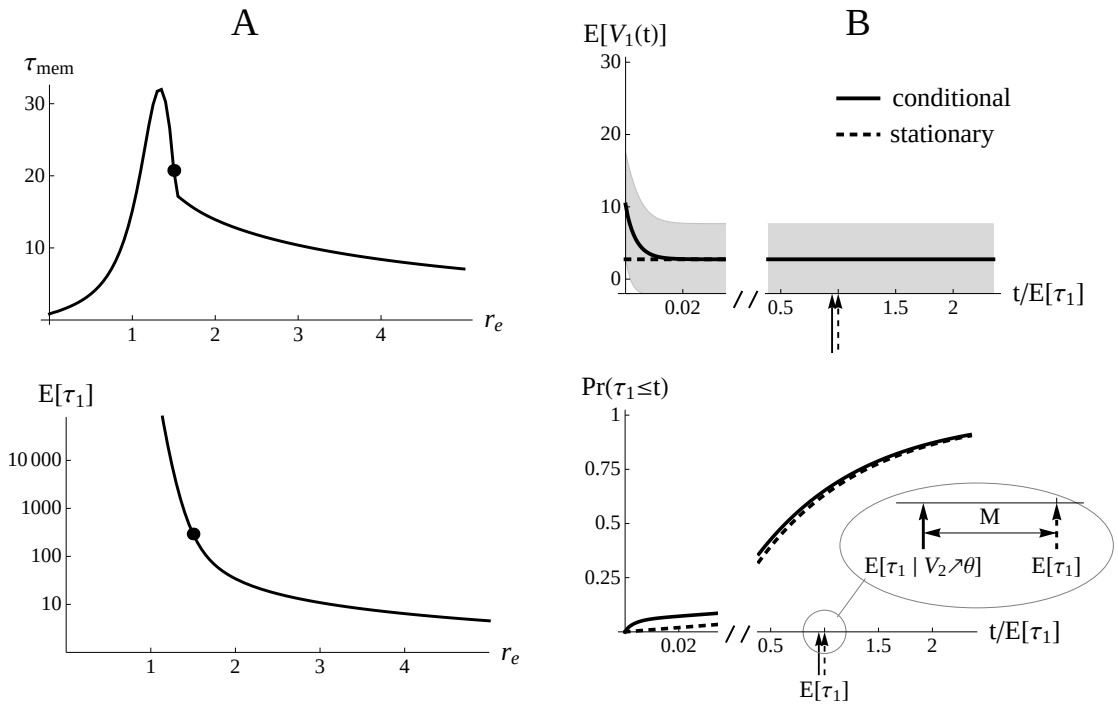

Figure 5: **The forgetfulness of cells in the fluctuation dominated regime. (A)** The memory timescale (τ_{mem}) and the spiking timescale ($E[\tau_1]$) plotted as a function of r_{e} when $r_{\text{i}} = 1$ and $\bar{I}_L = 0.5$. The filled circles indicate the boundary between the fluctuation and drift dominated regimes: $r_{\text{e}} = r_{\text{i}} + \bar{I}_L$. **(B)** Top: The mean membrane potential of neuron 1 conditioned on a spike in neuron 2 at time $t = 0$ (solid line). The shaded region represents the mass within one standard deviation of the mean and the dashed line indicates the stationary mean. Bottom: The cumulative probability distribution of the waiting time, τ_1, of the next spike in neuron 1, conditioned on a spike in neuron two at time $t = 0$ (solid line) and in the stationary case (dashed line). Arrows indicate the expected value of τ_1 in the stationary (solid) and conditional (dashed) cases. The distance between the two arrows is $M = \left(E[\tau_1] - E[\tau_1 \,|\, V_2 \nearrow \theta]\right) / E[\tau_1]$. Parameters in (B) are $r_{\text{e}} = 1.25$, $r_{\text{i}} = 1$, $\bar{I}_L = 0.5$, $\rho_{\text{ee}} = \rho_{\text{ii}} = 0.5$ and $\rho_{\text{ei}} = 0$.

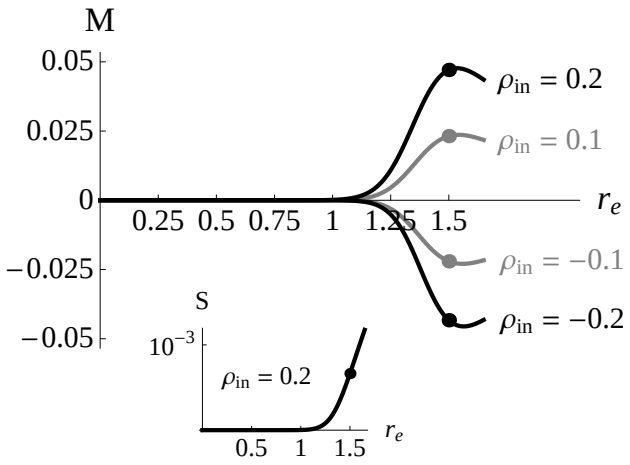

Figure 6: **Dependence of M on r_e for the dLIF model.** Here $\rho_{ee} = \rho_{ii} = \rho_{in}$ and $\rho_{ei} = 0$ for lines in the upper half. For lines in the lower half, $\rho_{ee} = \rho_{ii} = 0$, and ρ_{ei} is chosen so that $\rho_{in} = -0.2$ and -0.1, respectively. For all four lines, $r_i = 1$ and $\bar{I}_L = 0.5$ are fixed (so that r_e and $\bar{I}_L$ are given in units of r_i). The inset shows the output synchrony, S_{out}, as a function of r_e with $\rho_{ee} = \rho_{ii} = 0.2$ and $\rho_{ei} = 0$. Filled circles indicate the values for which $r_e = r_i + \bar{I}_L = 1.5$, which defines the boundary between the fluctuation and drift dominated regimes. When $r_e \ll r_i + \bar{I}_L$, M is approximately 0. As the cell approaches the drift dominated regime, $|M|$ increases. Interestingly, $|M|$ decreases with r_e in the drift dominated regime. However, in this regime S is no longer negligible and CV_{out} decreases with r_e (see inset and Fig. 3B), so that the value of M alone is no longer a good indicator of the value of ρ_{out}.

an increasing relative impact on the expected waiting time until V_1 spikes and, as a result, $|M|$ increases (see Fig. 6). Since $|M|$ dominates in Eq. (4), $|\rho_{out}|$ also increases as the drift dominate regime is approached. Inside the drift dominated regime, $\rho_{out} \approx \rho_{in}$ as discussed previously. The dependence of ρ_{out} on the level of excitation is illustrated for the dLIF in Fig. 7 and is consistent with the LIF simulations in Fig. 4.

The reduction of dependencies between the output spike trains in the fluctuation dominated regime does not depend on our choice of the Pearson correlation coefficient as a measure. When firing is rare, output spike trains become nearly independent. Thus, any reasonable measure of dependence between output spike trains tends to zero in the fluctuation dominated limit. We revisit this observation in the Discussion.

Some combinations of the correlation parameters can lead to non-monotonic behavior of ρ_{out} with respect to r_e. For instance, in Fig. 7B, $\rho_{in} > 0$ so that ρ_{out} initially increases with r_e from 0 towards $\rho_{in} > 0$. However, as r_e continues to grow, uncorrelated excitation dominates and ρ_{out} decreases towards $\rho_{in} \approx \rho_{ee} = 0$. The opposite occurs in Fig. 7C: correlation initially decreases from 0 towards $\rho_{in} < 0$ then increases towards $\rho_{ee} = 0$.

A non-monotonic relationship between r_e and ρ_{out} yields a non-monotonic relationship between r_{out} and ρ_{out} since r_{out} increases with r_e. Therefore, correlations do not necessarily increase with firing rate (de la Rocha et al., 2007). Such mechanisms could underly the attention induced decreases in correlations accompanied by increases in firing rates (Cohen and Maunsell, 2009). This result

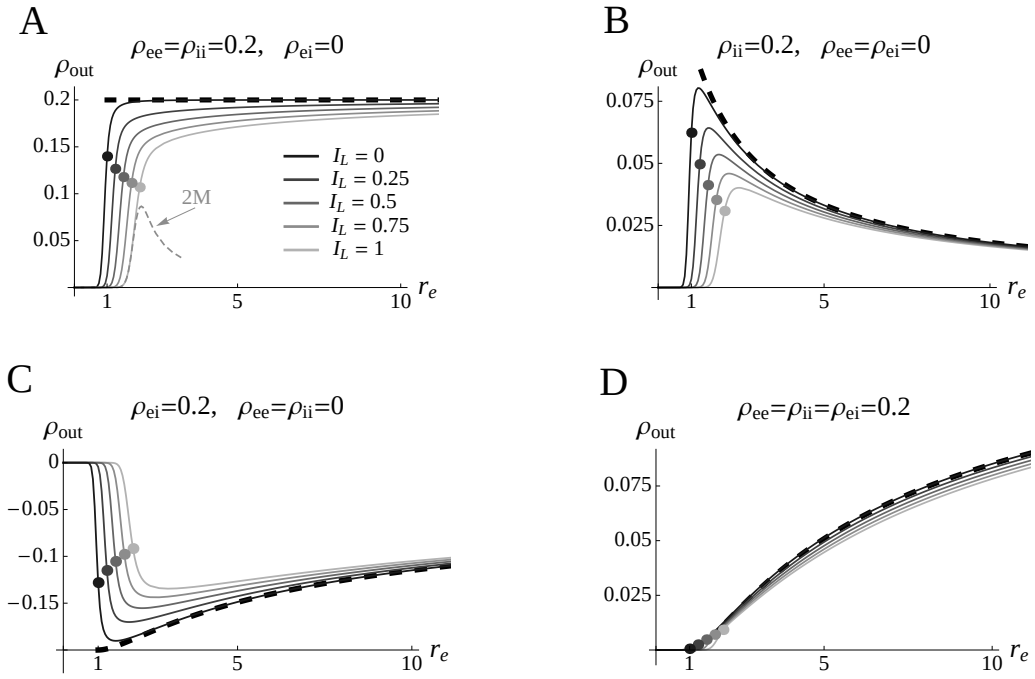

Figure 7: **Correlation transfer for the dLIF model.** The output correlation as a function of the excitatory input rate, $r_{\rm e}$, for different combinations of the correlations parameters, $\rho_{\rm ee}$, $\rho_{\rm ii}$, $\rho_{\rm ei}$, and the mean leak current, $\bar{I}_L$. We fixed $r_{\rm i} = 1$ and varied $r_{\rm e}$ and $\bar{I}_L$, thus $r_{\rm e}$ and $\bar{I}_L$ are given in units of $r_{\rm i}$. The solid lines represent the output correlations for the dLIF and the dashed lines represent values for the PIF (equivalently the input correlation, $\rho_{\rm in}$). The mean leak current, $\bar{I}_L$, decreases with the darkness of the solid lines. The darkest solid line is obtained by setting $\bar{I}_L = 0$, eliminating the leak current altogether. In this case, the dLIF differs from the PIF only by the presence of a lower reflecting barrier at β. When $r_{\rm e} < r_{\rm i}$, this lower barrier has a decorrelating effect. When excitation is stronger, the lower barrier has an insignificant effect on correlations since it is visited rarely. The filled circles indicate the boundary between the drift and fluctuation dominated regimes, $r_{\rm e} = r_{\rm i} + \bar{I}_L$. The correlation parameters are **(A)** $\rho_{\rm ee} = \rho_{\rm ii} = 0.2$ and $\rho_{\rm ei} = 0$. **(B)** $\rho_{\rm ii} = 0.2$ and $\rho_{\rm ee} = \rho_{\rm ei} = 0$. **(C)** $\rho_{\rm ei} = 0.2$ and $\rho_{\rm ee} = \rho_{\rm ii} = 0$. **(D)** $\rho_{\rm ee} = \rho_{\rm ii} = \rho_{\rm ei} = 0.2$.

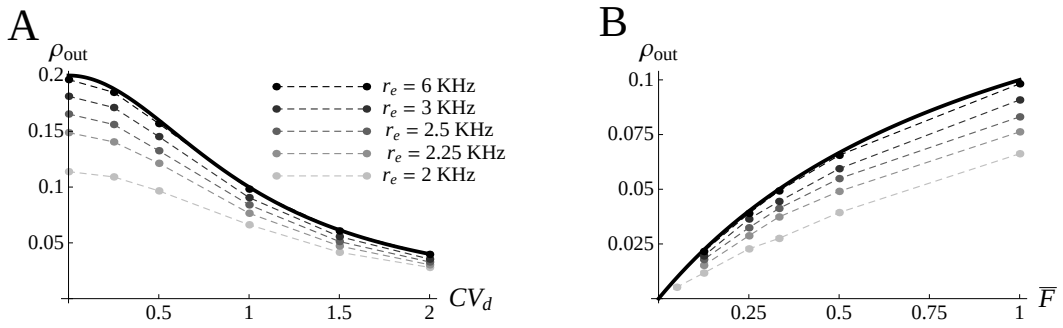

Figure 8: **The effects of synaptic variability on correlation transfer.** Solid lines indicate theoretical values for a pair of PIFs (*cf.* Eq. (10)) and thin dashed lines were obtained from simulations of a pair of LIFs. For the LIF simulations, $r_{\text{i}} = 1$KHz and $\tau_m = 20$ms are fixed and the excitatory input rate, r_{e}, increases with the darkness of the lines (see legend). The correlation parameters are $\rho_{\text{ee}} = \rho_{\text{ii}} = 0.2$ and $\rho_{\text{ei}} = 0$. Inputs are renewal with gamma distributed ISI's (see Appendix A) and postsynaptic amplitudes are random, with peak values drawn independently from a gamma distribution with mean 1 and coefficient of variation CV_d. In the drift dominated limit, the PIF accurately approximates the LIF (see the darkest dotted lines). Outside of this regime (the lighter lines), correlations are reduced but obey the same dependence on the parameters. **(A)** The input Fano factor, $\overline{F}$, is fixed at unity (inputs are Poisson), and the magnitude of synaptic noise, CV_d, is varied. **(B)** The degree of synaptic variability is fixed at $\text{CV}_d = 1$ and $\overline{F}$ is varied.

is not necessarily in opposition to the central result in de la Rocha et al. (2007), which implies an increase in the *correlation susceptibility*, $\rho_{\text{out}}/\rho_{\text{in}}$, with respect to firing rates. In Fig. 7 A, B, and C the correlation susceptibility increases with r_{out} . However in Fig. 7D, $\rho_{\text{in}} = 0$ when $r_{\text{e}} = r_{\text{i}}$, but $\rho_{\text{out}} > 0$ so that the correlation susceptibility is undefined at this point. This phenomenon is explored further in Sec. 8.1.

5 Synaptic variability reduces correlations

Synapses can have a range of efficacies and spikes in presynaptic neurons can elicit a variety of post-synaptic response amplitudes. Furthermore, synaptic failure and random response amplitudes result in variability at the level of single synapses. Release probabilities at a synapse range between less than 0.1 and up to 0.9 (Allen and Stevens, 1994; Thomson, 2000), and the magnitude of the postsynaptic response, evoked by the same cell, can vary with a CV from .25 to 1.5 (Mason et al., 1991; Hessler et al., 1993; Brémaud et al., 2007).

To model synaptic variability, assume that an excitatory (inhibitory) spike at time t_{e}^k (t_{i}^k), causes a random "jump" d_{e}^k (d_{i}^k) in the membrane potential of the post-synaptic cell. Assume that the jumps are drawn independently from a distribution with mean $\overline{d}_{\text{e}}$ ($\overline{d}_{\text{i}}$) and variance $\sigma_{d_{\text{e}}}^2$ ($\sigma_{d_{\text{i}}}^2$). For simplicity we let $\overline{d}_{\text{e}} = \overline{d}_{\text{i}} = \overline{d}$ and $\sigma_{d_{\text{e}}}^2 = \sigma_{d_{\text{i}}}^2 = \sigma_d^2$ (more general results are given in Appendix E.1).

Synaptic noise adds stochasticity to the relationship between input and output spike counts given for the PIF by Eq. (8), but randomness is only introduced at each input spike. As a result, the variance is increased by an amount which depends on the input rates. In particular, for the PIF,

$\sigma^2_{\text{out}} = \left(\sigma^2_{\text{in}} + \text{CV}_d{}^2(r_\text{e} + r_\text{i})\right)/\hat{\theta}^2$ where $\hat{\theta} = \theta/\overline{d}$ is the average number of excitatory kicks needed to reach threshold and $\text{CV}_d = \sigma_d/\overline{d}$. Since synaptic noise was assumed to be independent, the covariance of the outputs is not changed by the noisiness of the synapses, $\gamma_{\text{out}} = \gamma_{\text{in}}/\hat{\theta}^2$. Correlations are therefore reduced as (see Appendix E.1)

$$\rho_{\text{out}} = \left(\frac{\overline{F}}{\overline{F} + \text{CV}_d^2}\right)\rho_{\text{in}} \tag{10}$$

where $\overline{F} = (F_\text{e} r_\text{e} + F_\text{i} r_\text{i})/(r_\text{e} + r_\text{i})$ is the weighted average of the excitatory and inhibitory input Fano factors. The decrease in correlations due to synaptic noise is illustrated in Fig. 8A. Interestingly an increase in the randomness of the input, as measured by $\overline{F}$, will increase the output correlation, but only in the presence of synaptic noise. This effect is illustrated in Fig. 8B.

Synaptic failure can be modeled by assuming that d^k_e and d^k_i are binary random variables in which case $\text{CV}_d^2 = (1-p)/p$, where p is the probability of release. For example when inputs are Poisson ($\overline{F} = 1$), $\rho_{\text{out}} = p$ for the PIF model. Hence, ρ_{out} decreases with an increase in the probability of synaptic failure. When p is small (Allen and Stevens, 1994; Thomson, 2000), correlations are significantly reduced by synaptic failure.

Combining the effects of synaptic failure and variable postsynaptic amplitudes, we obtain

$$\rho_{\text{out}} = \left(\frac{p\,\overline{F}}{p\,\overline{F} + (1-p) + \text{CV}_d^2}\right)\rho_{\text{in}} \tag{11}$$

where we have assumed that a proportion p of the inputs successfully elicit a response, and the amplitudes of the successful synaptic responses are variable with a CV of CV_d. Realistic choices of parameters yeild dramatic reductions in correlations. For example, taking $p = 0.5$, $\text{CV}_d = 1$, and $\text{CV}_\text{e} = \text{CV}_\text{i} = 0.6$ (where $\overline{F} = \text{CV}_\text{e}^2$), correlations are reduced by nearly an order of magnitude by the PIF ($\rho_{\text{out}} = 0.107\rho_{\text{in}}$). Correlations are reduced even further by leaky models, especially in the fluctuation dominated regime. In Fig. 9 we illustrate the effects of synaptic variability on correlations in a simple population model.

As the release probability and PSP amplitude are dependent on input statistics (Czubayko and Plenz, 2002), the independence assumptions made in this section can only be taken as a first approximation. However, the model can be extended to take such dependencies into account.

6 The effect of coupling on correlations

Recurrent connections are common in many parts of the central nervous system, and may play an important role in information processing (Gawne and Richmond, 1993; Kisvárday et al., 1997; Gibson et al., 1999; Lamme and Roelfsema, 2000; Oswald et al., 2009). Synaptic coupling or gap junctions can actively modulate the transfer of correlated inputs (Schneider et al., 2006; Ly and Ermentrout, 2009), and thus affect the information carried by a population of cells (Gutnisky and Dragoi, 2008; Josić et al., 2009).

To model recurrent coupling between two cells, suppose that an action potential in one cell instantaneously raises the membrane potential of the other. We consider a pair of identical, re-

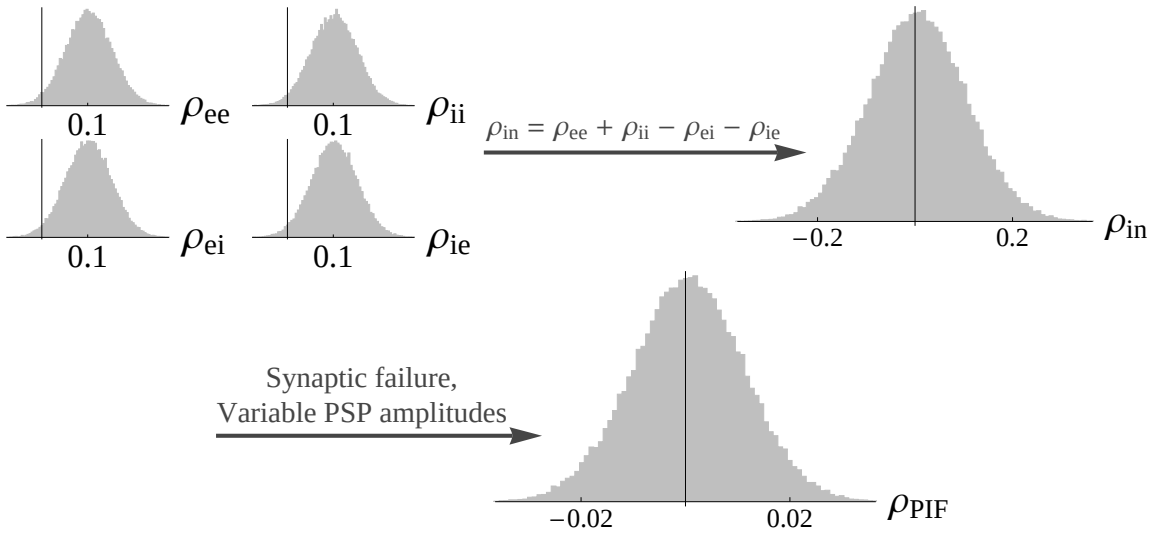

Figure 9: **Correlations are dramatically reduced by unreliable synapses.** The input population has excitatory-to-excitatory, inhibitory-to-inhibitory, and excitatory-to-inhibitory correlations distributed according to a normal distribution with a mean of 0.1 and a standard deviation of 0.05. Assuming homogeneous rates and balanced excitation and inhibition, the input correlations to downstream cells are normally distributed with a mean of 0 (the ei correlations "cancel" with the ee and ii correlations), and a standard deviation of $2 \times 0.05 = 0.1$ (the variances sum). However, realistic levels of synaptic failure, variability of synaptic amplitudes, and non-Poisson input statistics ($\mathrm{CV}_d = 1$, $p = 0.5$, $\mathrm{CV_e} = \mathrm{CV_i} = 0.6$, $\overline{F} = \mathrm{CV_e^2}$, see Sec. 5) decrease output correlations for the PIF by almost an order of magnitude, $\mathrm{std}(\rho_{\mathrm{PIF}}) = 0.0107$. Correlations are reduced even further for leaky models, especially in fluctuation dominated regimes (see Sec. 4 and Fig. 8).

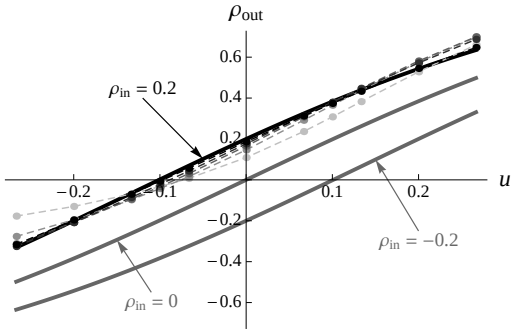

Figure 10: **The effect of coupling on correlation transfer.** Solid lines indicate theoretical values for a pair of PIFs (*cf.* Eq. (12)) and thin dashed lines were obtained from simulations of a pair of LIFs with $\rho_{ee} = \rho_{ii} = 0.2$, $\rho_{ei} = 0$ so that $\rho_{in} = 0.2$. Parameters are the same as in Fig. 8, except that inputs are strictly Poisson and synapses are not random.

ciprocally coupled cells here, but more general results are given in Appendix E.2. The jump in membrane potential, c, due to reciprocal coupling is assumed to be smaller than θ.

The membrane potentials of a pair of coupled PIFs are described by the coupled differential equations.

$$dV_1 = \mathrm{in}_1(t)dt + c\,\mathrm{out}_2(t)dt, \qquad dV_2 = \mathrm{in}_2(t)dt + c\,\mathrm{out}_1(t)dt,$$

with the usual threshold and reset boundary conditions. The analogue of Eq. (8) in this case is a coupled set of linear equations. Their solution can be used to compute the output variance and covariance for the PIF (see Appendix E.2),

$$\sigma^2_{\mathrm{out}} = \frac{\sigma^2_{\mathrm{in}}}{\left(\theta^2 - c^2\right)^2}\left[(\theta^2 + c^2) + 2c\theta\rho_{\mathrm{in}}\right], \qquad \text{and} \qquad \gamma_{\mathrm{out}} = \frac{\gamma_{\mathrm{in}}}{\left(\theta^2 - c^2\right)^2}\left[(\theta^2 + c^2) + \frac{2c\theta}{\rho_{\mathrm{in}}}\right].$$

Since $|\rho_{\mathrm{in}}| < 1$, it follows that coupling has a larger effect on the covariance than on the variance. This can be understood by noting that coupling affects the covariance directly and affects the variance only indirectly (Rangan, 2009): when neuron 1 spikes, the membrane potential of neuron 2 (and therefore the timing of its spikes) is affected directly due to coupling. However, the effect on neuron 1 itself is indirect – a spike in neuron 1 affects the propensity of neuron 2 to spike, which in turn affects the timing of spikes in neuron 1.

The output correlation is

$$\rho_{\mathrm{out}} = \frac{(1+u^2)\rho_{\mathrm{in}} + 2u}{(1+u^2) + 2u\rho_{\mathrm{in}}}, \tag{12}$$

where $u = c/\theta < 1$ is synaptic amplitude relative to the distance from reset to threshold, and measures the strength of the coupling. If the coupling is not too strong, then to first order in u, $\rho_{\mathrm{out}} = \rho_{\mathrm{in}} + 2(1-\rho^2_{\mathrm{in}})u + \mathcal{O}\left(u^2\right)$. Fig. 10 illustrates the dependence of ρ_{out} on u when ρ_{in} is fixed. Not surprisingly, excitatory coupling ($u > 0$) increases correlations and inhibitory coupling ($u < 0$) decreases correlations. Frequently, the amplitude of a single PSP is much smaller than the distance from reset to threshold (i.e., u is small) and therefore the effect of coupling on correlations is small.

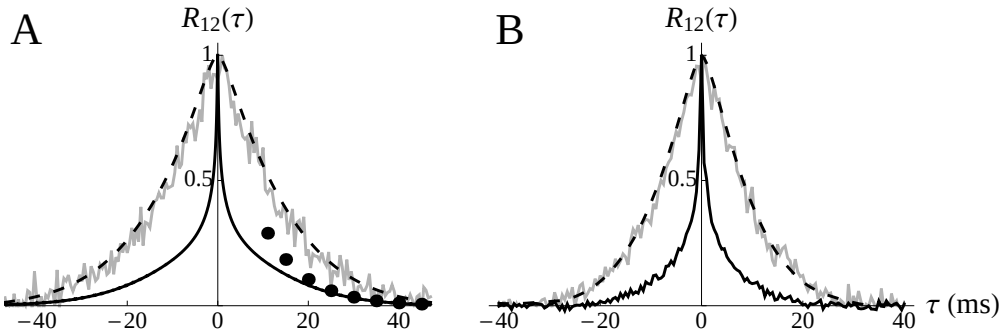

Figure 11: **Output cross-covariance functions.** The output cross-covariance function when inputs are delta-correlated (black lines) decay with a timescale of $\tau_{\rm mem}$ (heavy dots follow $e^{-\tau/\tau_{\rm mem}}$). The gray lines show the output cross-covariance when the input cross-covariance is a double exponential, $(\gamma_{\rm in}/2)e^{-|\tau|/5}$, instead of a delta-function. The dashed line was obtained by convolving the input cross-covariance function with the output cross-covariance function obtained in the delta-correlated case. **(A)** Cros-covariance functions for the dLIF with $r_{\rm e} = 3$, $r_{\rm i} = 2$, $\rho_{\rm ee} = \rho_{\rm ii} = 0.2$, $\rho_{\rm ei} = 0$, and $\bar{I}_L = 0.877$ chosen so that the output rate ($r_{\rm out} = 8.4$Hz) matches with the LIF simulations in (B). The black solid and dashed lines were obtained exactly, without simulations. The grey line is from simulations. **(B)** Cross-covariance function from LIF simulations with the same parameters as in (A) and $\tau_m = 20$ms. In both plots, inputs are Poisson (see Appendix A) and cross-covariance functions are normalized to have a peak value of 1.

7 Cross-covariance functions and the timescale of correlations

So far we have focused on the magnitude of correlations over asymptotically large windows (see the definition of ρ_{ab} in the Methods). However, the timescale over which correlations occur is often of interest in both theoretical and experimental studies (Maršálek et al., 1997; Brody, 1999; Kohn and Smith, 2005; Moreno-Bote and Parga, 2006; Ostojić et al., 2009). We provide a brief discussion of the topic here. A full treatment of the topic will be addressed in a forthcoming publication.

The timescale over which two spike trains are correlated can be measured by their auto- and cross-covariance functions, which can be computed exactly for the dLIF model (see Methods and Appendix D). When inputs are delta-correlated, the tail of the cross-covariance function, $R_{12}(\tau)$, decays exponentially as $\tau \to \infty$. The timescale of this decay is given by the memory timescale, $\tau_{\rm mem}$, of neuron 2 (see dotted line in Fig. 11A) and the $\tau \to -\infty$ tail decays as the memory timescale of neuron 1.

To address the question of how correlation timescales are transferred, the timescale of input correlations must be taken into account. So far, we have concentrated on "delta-correlated" inputs, i.e. inputs whose cross-covariance is a delta function. In particular, the analysis of the dLIF model relied on this assumption. In Fig. 11, we show that the cross-covariance obtained from delta-correlated inputs can be used as an impulse response function for the transfer of cross-covariance functions: The output cross-covariance is well approximated by convolving the input cross-covariance with the output cross-covariance obtained with delta-correlated inputs (compare gray and dashed lines in

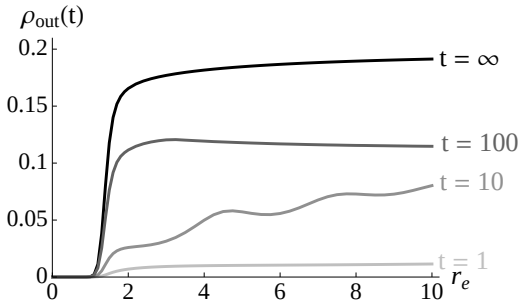

Figure 12: **Correlation over finite windows.** The output spike count correlation, $\rho_{\text{out}}(t)$, over a window of size t, plotted as a function of the input excitatory rate, r_{e} for various values of t. Correlations are smaller for smaller window sizes, but obey the same general dependence on r_{e}.

Fig. 11). Thus, the timescale of output correlations is given by $\tau_{\text{out}} = \max\{\tau_{\text{mem}}, \tau_{\text{in}}\}$ where τ_{in} and τ_{out} are the timescales of the input and output cross-covariance functions respectively. Fig. 11 also illustrates that the cross-covariance functions for the dLIF match those for the LIF qualitatively when the two models have identical input parameters and $\bar{I}_L$ is chosen so that the rates of the two models are matched.

The spike count correlations over finite windows can be computed from the auto- and cross-covariance functions *cf.* Eq. (1). Correlations are smaller for smaller window sizes for an LIF model with white noise inputs (Shea-Brown et al., 2008). The dependence of output correlations on window sizes will be discussed in the authors' forthcoming work.

8 Comparison of results with other models

8.1 Comparison with a white noise Gaussian model

In Fig. 13A, we compare the analytical results for the dLIF and simulations of the LIF with Poisson inputs to a linear response approximation of the LIF with Gaussian white noise inputs, as described by de la Rocha et al. (2007) . The models exhibit the same qualitative dependence on r_{out}, but the dLIF differs from the LIF quantitatively to some extent. Both models are caricatures of actual neurons, and neither should be expected to agree quantitatively with actual recordings. The dLIF has the advantage of being more amenable to analysis and simpler to understand mechanistically. We next describe a regime where the dLIF differs from Gaussian models even qualitatively.

When $2\rho_{\text{ei}}\sqrt{r_{\text{e}}r_{\text{i}}} = \rho_{\text{ee}}r_{\text{e}} + \rho_{\text{ii}}r_{\text{i}}$ the correlation between the total input currents, ρ_{in}, is zero. In such cases, output correlations for the dLIF are positive, but very small – about two orders of magnitude smaller than ρ_{ee} and ρ_{ii} (See Fig. 13B). Note that small correlations on this scale have the potential to significantly impact coding and downstream activity when the output from several neurons is pooled (Zohary et al., 1994; Renart et al., 2010; Rosenbaum et al., 2010). This might explain why large correlations are observed in deeper layers of feedforward networks even when excitation and inhibition are balanced (Litvak et al., 2003; Rosenbaum et al., 2010)

Integrate-and-fire models are able to transfer uncorrelated input currents to correlated outputs because uncorrelated input currents are not necessarily independent. Since the integrate-and-fire

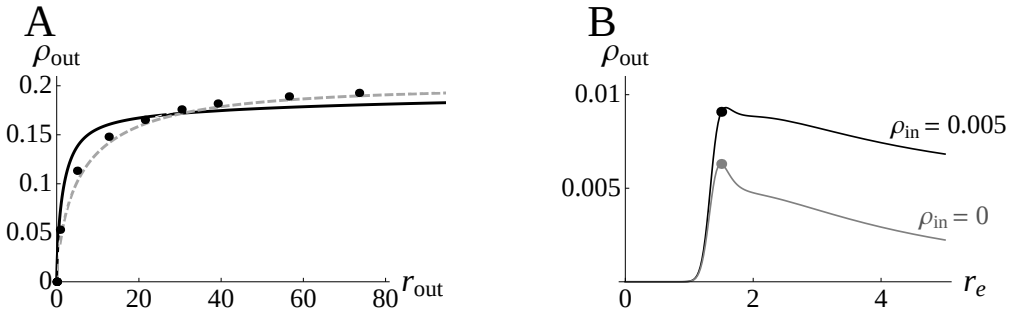

Figure 13: **Comparing the dLIF with a Gaussian model. (A)** Output correlation plotted as a function of output rate for three models: analytical results for the dLIF (solid black line), simulations of an LIF with Poisson inputs (heavy black dots), and a linear response approximation to an LIF with Gaussian white noise input (gray dashed line). For the dLIF and LIF with Poisson inputs, the input parameters are as in Figs. 4A and 7A with $\tau_m = 20$ms and $\bar{I}_L = 0.5$. The white noise inputs for the gray dashed line has bias $\mu = r_{\text{e}} - r_{\text{i}}$, variance $\sigma^2 = r_{\text{e}} + r_{\text{i}}$, and correlation $\rho_{\text{in}} = 0.2$. **(B)** Results are from the dLIF model with $r_{\text{i}} = 1$, $\bar{I}_L = 0.5$, $\rho_{\text{ee}} = 0.5$, and $\rho_{\text{ii}} = 0.5$ fixed. The filled circles indicate the boundary between the drift and fluctuation dominated regimes, $r_{\text{e}} = r_{\text{i}} + \bar{I}_L$. As r_{e} changes, ρ_{ei} is varied so that $\rho_{\text{in}} = 0$ and $\rho_{\text{in}} = 0.005$ respectively. Output correlations are positive even when $\rho_{\text{in}} = 0$. When $\rho_{\text{in}} = 0.005$, correlations can double from input to output. Gaussian models cannot exhibit such increases in correlations.

filter is non-linear, it is possible for moments to "mix" so that higher order input correlations are transferred to second order output correlations. This phenomenon cannot be observed when inputs are modeled by Gaussian processes, since uncorrelated Gaussian processes are necessarily independent. Furthermore, when $2\rho_{\text{ei}}\sqrt{r_{\text{e}} r_{\text{i}}} \approx \rho_{\text{ee}} r_{\text{e}} + \rho_{\text{ii}} r_{\text{i}}$ correlations nearly cancel and $\rho_{\text{in}} \approx 0$. In such cases it is possible that $|\rho_{\text{out}}| > |\rho_{\text{in}}| > 0$ for the dLIF model (See Fig. 13B). This would again be impossible if inputs were modeled using Gaussian processes (Lancaster, 1957).

8.2 Comparison with a conductance-based model

We now compare the results above to simulations of a conductance based integrate-and-fire model (Dayan and Abbott, 2001) similar to the model used in Salinas and Sejnowski (2000). This type of model can accurately capture the statistics of a variety of neuronal responses (Kobayashi et al., 2009). The subthreshold potential obeys the differential equation

$$C_m \dot{V} = -g_L(V - E_L) - g_{AMPA}(t)(V - E_{AMPA}) - g_{GABA}(t)(V - E_{Cl}),$$

where $g_{AMPA}(t) = (\text{e} * epsc)(t)$ and $g_{GABA}(t) = (\text{i} * ipsc)(t)$ are convolutions of the excitatory and inhibitory inputs with post-synaptic conductance kernels. The excitatory (AMPA) post-synaptic conductances were modeled as exponential functions with time constant τ_{AMPA} and peak value $\bar{g}_{AMPA}$,

$$epsc(t) = \bar{g}_{AMPA} e^{-t/\tau_{AMPA}}$$

and the inhibitory (GABA) post-synaptic conductance are double exponentials,

$$ipsc(t) = \frac{\bar{g}_{GABA}}{D}\left(e^{-t/\tau^{(1)}_{GABA}} - e^{-t/\tau^{(2)}_{GABA}}\right)$$

where D is a constant chosen so that $\bar{g}_{GABA}$ is the maximum value of $ipsc(t)$. When the cell crosses threshold, V_θ, a spike is produced and the potential is reset to E_L where it is held for a refractory period, τ_{ref}.

The parameters used in the simulations are $E_L = -60$mV, $E_{AMPA} = 0$, $E_{Cl} = -62$mV, $V_\theta = -54$mV, $\tau_m = 20$ms, $\tau_{AMPA} = 5$ms, $\tau^{(1)}_{GABA} = 5.6$ms, $\tau^{(2)}_{GABA} = .285$ms, $\tau_{\text{ref}} = 2$ms (compare to parameters in Salinas and Sejnowski (2000)). In Figs. 15 and 16, $g_L = C_m/(20\text{ms})$ is fixed to obtain a membrane time constant of $\tau_m = 20$ms. For the simulations in Fig. 14, we used several different values of g_L. For all simulations we set $\bar{g}_{AMPA} = C_m/(909\text{ms})$ so that 30 synchronous excitatory input spikes are required to bring the cell from reset to threshold, in accordance with our choice of $\theta = 30$ for the current based models (see Methods). We then set $\bar{g}_{GABA} = 10.3215 g_{AMPA}$ so that an IPSP is about twice the size of an EPSP when the membrane potential is halfway between rest and threshold.

Figs. 14, 15, and 16 show that the conductance based model transfers correlations in accordance with the theory developed above and illustrated in Figs. 4, 7, 8, and 10. However, in Fig. 14 the magnitude of correlations begin to decay with r_{e} when r_{e} gets large. This is consistent with Shea-Brown et al. (2008) where such a decrease in correlations is attributed to the refractory period. The effect is only significant when r_{out} is on the same order as $1/\tau_{\text{ref}}$. To illustrate this point, we plotted the correlation when $\tau_{\text{ref}} = 0$ and $\tau_m = 30$ms as a dashed line in Fig. 14 (compare to the darkest solid line). The presence of a refractory period causes noticeable decorrelation only once $r_{\text{e}} \geq 3$KHz at which point $r_{\text{out}} \approx 60$Hz.

We also observe that correlations are generally smaller in magnitude for the conductance based model than for the current based models considered above. This may be a consequence of the fact that the model has more sources of non-linearity than the current based models. Another potential explanation is that the effective membrane time constant is reduced when inputs are stronger (Brunel et al., 2001; Kuhn et al., 2004) so that excitation cannot significantly outweigh the "effective leak".

9 Discussion

We used simplified random walk models of neural dynamics to investigate correlation transfer in a variety of settings, and verified that more realistic models obey the same trends. We found that correlations are well preserved in drift dominated regimes when synaptic variability is not taken into account. However, correlations are reduced outside of the drift dominated regime, and reduced further in the presence of synaptic variability and synaptic failure. Positive coupling can increase correlated variability, but only to moderate levels, unless the coupling is strong.

Recent experimental and theoretical studies (Hertz, 2010; Renart et al., 2010) suggest that recurrent network dynamics can modulate correlations to prevent the potential blowup of correlations observed in a feedforward setting (Reyes, 2003; Rosenbaum et al., 2010). These studies agree with *in vivo* recordings that show small (Ecker et al., 2010; Renart et al., 2010) or moderate (Zohary

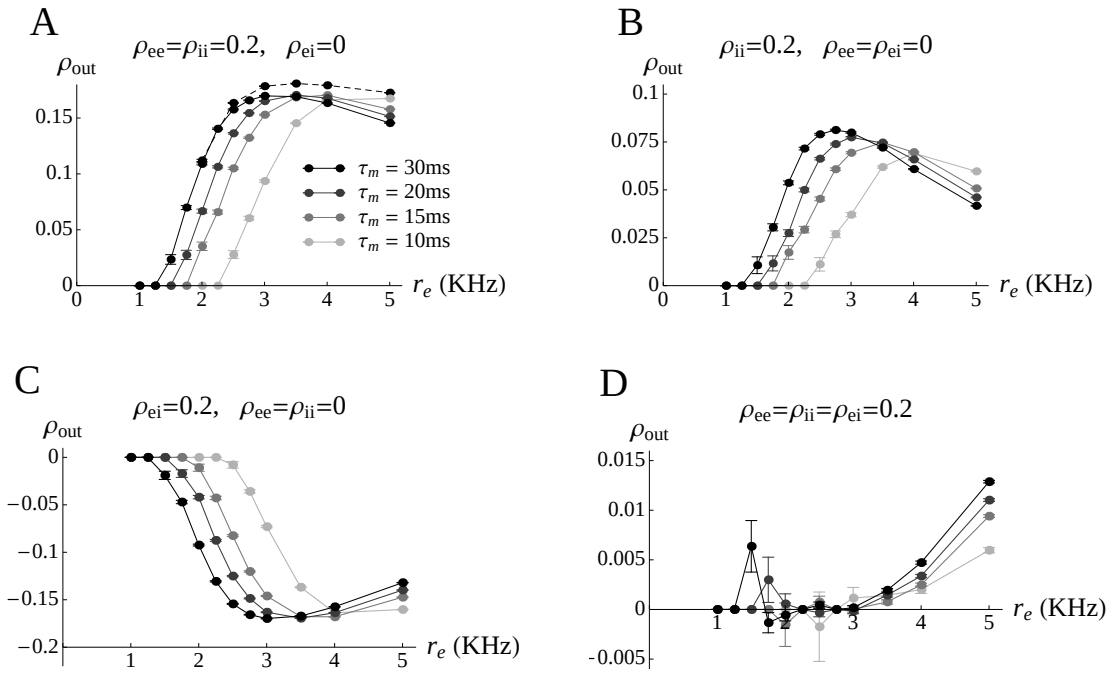

Figure 14: **Output correlation as a function of $r_{\rm e}$ in a conductance based model.** Results from Figs. 4 and 7 are reproduced with a conductance based model. Here, $r_{\rm e}$ and $\tau_m = C_m/g_L$ are varied while $r_{\rm i} = 1$KHz is fixed. The membrane time constant, τ_m, is varied by changing g_L and keeping C_m fixed, so that synaptic conductances are not affected. Inputs are correlated Poisson processes. Output rates varied from $< .01$Hz to 130Hz. For the dashed line in (A), we set $\tau_{\rm ref} = 0$ and $\tau_m = 30$ms to illustrate the effect of a refractory period. Correlations in the inputs are **(A)** $\rho_{\rm ee} = \rho_{\rm ii} = 0.2$ and $\rho_{\rm ei} = 0$. **(B)** $\rho_{\rm ii} = 0.2$ and $\rho_{\rm ee} = \rho_{\rm ei} = 0$. **(C)** $\rho_{\rm ei} = 0.2$ and $\rho_{\rm ee} = \rho_{\rm ii} = 0$. **(D)** $\rho_{\rm ee} = \rho_{\rm ii} = \rho_{\rm ei} = 0.2$.

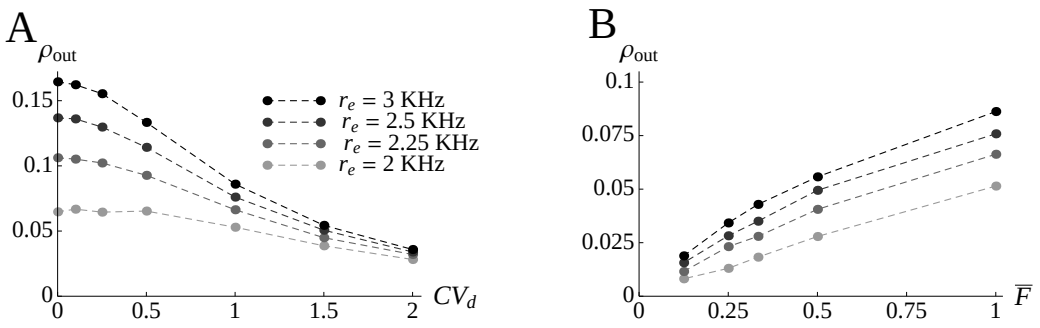

Figure 15: **The effects of synaptic variability on correlation transfer in a conductance-based model.** Results in Fig. 8 are reproduced. Parameters $r_{\text{i}} = 1$KHz and $\tau_m = 20$ms are fixed and the excitatory input rate, r_{e}, increases with the darkness of the lines (see legend). The input correlation parameters are $\rho_{\text{ee}} = \rho_{\text{ii}} = 0.2$ and $\rho_{\text{ei}} = 0$. Inputs are renewal with gamma distributed ISI's (see Appendix A) and EPSCs are random, with peak values drawn independently from a gamma distribution with mean $\bar{g}_{AMPA}$ and coefficient of variation CV_d. **(A)** The input Fano factor, $F_{\text{e}} = F_{\text{i}} = \overline{F}_{\text{in}} = 1$, is fixed and CV_d is varied. **(B)** The synaptic variability, $\text{CV}_d = 1$, is fixed and $F_{\text{e}} = F_{\text{i}} = \overline{F}$ is varied.

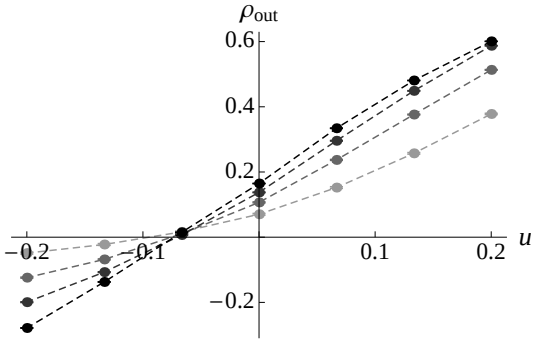

Figure 16: **The effects of coupling on correlation transfer in a conductance-based model.** Results in Fig. 10 are reproduced. Parameters are the same as in Fig. 15, except inputs are strictly Poisson and synapses are deterministic. When $u > 0$, a spike in one neuron adds a PSC to the AMPA conductance of the second. The peak value of the EPSC is given by $u \cdot \bar{g}_{AMPA}/30$ so that the corresponding PSP amplitude is roughly a proportion u of the distance from rest to threshold. When $u < 0$ spikes in one neuron add a PSC with peak value $u \cdot \bar{g}_{GABA}/60$ to the GABA conductance of the other, to obtain a similar scaling.

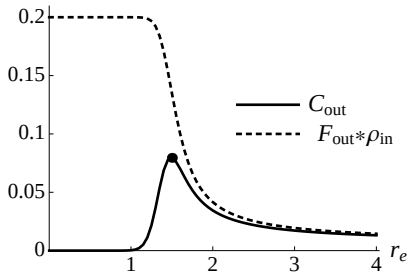

Figure 17: **An alternate measure of correlation.** The covariation factor (solid line) and $F_{\text{out}}\rho_{\text{in}}$ (dashed line) as a function of r_{e} for the dLIF with $r_{\text{i}} = 1$, $\bar{I}_L = 0.5$, $\rho_{\text{ee}} = \rho_{\text{ii}} = 0.2$ and $\rho_{\text{ei}} = 0$. The filled circle indicates the boundary between the drift and fluctuation dominated regimes, $r_{\text{e}} = r_{\text{i}} + \bar{I}_L$. The covariation factor is nearly zero for small values of r_{e}. As r_{e} grows, the cells become less forgetful and C_{out} increases initially. In the drift dominated regime, C_{out} decreases towards $F_{\text{out}}\,\rho_{\text{in}}$ since $\rho_{\text{out}} \nearrow \rho_{\text{in}}$ (see Sec. 3) and $\rho_{\text{out}} = C_{\text{out}}/F_{\text{out}}$.

et al., 1994) correlations between cells. We showed that correlations are also strongly modulated by dynamics at the cellular level. The correlation structure at the level of networks is shaped by the interplay between such local and global effects.

Analytical approximations of the correlation between the outputs of two current or conductance based LIF neurons in the diffusive limit have been obtained previously (de la Rocha et al., 2007; Shea-Brown et al., 2008; Tchumatchenko et al., 2008; Ostojić et al., 2009). Since integrate-and-fire models are only caricatures of actual neurons, it is useful to complement such analytical approaches with a mechanistic understanding. We characterized the mechanisms that shape correlation transfer in an intuitive way, providing insights into how correlations are affected by various aspects of neural dynamics. Moreover, the use of point processes to model inputs allowed us to address questions that are more difficult to formulate for diffusive models, and helped maintain a more direct connection to physiology.

9.1 Non-stationary inputs

Throughout the text, we have assumed that inputs are stationary. Although this assumption is frequently made in theoretical studies (Moreno-Bote and Parga, 2006; de la Rocha et al., 2007; Shea-Brown et al., 2008; Ostojić et al., 2009), neurons *in vivo* receive inputs with time-dependent statistics. The assumption of stationarity is a good approximation when the input statistics change more slowly than the timescale of correlations and synaptic responses.

The dLIF model can be extended to take time dependent rates and correlations into account whilst maintaining its numerical tractability. The master equation for the membrane potentials is transformed from an linear autonomous system of ODEs (see Appendix D), to a linear non-autonomous system, $p'(t) = A(t)p(t)$ where $A(t)$ is the time-dependent infinitesimal generator matrix (Karlin and Taylor, 1975; Gardiner, 1985). The methods in the Appendix can then be extended to investigate time-dependent spiking statistics.

9.2 Alternate measures of correlation

There is no unique way to quantify dependencies between pairs of spike trains. We chose to use the Pearson correlation coefficient because it is a unitless quantity that is widely used and understood. However, the random walk models we presented are mathematically tractable and our analysis can be applied to other measures of statistical dependence.

For example, measures of correlation where the covariance is normalized by the firing rates have been proposed and may offer information theoretic advantages (Amari, 2009; Roudi et al., 2009). As an example, we consider the unitless *covariation factor*, $C_{\text{out}} = \gamma_{\text{out}}/r_{\text{out}}$. This quantity is an extension of the Fano factor that measures the dispersion of a bivariate distribution.

To analyze the covariation factor, reorganize Eq. (4) as

$$F_{\text{out}} = \text{CV}^2_{\text{out}}, \qquad \text{and} \qquad C_{\text{out}} = (F_{\text{out}} + 1)\left(\frac{E[\tau_1] - E[\tau_1 \,|\, V_2 \nearrow \theta]}{E[\tau_1]}\right) + S_{\text{out}}.$$

The behavior of C_{out} now parallels that of ρ_{out}: In the fluctuation dominated limit, when $r_{\text{e}} \ll r_{\text{i}}+\bar{I}_L$, the effect of a spike in one neuron is forgotten by the time the second spikes, so that $C_{\text{out}} \approx 0$. As r_{e} increases towards the drift dominated regime, the cells become less "forgetful" and $|C_{\text{out}}|$ increases. As r_{e} increases into the drift dominated regime, the cells behave like PIFs, transferring spike counts linearly and preserving correlations, so that $C_{\text{out}} = \rho_{\text{out}}F_{\text{out}} \approx \rho_{\text{in}}F_{\text{out}}$ (See Fig. 17B).

The "forgetfulness" of cells diminishes the dependence between the output of the cells in the fluctuation dominated regime. The effect of a spike in one neuron is forgotten before the second neuron spikes, and the output spike trains are nearly independent as a result. This is a fundamental property of excitable systems, and not due to the particular choice of the Pearson correlation coefficient or the neuron model employed.

9.3 Higher-order correlations

Pairwise correlations play a significant role in the neural code, and it has been proposed that the first and second order statistics may fully characterize the response of a population (Schneidman et al., 2006; Shlens et al., 2006; Tang et al., 2008; Shlens et al., 2009, although see (Roudi et al., 2009)). However, the higher-order structure of the population response can have significant effects on the firing of downstream neurons (Kuhn et al., 2003), and the information carried by the response Roudi et al. (2009).

Eq. (8) can be used to show that a pair of PIFs preserve higher order correlations, and we therefore expect that a pair of leaky neurons in the drift dominated regime approximately preserve higher order correlations. In the fluctuation dominated limit, the forgetfulness of cells causes spiking to become independent and therefore higher order correlations are reduced. The analysis of synaptic noise and coupling can also be extended to higher order moments.

9.4 Physiologically realistic models

We used random walk models in our analysis and verified our results with simulations of a conductance based integrate–and–fire model. This approach is common in studies of stochastic response

properties of neurons (Salinas and Sejnowski, 2000; Rauch et al., 2003) and captures the fundamental mechanisms of a physiological cell. However, more detailed models of active conductances, synaptic plasticity, channel dynamics, and an extended dendritic morphology might reveal additional mechanisms that modulate correlations. Such models are outside of the scope of this study, but warrant further investigation. For instance, preliminary results suggest that correlations are reduced significantly in a Hodgkin Huxley model (E. Shea-Brown, private communication, 2010).

Acknowledgements

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A Generating correlated inputs

To generate a pair of correlated Poisson processes, we used an algorithm equivalent to the SIP model in Kuhn et al. (2003). We first generated three independent Poisson processes, a_1, a_2, and b, then defined $p_1 = a_1 + b$ and $p_2 = a_2 + b$. The processes p_1 and p_2 are correlated Poisson processes with $r_{p_j} = r_{a_j} + r_b$ and $\rho_{p_1 p_2} = r_b/\sqrt{r_{a_1} r_{a_2}}$. The cross-covariance function between these processes is a delta function with area $\gamma_{p_1 p_2} = \rho_{p_1 p_2}\sqrt{r_{p_1} r_{p_2}}$. Any pair of Poisson processes with a delta function cross-covariance is statistically equivalent to processes generated by the algorithm above. This algorithm is easily generalized to four Poisson processes with specified pairwise correlations. In generating such quadruplets, we constrain the processes so that the probability of more than two spikes occurring simultaneously is zero as $dt \to 0$. This algorithm generates delta-correlated Poisson processes. To generate pairs of Poisson processes with temporally extended correlations (for the gray lines in Fig. 11), we added an independent random number to each spike time in one of the excitatory and one of the inhibitory trains. The resulting processes are Poisson with a cross-covariance function given by the density of the random numbers used (Cox and Isham, 1980).

The simulations in Fig. 8 and 15, required correlated processes with CV $\neq 1$. For these simulations we generated pairs of correlated renewal processes with gamma distributed interspike intervals: Begin with a pair of delta correlated Poisson processes, p_1 and p_2, generated using the algorithm above. Let $n_j \in \mathbb{N}$ be positive integers, and let g_j be the spike train consisting of every n_j-th spike in p_j for $j = 1, 2$. Then g_j is a renewal processes with rate $r_{g_j} = r_{p_j}/n_j$, Fano factor $\mathrm{CV}^2 = F_{g_j} = 1/n_j$ and correlation coefficient $\rho_{g_1 g_2} = \rho_{p_1 p_2}$. The interspike intervals follow a gamma distribution with rate parameter r_{g_j} and shape parameter n_j. While the auto-correlations of such processes are oscillatory, the cross-covariance function between g_1 and g_2, is a delta function.

B Correlation Transfer for the PIF Model

We next derive the total output correlation for a pair of PIFs driven by correlated stationary inputs. In the main text, we assume that the input signals can be written as $\mathrm{in}_j(t) = \mathrm{e}_j(t) - \mathrm{i}_j(t)$ where

$e_j(t)$ and $i_j(t)$ are point processes. Here we derive the output statistics for a pair of PIFs driven by general stationary processes, $\text{in}_1(t)$ and $\text{in}_2(t)$, under weak assumptions.

We generalize the input pike count by defining $N_{\text{in}_j}(t) = \int_0^t \text{in}_j(s)ds$ for $j = 1, 2$. Note that $N_{\text{in}_j}(t)$ is not necessarily integer valued since $\text{in}_j(t)$ is not necessarily a point process. For the PIFs to have non-zero firing rates, we must assume that the inputs have positive mean, $\mu_{\text{in}_j} = E[\text{in}_j(t)] > 0$. The asymptotic variances, covariance and correlation are defined as, $\sigma_{\text{in}_j}(t) = \lim_{t\to\infty} \text{var}(N_{\text{in}_j}(t))/t$, $\gamma_{\text{in}} = \lim_{t\to\infty} \text{cov}(N_{\text{in}_1}(t), N_{\text{in}_2}(t))/t$, and $\rho_{\text{in}} = \gamma_{\text{in}}/(\sigma_{\text{in}_1}\sigma_{\text{in}_2})$. These quantities can also be interpreted in terms of the areas of auto- and cross-covariance functions as in the Methods. The membrane potentials $V_1(t)$ and $V_2(t)$ of two PIFs driven by input signals $\text{in}_1(t)$ and $\text{in}_2(t)$ obey the stochastic equations

$$dV_1 = \text{in}_1(t)dt$$
$$dV_2 = \text{in}_2(t)dt$$

with the added condition that when V_j reaches θ_j, it is reset to $V_j = 0$ and an output spike is produced. These stochastic equations can be interpreted unambiguously in the Itô sense whenever $(N_{\text{in}_1}(t), N_{\text{in}_2}(t))$ is a bivariate semimartingale (Métivier, 1982). However, any interpretation which yields Eq. (13) is sufficient. The output spike trains have rate, variance, covariance, and correlation r_{out_j}, $\sigma^2_{\text{out}_j}$, γ_{out}, and ρ_{out} as defined in the Methods. We make the following ergodicity assumptions,

1. σ_{in}, γ_{in}, σ_{out_j}, and γ_{out} are finite and σ_{in_j},$\sigma_{\text{out}_j} > 0$; and
2. $\vec{V}(t) = (V_1(t), V_2(t))$ is ergodic and its stationary distribution has finite, positive variances.

These assumptions are necessary for the outputs to be stationary and for the asymptotic input and output correlations to exist. They are true for typical processes used to model stochastic inputs, but can be violated for periodic processes or processes obtained from deterministically periodic driving forces. We assume that $\vec{V}(0)$ is drawn from the stationary distribution of $\vec{V}(t)$ so that the process is stationary. We will first derive the output statistics for a pair of uncoupled PIFs driven by stationary inputs, then separately consider the case of variable synaptic responses and reciprocal coupling in Appendix E.

The output spike counts are given by

$$N_{\text{out}_1}(t) = \frac{N_{\text{in}_1}(t) + V_1(0) - V_1(t)}{\theta}, \qquad \text{and} \qquad N_{\text{out}_2}(t) = \frac{N_{\text{in}_2}(t) + V_2(0) - V_2(t)}{\theta} \tag{13}$$

Before deriving the output spiking statistics, we must prove a simple lemma. The notation $f(t) \sim o(t)$ below is shorthand for $\lim_{t\to\infty} f(t)/t = 0$.

Lemma 1. *Suppose X_t and C_t are stochastic processes such that $\lim_{t\to\infty} \text{var}(X_t)/t = c$ for some finite positive number c and $\text{var}(C_t) \sim o(t)$. Then $\text{cov}(X_t, C_t) \sim o(t)$.*

Proof. By the Cauchy-Schwarz inequality,

$$\lim_{t\to\infty} \frac{|\text{cov}(X_t, C_t)|}{t} \leq \lim_{t\to\infty} \frac{\sqrt{\text{var}(X_t)\text{var}(C_t)}}{t} = \lim_{t\to\infty} \sqrt{\frac{\text{var}(X_t)}{t}}\sqrt{\frac{\text{var}(C_t)}{t}} = 0.$$

□

We now derive the output spiking statistics

Theorem 1. *Under assumptions 1 and 2 above, the output spike count variance and covariance for a pair of PIFs driven by correlated stationary inputs,* $\mathrm{in}_1(t)$ *and* $\mathrm{in}_2(t)$*, are given by*

$$\sigma^2_{\mathrm{out}_j} = \frac{\sigma_{\mathrm{in}_j}}{\theta_j^2}, \qquad \textit{and} \qquad \gamma_{\mathrm{out}} = \frac{\gamma_{\mathrm{in}}}{\theta_1\theta_2}.$$

Thus the input correlation coefficient is preserved, $\rho_{\mathrm{out}} = \rho_{\mathrm{in}}$.

Proof. First note that $\mathrm{var}(V_j(t)) \sim o(t)$ by assumption 2 above and that $\lim_{t\to\infty} \mathrm{var}(N_{\mathrm{in}_j}(t))/t = \sigma^2_{\mathrm{in}_j}$ is finite and positive by assumption 1. From Eq. (13) and using the bilinearity of covariances we can calculate

$$\begin{aligned}
\gamma_{\mathrm{out}} &= \lim_{t\to\infty} \frac{1}{t}\mathrm{cov}\left(N_{\mathrm{out}_1}(t), N_{\mathrm{out}_2}(t)\right) \\
&= \lim_{t\to\infty} \frac{1}{t}\mathrm{cov}\left(\frac{N_{\mathrm{in}_1}(t) + V_1(0) - V_1(t)}{\theta_1}, \frac{N_{\mathrm{in}_2}(t) + V_2(0) - V_2(t)}{\theta_2}\right) \\
&= \frac{1}{\theta_1\theta_2} \lim_{t\to\infty} \frac{1}{t}\left[\mathrm{cov}\left(N_{\mathrm{in}_1}(t), N_{\mathrm{in}_2}(t)\right) + o(t)\right] \qquad (14) \\
&= \frac{\gamma_{\mathrm{in}}}{\theta_1\theta_2}
\end{aligned}$$

where (14) follows from Lemma 1 and the bilinearity of covariances. Using an identical argument we can derive $\sigma^2_{\mathrm{out}_j} = \frac{\sigma^2_{\mathrm{in}_j}}{\theta_j^2}$. It follows that $\rho_{\mathrm{out}} = \gamma_{\mathrm{out}}/\left(\sigma_{\mathrm{out}_1}\sigma_{\mathrm{out}_2}\right) = \rho_{\mathrm{in}}$. □

C Derivation of Eq. (4)

In this section, we derive Eq. (4) which gives the asymptotic correlation between the outputs of two integrate-and-fire neurons. The equation holds for any integrate-and-fire models for which the membrane potentials are Markov processes (marginally and jointly) and additionally satisfy

$$\Pr(V_j(t_3) \in A \,|\, V_j(t_2) = v_2,\, V_i(t_1) = v_1) = \Pr(V_j(t_3) \in A \,|\, V_j(t_2) = v_2) \qquad (15)$$

for $i, j \in \{1, 2\}$, any set A and any voltages v_1 and v_2, whenever $t_1 < t_2 < t_3$.

These conditions are met by a pair integrate-and-fire neurons whose sub-threshold membrane potentials are governed by equations of the form

$$\begin{aligned}
dV_1 &= f_1(V_1, \mathrm{in}_1)dt \\
dV_2 &= f_2(V_2, \mathrm{in}_2)dt
\end{aligned}$$

where $\mathrm{in}_1(t)$ and $\mathrm{in}_2(t)$ are stationary stochastic processes such that $\mathrm{in}_i(t)$ is independent of $\mathrm{in}_j(s)$ for $s \neq t$ and $i, j \in \{1, 2\}$. Here, we assume standard threshold and reset conditions at θ_j and 0 respectively (see Methods). In short, the two input processes must be delta-correlated (e.g., correlated Poisson processes, white noise, or any linear combination thereof) and the neurons must be uncoupled. For example, the conditions are met for the PIF, LIF and dLIF models considered

in the text with Poisson or white noise inputs, even in the case of random synaptic amplitudes. The conditions are not strictly met for the conductance-based model due to its non-instantaneous synapses. However, the results obtained here are approximately valid when the input is correlated in time or when synapses are not instantaneous, as long as the firing rates are significantly slower than both the synaptic time constants and the correlation time constants of the inputs.

Since the membrane potentials are marginally Markov, the output spike trains $a_1(t)$ and $a_2(t)$ are renewal processes. We assume that the bivariate membrane potential process $(V_1(t), V_2(t))$ is ergodic and its initial condition is drawn from its stationary distribution so that the output spike trains are stationary in a bivariate sense (Cox and Lewis, 1972). For ease of notation, we write $N_j(t)$ in place of $N_{a_j}(t)$ for the counting processes and similarly for other quantities.

For $t > 0$, define $Q_{ij}(t)$ to be the distribution of the waiting time until the first spike in $a_i(t)$ after a spike in $a_j(t)$,

$$Q_{ij}(t) = \lim_{\delta \to 0} \Pr\left(a_i \text{ spikes in } [t, t+\delta], \text{ but not in [0,t]} \mid a_j \text{ spikes in } [0, \delta] \right) / \delta.$$

The auto- and cross-covariance functions are related to the asymptotic spike count statistics by (Cox and Lewis, 1972),

$$\sigma_j^2 = 2 \int_{0^+}^{\infty} R_{jj}(t) dt + r_j, \quad j = 1, 2$$

and

$$\gamma_{12} = \int_{0^+}^{\infty} R_{12}(t) dt + \int_{0^+}^{\infty} R_{21}(t) dt + r_s. \tag{16}$$

where the $+$ on the lower limit of the integrals indicates that any delta function at the origin is omitted and r_s is the rate of synchronous spikes, which accounts for the area of the omitted delta function at the origin. Similarly, r_j accounts for the area of the delta function in $R_{jj}(t)$.

Due to the renewal properties of the outputs, we have that (Cox, 1962)

$$\begin{aligned} H_{jj}(t) &:= \lim_{\delta \to 0} \frac{1}{\delta} \sum_{k=1}^{\infty} \Pr\left(a_j \text{ spikes for the } k\text{th time in } [t, t+\delta] \,\middle|\, a_j \text{ spiked in } [0, \delta] \right) \\ &= \sum_{k=1}^{\infty} Q_{jj}^{(k)}(t), \quad t > 0,\ j = 1, 2 \end{aligned}$$

where $Q_{jj}^{(k)}$ is the k-fold convolution of Q_{jj} with itself. Similarly, due to the renewal properties of the outputs in addition to assumption (15), we have for $t > 0$,

$$\begin{aligned} H_{12}(t) &:= \lim_{\delta \to 0} \frac{1}{\delta} \sum_{k=1}^{\infty} \Pr\left(a_1 \text{ spikes for the } k\text{th time in } [t, t+\delta] \,\middle|\, a_2 \text{ spiked in } [0, \delta] \right) \\ &= Q_{12}(t) + \sum_{k=1}^{\infty} \left(Q_{12} * Q_{11}^{(k)} \right)(t) \\ &= Q_{12}(t) + (Q_{12} * H_{11})(t) \end{aligned} \tag{17}$$

where $*$ denotes convolution. Similarly, $H_{21}(t) = Q_{21}(t) + (Q_{21} * H_{22})(t)$ for $t > 0$.

We proceed by considering the Laplace transform of the cross-covariance functions. The Laplace transform of a function $f(t)$ is given by $\hat{f}(u) = \int_{0^+}^{\infty} e^{-ut} f(t)dt$. Using elementary properties of the Laplace transform, Eq. (17) can be rewritten as $\hat{H}_{12}(u) = \hat{Q}_{12}(u) + \hat{Q}_{12}(u)\hat{H}_{11}(u)$. Now, using the definition of $R_{12}(t)$ we can write

$$\begin{aligned}
\hat{R}_{12}(u) &= r_2 \left(\hat{H}_{12}(u) - \frac{r_1}{u} \right) \\
&= r_2 \left(\hat{Q}_{12}(u) + \hat{Q}_{12}(u)\hat{H}_{11}(u) - \frac{r_1}{u} \right) \\
&= r_2 \left(\hat{Q}_{12}(u) + \frac{1}{r_1}\hat{Q}_{12}(u)\hat{R}_{11}(u) + r_1 \left(\frac{\hat{Q}_{12}(u) - 1}{u} \right) \right)
\end{aligned}$$

where the last step follows from the fact that $\hat{H}_{11}(u) = \hat{R}_{11}(u)/r_1 + r_1/u$. From this, we can calculate the area of the cross-covariance function,

$$\begin{aligned}
\int_{0^+}^{\infty} R_{12}(t)dt &= \lim_{u \to 0} \hat{R}_{12}(u) \\
&= r_2 \left(1 + \frac{\sigma_1^2 - r_1}{2r_1} + r_1 \hat{Q}'_{12}(0) \right) \\
&= r_2 \left(\frac{\mathrm{CV}_1^2 + 1}{2} - r_1 E[\tau_1 \,|\, V_2 \nearrow \theta_2] \right) \qquad (18)
\end{aligned}$$

where where $F_1 = \mathrm{CV}_1^2 = \sigma_1^2/r_1$ is the output Fano factor and $E[\tau_1 \,|\, V_2 \nearrow \theta_2] = -\hat{Q}'_{12}(0)$ is the expected time until the first spike in a_1 after a spike in a_2 (Feller, 1991). In the derivation above, we used the facts that $\lim_{u \to 0} \hat{Q}_{12}(u) = 1$ and $\lim_{u \to 0} \hat{R}_{11}(u) = (\sigma_1^2 - r_1)/2$.

By an identical argument, we get an analogous expression for $\int_{0^+}^{\infty} R_{21}(t)dt$. From these expressions and Eq. 16, we can write the output correlation, $\rho_{\mathrm{out}} = \gamma_{12}/(\sigma_1\sigma_2)$, as

$$\rho_{\mathrm{out}} = \frac{\sqrt{r_1 r_2}\,(E[\tau_1] - E[\tau_1 \,|\, V_2 \nearrow \theta_2] + E[\tau_2] - E[\tau_2 \,|\, V_1 \nearrow \theta_1]) + S_{12}}{\mathrm{CV}_1 \mathrm{CV}_2} \qquad (19)$$

where $E[\tau_1] = (\mathrm{CV}_1^2 + 1)/(2r_1)$ is the expected recurrence time (Cox, 1962), which is the expected time until the next spike in a_1 starting from an arbitrary time (i.e., with V_1 starting from its stationary distribution) and similarly for $E[\tau_2]$.

To obtain the form given in Eq. (4), we note that Eq. (18) can be written as

$$\int_{0^+}^{\infty} R_{12}(t)dt = \frac{r_2}{2}(\mathrm{CV}_1^2 + 1) \left(\frac{E[\tau_1] - E[\tau_1 \,|\, V_2 \nearrow \theta_2]}{E[\tau_1]} \right).$$

In the symmetric case, combining this with Eq. (16) gives Eq. (4).

Eq. (19) can be used to calculate the correlation between simulated data when the model satisfies the Markov assumptions made above. To apply the equation to data, we only need to obtain estimates for r_j, CV_j, $E[\tau_1 \,|\, V_2 \nearrow \theta_2]$, $E[\tau_2 \,|\, V_1 \nearrow \theta_1]$, and S_{12}. The rates and ISI CV's can easily be estimated from a sample of the univariate interspike intervals, $r_j = 1/E[ISI_j]$ and $\mathrm{CV}_j^2 = \mathrm{var}(ISI_j)/E[ISI_j]^2$. The synchrony is easily estimated by counting the occurrence of synchronous

spikes. To estimate $E[\tau_1 \,|\, V_2 \nearrow \theta_2]$, one only needs to calculate the average time between a spike in a_2 and the next spike in a_1, and similarly for $E[\tau_2 \,|\, V_1 \nearrow \theta_1]$.

We used Eq. (19) to obtain the estimates of ρ_{out} for the LIF model in Figs. 4 and 8. We found several advantages to using this method versus conventional methods, such as computing the cross-covariance functions or counting spikes over sliding windows. When calculating the correlation from the integrals of the cross-covariance functions, one must bin time and also choose a large window size over which to integrate. Similarly, when using the sample covariance of spike counts between sliding windows, one must choose a large window size for the sliding window. The quantities in Eq. (19) can all be estimated by looking at the time intervals between spikes. It is therefore not necessary to bin time or to fix a large window size over which to calculate the correlation. The algorithm based on (19) appears to be faster than algorithms using the other two methods. Though a deeper investigation is necessary, it also appears that the estimator is more accurate.

Next, we show how Eq. (19) can be used to calculate the exact correlation coefficient for the dLIF model.

D Analysis of the dLIF model

The methods used to analyze the PIF model cannot be applied to the dLIF model since there is no analogue to Eq. (8) due to the lower reflecting barrier at β. However, when the inputs are correlated Poisson processes as defined in Appendix A, we can use the theory of continuous time Markov chains in combination with Eq. (19) to derive the output spiking statistics. A suite of Matlab programs that implement the methods described below can be found at `http://www.mathworks.com/matlabcentral/fileexchange/28686`.

For notational simplicity in the analysis below, we consider only the case where $\bar{I}_L = 0$, i.e. there is no leak term. However, note that the leak current in this model is equivalent to an uncorrelated inhibitory input current. To recover the results for $\bar{I}_L > 0$ from the results below, simply make the substitutions $r_{\text{i}_j} \to r_{\text{i}_j} + \bar{I}_{L_j}$, $\rho_{\text{ii}} \to \rho_{\text{ii}}/(1 + \bar{I}_L/r_{\text{i}})$, and $\rho_{\text{ei}} \to \rho_{\text{ei}}/\sqrt{1 + \bar{I}_L/r_{\text{i}}}$.

We first derive the univariate statistics for a single dLIF driven by Poisson inputs (see Methods). The membrane potential $V(t)$ for this model is a continuous time Markov random walk and the output spike trains are renewal processes. The Laplace transform of the first passage time densities for reflected random walks are obtained by Khantha and Balakrishnan (1983). The moments of the first passage times can be found from the derivatives of these Laplace transforms (Feller, 1991). For the mean first passage time of $V(t)$ over θ starting from $k \in \{\beta, \beta+1, \ldots, \theta-1\}$,

$$\mu_{k\to\theta} = \frac{q\left(-q^{\beta-k} - kq + k + q^{\beta-\theta} + q\theta - \theta\right)}{(q-1)^2\, r_{\text{e}}}.$$

To derive this expression, we needed to correct an error in Khantha and Balakrishnan (1983) in going from their Eq. (6) to Eq. (7): the inner expression in their Eq. (7) should read $(m - m_0) + (f^{-m} - f^{-m_0})/(f-1)$ instead of $(m-m_0) - (f^{-m} - f^{-m_0})/(f-1)$. The variance of the first passage

time from reset to threshold is given by

$$\sigma^2_{0\to\theta} = \frac{q^2\left(-4(\beta(q-1)-q(\theta+1)+\theta)q^{\beta-\theta}+q^{2(\beta-\theta)}-q^{2\beta}+4(\beta(q-1)-q)q^{\beta}+(q^2-1)\,\theta\right)}{(q-1)^4\,r_{\mathrm{e}}^2}.$$

The output firing rate is then given by $r_{\mathrm{out}} = 1/\mu_{0\to\theta}$, the asymptotic spike count variance is $\sigma^2_{\mathrm{out}} = \sigma^2_{0\to\theta}/\mu^3_{0\to\theta}$ and the output Fano factor is $F_{\mathrm{out}} = \mathrm{CV}^2_{\mathrm{out}} = \sigma^2_{0\to\theta}/\mu^2_{0\to\theta} = \sigma^2_{\mathrm{out}}/r_{\mathrm{out}}$ (Cox, 1962). Simplified expressions for r_{out} and $\mathrm{CV}_{\mathrm{out}}$ are given in the Methods.

Other univariate statistics can be found from the infinitesimal generator matrix of the membrane potential process, $V(t)$ (Karlin and Taylor, 1975). The off-diagonal terms ($i \neq j$) of this matrix are given by

$$\mathcal{B}_{ij} := \lim_{h\to 0}\frac{1}{h}\Pr(V(t+h)=j\,|\,V(t)=i) = \begin{cases} r_{\mathrm{e}} & j=i+1 \\ r_{\mathrm{i}} & j=i-1 \\ r_{\mathrm{e}} & i=\theta-1,\, j=0 \end{cases} \tag{20}$$

The diagonal terms are then chosen so that each row sums to zero: $\mathcal{B}_{ii} = -\sum_{j\neq i}\mathcal{B}_{ij}$. The distribution of $V(t)$ is then given by $P(t) = P(0)e^{\mathcal{B}t}$ where $P(t)$ is a time dependent vector with $P_j(t) = \Pr(V(t)=j)$ and $P(0)$ is the initial distribution. The stationary distribution $p(j) = \lim_{t\to\infty} P_j(t)$ is then given by the left eigenvector corresponding to the dominant left eigenvalue, $\lambda_0 = 0$. The remaining eigenvalues have negative real part. The non-zero eigenvalue with maximal real part, λ_1, determines the timescale with which $P_j(t) \to p(j)$. In particular, $|P_j(t) - p(j)| \sim e^{-t/\tau_{\mathrm{mem}}}$ where $\tau_{\mathrm{mem}} = -1/\mathrm{Real}(\lambda_1)$.

The univariate stationary distribution, $p(j)$, can be found by deriving the dominant left eigenvector as discussed above, which is equivalent to solving the detailed balance equation $p = pe^{\mathcal{B}}$. This is a linear recurrence equation and can be solved using a computer algebra system or by hand using the method of generating functions. We obtained the solution

$$p(k) = \lim_{t\to\infty}\Pr(V(t)=k) = \frac{q-1}{q^{\beta}-q^{\theta}(q^{\beta}+\theta-q\theta)} \times \begin{cases} (q^{\theta+k}-q^k) & \beta \leq k \leq 0 \\ (q^{\theta}-q^k) & 0 < k < \theta \end{cases}. \tag{21}$$

The output rate, derived from the first passage properties above, is also given by the probability flux across threshold: $r_{\mathrm{out}} = r_{\mathrm{e}}p(\theta-1)$.

We now consider the case of two dLIF neurons driven by correlated Poisson inputs (see Methods). We will use Eq. (19) to calculate the correlation coefficient, ρ_{out}, between the output spike trains. First we must calculate the stationary distribution of the bivariate process, $(V_1(t), V_2(t))$. We first enumerate the state space, $\theta_1 \times \theta_2$ into a single vector of length $\theta_1\theta_2$ and calculate the infinitesimal generator matrix, in a similar fashion to the univariate case described above. Complicated boundary conditions make it impractical to include the full bivariate generator matrix in the text. The commented Matlab code that accompanies this paper can be used to generate this matrix.

The bivariate stationary distribution, $p(k_1, k_2)$, is the the basis vector for the one-dimensional nullspace of the transpose of $\mathcal{A}$, equivalently it is the left eigenvector of $\mathcal{A}$ associated with the left eigenvalue $\lambda = 0$. There are a variety of numerical techniques for finding this vector. Note that the vector must be normalized so that its elements sum to 1 since $p(k_1, k_2)$ is a probability distribution.

A synchronous output spike occurs whenever both cells are just below threshold and receive a synchronous excitatory input. The rate of synchronous outputs spike is therefore given by $r_s = p(\theta_1 - 1, \theta_2 - 1)\rho_{\mathrm{ee}}\sqrt{r_{\mathrm{e}_1} r_{\mathrm{e}_2}}$. The output synchrony is then $S_{12} = r_s/\sqrt{r_{\mathrm{out}_1} r_{\mathrm{out}_2}}$.

Since the univariate statistics are known in closed form (see above), the only quantities from Eq. (19) left to calculate are $E[\tau_1 \,|\, V_2 \nearrow \theta_2]$ and $E[\tau_2 \,|\, V_1 \nearrow \theta_1]$. First define the conditional distribution,

$$p_1(k_1 \,|\, V_2 = \theta_2 - 1) = \frac{p(k_1, \theta_2 - 1)}{p_2(\theta_2 - 1)}$$

where $p_2(\theta_2 - 1)$ is the value of univariate stationary distribution for $V_2(t)$ at $\theta_2 - 1$ from Eq. 21. From here, we need to calculate the conditional distribution $p_1(k_1 \,|\, V_2 \nearrow \theta_2)$ which represents the distribution of V_1 given that V_2 has just crossed threshold. This can be calculated by evolving a proportion $\rho_{\mathrm{ee}}\sqrt{r_{\mathrm{e}_1}/r_{\mathrm{e}_2}}$ of the probability mass in $p(k_1 \,|\, V_2 = \theta_2 - 1)$ by one excitatory spike and evolving a separate proportion $\rho_{\mathrm{i}_1\mathrm{e}_2}\sqrt{r_{\mathrm{i}_1}/r_{\mathrm{e}_2}}$ by one inhibitory spike (see the linked Matlab code). We can then use the mean first passage times derived above to calculate

$$E[\tau_1 \,|\, V_2 \nearrow \theta_2] = \sum_{k=\beta_1}^{\theta_1} p(k \,|\, V_2 \nearrow \theta_2)\, \mu_{k \to \theta_1}.$$

An identical method is used to calculate $E[\tau_2 \,|\, V_1 \nearrow \theta_1]$. Now, ρ_{out} can be calculated from Eq. (19).

We now describe how to calculate time dependent statistics for Figs. 5 and Fig. 17A. Let $\mathcal{B}_1$ be the infinitesimal generator matrix of the marginal process $V_1(t)$ (see Eqn. (20) above). Given an initial distribution, $V_1(0)$, the distribution of $V_1(t)$ is given by $e^{\mathcal{B}_1 t}V_1(0)$. The conditional distribution of $V_1(t)$ after a spike in V_2 is then given by $\Pr(V_1(t) = k \,|\, V_2(0) \nearrow \theta_2) = \left[e^{\mathcal{B}_1 t}V_1(0)\right]_k$ where $[\cdot]_k$ denotes the kth component and the initial distribution is the conditional distribution described above, $[V_1(0)]_k = p_1(k \,|\, V_2 \nearrow \theta_2)$.

The time dependent conditional mean and standard deviation for the top row of Fig. 5 can be calculated directly from $p_1(k_1 \,|\, V_2 \nearrow \theta_2)$. The instantaneous firing rate, given an initial distribution $V_1(0)$ is given by the flux across threshold, $\nu(t \,|\, V_1(0)) = r_{\mathrm{e}} \left[e^{\mathcal{B}_1 t}V_1(0)\right]_{\theta_1 - 1}$. The conditional intensity function, $H_{12}(t)$, is then given by $\nu(t \,|\, V_1(0))$ with $[V_1(0)]_k = p_1(k \,|\, V_2 \nearrow \theta_2)$. The auto conditional intensity function, $H_{11}(t)$ is given by $\nu(t \,|\, V_1(0))$ with $[V_1(0)]_k = \delta_{0,k}$. The functions $H_{21}(t)$ and $H_{22}(t)$ are derived analogously. The auto- and cross-covariance functions are then given by $R_{ij}(t) = r_j(H_{ij}(t) - r_i)$.

For the bottom row of Fig. 5, we needed to calculate the cumulative distribution of the first passage time of V_1 over θ_1 given an initial distribution. This can be achieved by adding an absorbing state at threshold, θ_2, to the marginal infinitesimal generator, $\mathcal{B}_1$. Then the cumulative distribution of the waiting time until the next spike is simply given by the amount of mass in the absorbing state at time t. That is, $\Pr(\tau_1 \leq t) = \left[e^{\mathcal{B}_1 t}V_1(0)\right]_{\theta_1}$ where $\mathcal{B}_1$ has an absorbing state at θ_1 and $V_1(0)$ is the appropriate initial distribution. The conditional distribution (solid line in the bottom row of Fig. 5) is found by setting $[V_1(0)]_k = p_1(k \,|\, V_2 \nearrow \theta_2)$ as above. The stationary case (dashed line) is found by setting $[V_1(0)]_k = p_1(k)$ where $p_1(k)$ is the marginal stationary distribution from Eq. 21.

E Generalizations of the PIF model

In this section, we generalize Theorem 1 to take synaptic variability and coupling into account in the PIF model, thereby obtaining the main equations in Secs. 5 and 6.

E.1 The PIF with random post-synapitc responses

Suppose that the ith excitatory input to neuron j increments $V_j(t)$ by a random amount $d^i_{\mathrm{e}_j}$ and that the ith inhibitory input to neuron j decrements $V_j(t)$ by a random amount $d^i_{\mathrm{i}_j}$. Each $d^i_{\mathrm{e}_j}$ ($d^i_{\mathrm{i}_j}$) is drawn independently from a distribution with mean $\overline{d}_{\mathrm{e}_j}$ ($\overline{d}_{\mathrm{i}_j}$) and variance $\sigma^2_{d_{\mathrm{e}_j}}$ ($\sigma^2_{d_{\mathrm{i}_j}}$) for $j = 1, 2$. To guarantee positive firing rates $r_{\mathrm{out}_j} = (r_{\mathrm{e}_j}\overline{d}_{\mathrm{e}_j} - r_{\mathrm{i}_j}\overline{d}_{\mathrm{i}_j})/\theta_j > 0$ we assume $\mu_{Y_j} = r_{\mathrm{e}_j}\overline{d}_{\mathrm{e}_j} - r_{\mathrm{i}_j}\overline{d}_{\mathrm{i}_j} > 0$. This model equivalent to a PIF with inputs

$$Y_j(t) = \sum_{t_i \in \Gamma_{\mathrm{e}_j}} d^i_{\mathrm{e}_j}\delta(t - t_i) - \sum_{t_i \in \Gamma_{\mathrm{i}_j}} d^i_{\mathrm{i}_j}\delta(t - t_i), \quad j = 1, 2.$$

Thus by Theorem 1, $\sigma^2_{\mathrm{out}_1} = \frac{\sigma^2_{Y_j}}{\theta_j^2}$, $\gamma_{\mathrm{out}} = \frac{\gamma_{Y_1Y_2}}{\theta_1\theta_2}$, and $\rho_{\mathrm{out}} = \rho_{Y_1Y_2}$.

The accumulated effective input process $N_{Y_j}(t) = \int_0^t Y_j(s)ds$ can be written as

$$N_{Y_j}(t) = \sum_{i=1}^{N_{\mathrm{e}_j}(t)} d^i_{\mathrm{e}_j} - \sum_{i=1}^{N_{\mathrm{i}_j}(t)} d^i_{\mathrm{i}_j} \tag{22}$$

The two terms on the right hand side of Eq. (22) are random sums with variances (Karlin and Taylor, 1975) given by

$$\mathrm{var}(N_{Y_j}(t)) = \mathrm{var}(N_{\mathrm{e}_j}(t))\overline{d}^2_{\mathrm{e}_j} + E[N_{\mathrm{e}_j}(t)]\sigma^2_{d_{\mathrm{e}_j}} + \mathrm{var}(N_{\mathrm{i}_j}(t))\overline{d}_{\mathrm{i}_j} + E[N_{\mathrm{i}_j}(t)]\sigma^2_{d_{\mathrm{i}_j}}.$$

Dividing by t and taking $t \to \infty$ gives

$$\sigma^2_{Y_j} = \lim_{t\to\infty}\frac{1}{t}\mathrm{var}(N_{Y_j}(t)) = \sigma^2_{\mathrm{e}_j}\overline{d}^2_{\mathrm{e}_j} + r_{\mathrm{e}_j}\sigma^2_{d_{\mathrm{e}_j}} + \sigma^2_{\mathrm{i}_j}\overline{d}^2_{\mathrm{i}_j} + r_{\mathrm{i}_j}\sigma^2_{d_{\mathrm{i}_j}}.$$

Covariances can be derived similarly to obtain,

$$\gamma_{Y_1Y_2} = \overline{d}_{\mathrm{e}_1}\overline{d}_{\mathrm{e}_2}\gamma_{\mathrm{e}_1\mathrm{e}_2} + \overline{d}_{\mathrm{i}_1}\overline{d}_{\mathrm{i}_2}\gamma_{\mathrm{i}_1\mathrm{i}_2} - \overline{d}_{\mathrm{e}_1}\overline{d}_{\mathrm{i}_2}\gamma_{\mathrm{e}_1\mathrm{i}_2} - \overline{d}_{\mathrm{i}_1}\overline{d}_{\mathrm{e}_2}\gamma_{\mathrm{i}_1\mathrm{e}_2}.$$

Thus,

$$\sigma^2_{\mathrm{out}_j} = \left(\sigma^2_{\mathrm{e}_j}\overline{d}^2_{\mathrm{e}_j} + r_{\mathrm{e}_j}\sigma^2_{d_{\mathrm{e}_j}} + \sigma^2_{\mathrm{i}_j}\overline{d}^2_{\mathrm{i}_j} + r_{\mathrm{i}_j}\sigma^2_{d_{\mathrm{i}_j}}\right)/\theta_j^2,$$

and

$$\gamma_{\mathrm{out}} = \left(\overline{d}_{\mathrm{e}_1}\overline{d}_{\mathrm{e}_2}\gamma_{\mathrm{e}_1\mathrm{e}_2} + \overline{d}_{\mathrm{i}_1}\overline{d}_{\mathrm{i}_2}\gamma_{\mathrm{i}_1\mathrm{i}_2} - \overline{d}_{\mathrm{e}_1}\overline{d}_{\mathrm{i}_2}\gamma_{\mathrm{e}_1\mathrm{i}_2} - \overline{d}_{\mathrm{i}_1}\overline{d}_{\mathrm{e}_2}\gamma_{\mathrm{i}_1\mathrm{e}_2}\right)/\left(\theta_1\theta_2\right),$$

and therefore

$$\rho_{\text{out}} = \frac{\overline{d}_{\text{e}_1}\overline{d}_{\text{e}_2}\gamma_{\text{e}_1\text{e}_2} + \overline{d}_{\text{i}_1}\overline{d}_{\text{i}_2}\gamma_{\text{i}_1\text{i}_2} - \overline{d}_{\text{e}_1}\overline{d}_{\text{i}_2}\gamma_{\text{e}_1\text{i}_2} - \overline{d}_{\text{i}_1}\overline{d}_{\text{e}_2}\gamma_{\text{i}_1\text{e}_2}}{\sqrt{\left(\sigma^2_{\text{e}_1}\overline{d}^2_{\text{e}_1} + r_{\text{e}_1}\sigma^2_{d_{\text{e}_1}} + \sigma^2_{\text{i}_1}\overline{d}^2_{\text{i}_1} + r_{\text{i}_1}\sigma^2_{d_{\text{i}_1}}\right)\left(\sigma^2_{\text{e}_2}\overline{d}^2_{\text{e}_2} + r_{\text{e}_2}\sigma^2_{d_{\text{e}_2}} + \sigma^2_{\text{i}_2}\overline{d}^2_{\text{i}_2} + r_{\text{i}_2}\sigma^2_{d_{\text{i}_2}}\right)}}.$$

In the symmetric case discussed in the text, this simplifies to Eq. (10).

To combine variable PSP amplitudes (i.i.d. random jumps, d_i, with CV=CV_d) with synaptic failure (probability of release p), we can multiply each jump d_i by an i.i.d. binomial variable, b_i (with $\Pr(b_i = 1) = p$) to obtain the "effective" jumps. In the symmetric case, the CV of this product is given by $\sqrt{(\text{CV}_d^2 + 1 - p)/p}$. Making the substitution $\text{CV}_d \to \sqrt{(\text{CV}_d^2 + 1 - p)/p}$ in Eq. (10) gives Eq. (11).

E.2 The PIF with coupling

Now suppose that the subthreshold membrane potentials $V_1(t)$ and $V_2(t)$ of the PIFs driven by the stationary signals $\text{in}_1(t)$ and $\text{in}_2(t)$ obey the coupled equations

$$dV_1 = \text{in}_1(t)dt + c_1\text{out}_2(t)dt$$
$$dV_2 = \text{in}_2(t)dt + c_2\text{out}_1(t)dt,$$

with out_1 and out_2 the output spike trains. Thus, each output spike from neuron 2 increments V_1 by an amount c_1 and vice versa. We assume that $c_j < \theta_j$ so that a spike from one neuron cannot drive the other from reset to threshold. Then the output spike counts obey the coupled equations,

$$N_{\text{out}_1}(t) = \frac{N_{\text{in}_1}(t) + c_1 N_{\text{out}_2}(t) + V_1(0) - V_1(t)}{\theta} \tag{23}$$
$$N_{\text{out}_2}(t) = \frac{N_{\text{in}_2}(t) + c_2 N_{\text{out}_1}(t) + V_2(0) - V_2(t)}{\theta}.$$

Defining $n_j(t) = N_{\text{in}_j}(t) + V_j(0) - V_j(t)$, we can solve Eq. (23) for $N_{\text{out}_j}(t)$ to obtain

$$N_{\text{out}_1}(t) = \frac{\theta_2 n_1(t) + c_1 n_2(t)}{\theta_1\theta_2 - c_1c_2}, \qquad N_{\text{out}_2}(t) = \frac{\theta_1 n_2(t) + c_2 n_1(t)}{\theta_1\theta_2 - c_1c_2}. \tag{24}$$

Thus, in order to have non-zero firing rates, we must assume that $\theta_2\mu_{\text{in}_1} + c_1\mu_{\text{in}_2} > 0$ and $\theta_1\mu_{\text{in}_2} + c_2\mu_{\text{in}_1} > 0$ and the firing rates are $r_{\text{out}_1} = (\theta_2\mu_{\text{in}_1} + c_1\mu_{\text{in}_2})/(\theta_1\theta_2 - c_1c_2)$ and $r_{\text{out}_2} = (\theta_1\mu_{\text{in}_2} + c_2\mu_{\text{in}_1})/(\theta_1\theta_2 - c_1c_2)$.

The following theorem gives the total output correlation.

Theorem 2. *The output correlation coefficient between the output of a pair of coupled PIFs driven by correlated stationary inputs,* $\text{in}_1(t)$ *and* $\text{in}_2(t)$ *with coupling terms* c_1 *and* c_2 *is*

$$\rho_{\text{out}} = \frac{(\theta_1\theta_2 + c_1c_2)\gamma_{\text{in}} + c_2\theta_2\sigma^2_{\text{in}_1} + c_1\theta_1\sigma^2_{\text{in}_2}}{\sqrt{\left(\theta_2^2\sigma^2_{\text{in}_1} + c_1^2\sigma^2_{\text{in}_2} + 2c_1\theta_2\gamma_{\text{in}}\right)\left(\theta_1^2\sigma^2_{\text{in}_2} + c_2^2\sigma^2_{\text{in}_1} + 2c_2\theta_1\gamma_{\text{in}}\right)}}. \tag{25}$$

Proof. From Eq. (24)

$$\begin{aligned}
\gamma_{\text{out}} &= \lim_{t\to\infty} \frac{1}{t}\text{cov}\left(N_{\text{out}_1}(t), N_{\text{out}_2}(t)\right) \\
&= \lim_{t\to\infty} \frac{1}{t}\text{cov}\left(\frac{\theta_2 n_1(t) + c_1 n_2(t)}{\theta_1\theta_2 - c_1c_2}, \frac{\theta_1 n_2(t) + c_2 n_1(t)}{\theta_1\theta_2 - c_1c_2}\right) \\
&= \frac{1}{\left(\theta_1\theta_2 - c_1c_2\right)^2} \lim_{t\to\infty} \frac{1}{t}\text{cov}\left(\theta_2 n_1(t) + c_1 n_2(t), \theta_1 n_2(t) + c_2 n_1(t)\right) \\
&= \frac{1}{\left(\theta_1\theta_2 - c_1c_2\right)^2} \left((\theta_1\theta_2 + c_1c_2)\gamma_{n_1n_2} + c_2\theta_2\sigma_{n_1}^2 + c_1\theta_1\sigma_{n_2}^2\right).
\end{aligned}$$

By an identical argument,

$$\sigma_{\text{out}_1}^2 = \frac{1}{\left(\theta_1\theta_2 - c_1c_2\right)^2} \left(\theta_2^2\sigma_{n_1}^2 + c_1^2\sigma_{n_2}^2 + 2c_1\theta_2\gamma_{n_1n_2}\right),$$

with a a symmetric expression for $\sigma_{\text{out}_2}^2$. Therefore,

$$\rho_{\text{out}} = \frac{\gamma_{\text{out}}}{\sigma_{\text{out}_1}\sigma_{\text{out}_2}} = \frac{(\theta_1\theta_2 + c_1c_2)\gamma_{n_1n_2} + c_2\theta_2\sigma_{n_1}^2 + c_1\theta_1\sigma_{n_2}^2}{\sqrt{\left(\theta_2^2\sigma_{n_1}^2 + c_1^2\sigma_{n_2}^2 + 2c_1\theta_2\gamma_{n_1n_2}\right)\left(\theta_1^2\sigma_{n_2}^2 + c_2^2\sigma_{n_1}^2 + 2c_2\theta_1\gamma_{n_1n_2}\right)}}. \tag{26}$$

All that is left is to is to show that $\sigma_{n_j} = \sigma_{\text{in}_j}$ and $\gamma_{n_1n_2} = \gamma_{\text{in}}$. We have

$$\begin{aligned}
\gamma_{n_1n_2} &= \lim_{t\to\infty} \frac{1}{t}\text{cov}\left(n_1(t), n_2(t)\right) \\
&= \lim_{t\to\infty} \frac{1}{t}\text{cov}\left(N_{\text{in}_1}(t) + V_1(0) - V_1(t), N_{\text{in}_2}(t) + V_2(0) - V_2(t)\right) \\
&= \lim_{t\to\infty} \frac{1}{t}\left(\text{cov}\left(N_{\text{in}_1}(t), N_{\text{in}_2}(t)\right) + o(t)\right) \qquad (27) \\
&= \gamma_{\text{in}}
\end{aligned}$$

where (27) follows from Lemma 1 and the assumption that $(V_1(t), V_2(t))$ is ergodic with finite second moments. By an identical argument, we have $\sigma_{n_j}^2 = \sigma_{\text{in}_j}^2, \;\; j = 1, 2.$

□

In the symmetric case, we define $u = c/\theta$ and obtain Eq. (12).

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