Notes on Meanfield Models for Spiking Networks for CIRM Course, March 2022

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Contents

1   The diffusion approximation

In the first lecture we will discuss meanfield methods for networks of spiking neurons which rely on three main assumptions:

1. Each neuron in the network receives a large number of weakly correlated inputs.

2. The strength of each individual input is small.

3. The statistics of the input and output spike trains is Poisson.

These three assumptions allow us to develop a self-consistent theory for the probability distribution of membrane potentials in the network [3, 2]. In a practical sense this theory allows us to calculate the stationary states of the network and the linear stability of these states analytically for the so-called linear (or non-leaky) integrate-and-fire model [9], and leaky integrate-and-fire (LIF) neuron [5, 4]. Some asymptotic results can be obtained from other types of model neurons, such as the quadratic integrate-and-fire (QIF) neuron [6]. Finally we can also solve for the stationary states and linear stability (or response to weak inputs if stable) very efficiently for any 1D neuron spiking model [16].

1.1   A single neuron

First we consider a single LIF neuron which receives a large number of excitatory and inhibitory inputs. The membrane potential of the neuron obeys

$$C\dot{V} = -g_L(V - E) + \bar{I}(t), \tag{S.1}$$

where $[C] \equiv \mu\text{F}$ is the membrane capacitance, $[g_L] \equiv \text{mS}$ is the leak conductance and $[E] \equiv \text{mV}$ is the reset potential. The current then has units of μA. We say that a spike is generated at a time t_{spike} whenever $V(t_{\text{spike}}) = \tilde{V}_t$, after

which it is reset to $V(t_{\text{spike}}^{+}) = \tilde{V}_r$. The membrane potential is held at this reset potential for a refractory time τ_r.

For simplicity we will consider a shifted membrane potential so that the effective rest potential is zero. Also we will divide all terms by the leak conductance and so write

$$\tau\dot{V} = -V + I(t), \tag{S.2}$$

where $[\tau] \equiv$ ms is the membrane time constant and $I(t)$ is actually the input current divided by the the leak conductance, i.e. it has units of mV. The rescaled threshold and reset voltages are V_t and V_r respectively.

We consider an external current due to the bombardment of inputs from C_e excitatory and C_i inhibitory synapses. Specifically, we take

$$I(t) = J_e \sum_{j=1}^{C_e} \sum_k \delta(t - t_j^k) - J_i \sum_{j=1}^{C_i} \sum_k \delta(t - t_j^k), \tag{S.3}$$

where J_e and J_i are the synaptic efficacies and t_j^k is the time of the kth spike of input j. The Dirac-delta function indicates that the spike causes a jump (of amplitude J_e or -J_i) in the membrane potential of the post-synaptic neuron. We assume that the inputs are Poisson processes with rates ν_e and ν_i respectively.

1.1.1 Derivation of the Fokker-Planck Equation

Given that the dynamics of V are stochastic we can consider the evolution of the probability distribution for V, $P(V, t)$. The following integral expresses the sum of possible paths from an initial state (V_0, t_0) to a state after some brief time interval $(V, t + \Delta t)$ for a Markov process

$$P(V, t + \Delta t | V_0, t_0) = \int_{-\infty}^{\infty} dV' P(V, t + \Delta t | V', t) P(V', t | V_0, t_0). \tag{S.4}$$

We take Δt small enough so that there are only three possible paths for the voltage: 1 - there has been no input, 2 - there is one excitatory input or 3 - there is one inhibitory input. Then we can write

$$P(V, t + \Delta t | V', t) = P_0 \cdot \delta(V - V_0') + P_+ \cdot \delta(V - V_+') + P_- \cdot \delta(V - V_-'), \tag{S.5}$$

where $P_0 = 1 - (C_e\nu_e + C_i\nu_i)\Delta t$, $P_+ = C_e\nu_e\Delta t$ and $P_- = C_i\nu_i\Delta t$. The physical meaning of V_0' is that it is the voltage after a time Δt when the

initial voltage is V' and there are no inputs. We calculate its value from Eq.S.2 and do the same for V'_+ and V'_-. This yields

$$
\begin{aligned}
V'_0 &= V'e^{-\Delta t/\tau}, \\
&\sim V'(1 - \frac{\Delta t}{\tau}), & \text{(S.6)} \\
V'_+ &\sim V'(1 - \frac{\Delta t}{\tau}) + J_e, & \text{(S.7)} \\
V'_- &\sim V'(1 - \frac{\Delta t}{\tau}) - J_i. & \text{(S.8)}
\end{aligned}
$$

Plugging Eq.S.5 into Eq.S.4 gives

$$
\begin{aligned}
P(V, t + \Delta t) &= P_0 \int_{-\infty}^{\infty} dV' \delta(V - V'(1 - \frac{\Delta t}{\tau})) P(V', t) \\
&+ P_+ \int_{-\infty}^{\infty} dV' \delta(V - V'(1 - \frac{\Delta t}{\tau}) - J_e) P(V', t) \\
&+ P_- \int_{-\infty}^{\infty} dV' \delta(V - V'(1 - \frac{\Delta t}{\tau}) + J_i) P(V', t).
\end{aligned}
$$

For the first integral in Eq.S.9 we define the dummy variable $x = V'(1 - \frac{\Delta t}{\tau})$ and for the second and third integrals $x = V'(1 - \frac{\Delta t}{\tau}) + J_e$ and $x = V'(1 - \frac{\Delta t}{\tau}) - J_i$ respectively. Then, after the change of variables we have

$$
\begin{aligned}
P(V, t + \Delta t) &= P_0(1 + \frac{\Delta t}{\tau}) \int_{-\infty}^{\infty} dx \delta(V - x) P(x(1 + \frac{\Delta t}{\tau}), t) \\
&+ P_+ \int_{-\infty}^{\infty} dx \delta(V - x) P(x - J_e, t) \\
&+ P_- \int_{-\infty}^{\infty} dx \delta(V - x) P(x + J_i, t) + \vartheta(\Delta t^2). \quad \text{(S.9)}
\end{aligned}
$$

By using the definition of the probabilities P_0, P_+ and P_- we can write

$$
\begin{aligned}
P(V, t + \Delta t) &= \big(1 - (C_e \nu_e + C_i \nu_i) \Delta t \big) P(V(1 + \frac{\Delta t}{\tau})) \cdot (1 + \frac{\Delta t}{\tau}) \\
&+ C_e \nu_e \Delta t P(V - J_e) + C_i \nu_i \Delta t P(V + J_i, t). \quad \text{(S.10)}
\end{aligned}
$$

Now we expand the probability density in a Taylor series for small Δt, take the limit $\Delta t \to 0$ and obtain

$$
\frac{\partial P}{\partial t} = \frac{\partial}{\partial V}\Big(\frac{V}{\tau}P\Big) + C_e \nu_e \Big(P(V - J_e) - P(V, t)\Big) + C_i \nu_i \Big(P(V + J_i, t) - P(V, t)\Big).
$$
$$
\text{(S.11)}
$$

This equation is valid for synaptic inputs of any arbitrary amplitude. In the diffusion approximation we assume $J_e \ll 1$ and $J_i \ll 1$, expand

$$P(V - J_e) = P(V, t) - J_e \frac{\partial P}{\partial V} + \frac{J_e^2}{2} \frac{\partial^2 P}{\partial V^2} + \vartheta(J_e^3),$$

and truncate after the second order (diffusive) term. Finally, this leads to the Fokker-Planck equation

$$\frac{\partial P}{\partial t} = \frac{\sigma^2}{2\tau} \frac{\partial^2 P}{\partial V^2} + \frac{\partial}{\partial V}\left(\left(\frac{V - \mu}{\tau}\right)P\right), \qquad (\text{S.12})$$

where

$$\mu = \tau(J_e C_e \nu_e - J_i C_i \nu_i), \qquad (\text{S.13})$$
$$\sigma^2 = \tau(J_e^2 C_e \nu_e + J_i^2 C_i \nu_i). \qquad (\text{S.14})$$

Note that the Fokker-Planck equation can be written in the more general form of a continuity equation

$$\frac{\partial P}{\partial t} + \frac{\partial F}{\partial V} = 0, \qquad (\text{S.15})$$

where

$$F = -\frac{\sigma^2}{2\tau} \frac{\partial P}{\partial V} - \frac{(V - \mu)}{\tau} P, \qquad (\text{S.16})$$

is the probability flux. The boundary conditions for this Fokker-Planck equation are the following

1. $P(V_t, t) = 0$. The probability density is zero for all voltages above threshold. Therefore it must also go to zero at threshold, otherwise there would be a jump in the density which would generate an infinite firing rate.

2. $\nu(t) = F(V_t, t)$. The firing rate is just the probability flux at threshold. Using the definition of the flux, this leads to a condition for the slope of the probability density $\frac{\partial P}{\partial V} = -\frac{2\tau\nu(t)}{\sigma^2}$.

3. $\nu(t) = \lim_{\epsilon \to 0} F(V_r + \epsilon, t) - F(V_r - \epsilon, t)$. The flux which passes through threshold gets re-injected at the reset potential. This again leads to a condition on the jump in the slope of the probability density at reset.

4. $\int_{-\infty}^{V_t} dV P(V, t) + \tau_{ref} \nu(t) = 1$. $P(V, t)$ is a probability density.

5

1.1.2 Steady State Distribution

We can integrate Eq.S.12 directly and apply the boundary conditions to find the steady state solution. Setting the time derivative to zero and integrating Eq.S.12 once we find

$$\frac{\partial P_0}{\partial V} + \frac{2(V-\mu)}{\sigma^2} P_0 = C, \tag{S.17}$$

where $P_0(V)$ is the stationary distribution and C is a constant of integration. Applying the jump in the slope across the rest voltage we find that $C = 0$ for $V < V_r$ and $C = -\frac{2\tau\nu_0}{\sigma^2}$ for $V \geq V_r$, where ν_0 is the steady-state firing rate. Now we can write

$$\frac{\partial P_0}{\partial V} + \frac{2(V-\mu)}{\sigma^2} P_0 = -\frac{2\tau\nu_0}{\sigma^2} H(V - V_r), \tag{S.18}$$

where

$$H(x) = \begin{cases} 0, & \text{if } x < 0 \\ 1, & \text{if } x \geq 0. \end{cases} \tag{S.19}$$

We can integrate Eq.S.18 directly to obtain

$$P_0(V) = \frac{2\tau\nu_0}{\sigma} e^{-\frac{(V-\mu)^2}{\sigma^2}} \int_{\frac{V-\mu}{\sigma}}^{\frac{V_t-\mu}{\sigma}} dy e^{y^2} H\left(y - \frac{(V_r-\mu)}{\sigma}\right). \tag{S.20}$$

To find the firing rate we use the normalization condition number 4. After some algebra (hint: you will need to change the order of integration in the double integral) we find

$$\nu_0 = \phi(\mu, \sigma^2) = \left[\tau\sqrt{\pi} \int_{\frac{V_r-\mu}{\sigma}}^{\frac{V_t-\mu}{\sigma}} dy e^{y^2} \text{erfc}(-y) + \tau_{ref}\right]^{-1}. \tag{S.21}$$

1.1.3 The linear response

Let's assume that in addition to the background synaptic bombardment the neuron also receives a time-varying current injection and we want to predict the firing rate response. In general this is a very hard problem, but we can make progress by assuming that the time-varying current has a small amplitude. This allows us to linearize the dynamics around the stationary

state. In particular we now assume that $I(t) = I_{background}(t) + \epsilon I_{ext}(t)$, where $I_{background}$ is the input we have considered in the previous section. In the linear approximation we can just consider the Fourier decomposition of the external current injection and solve for each frequency separately. That is, we need only consider the response of the neuron to $I(t) = I_{background}(t) + \epsilon I_1 e^{i\omega t}$ (in the end we take the real part of the response) and then for an arbitrary input we can just some up modes with the appropriate weights. This input suggests the following ansatz

$$
\begin{aligned}
P(V,t) &= P_0(V) + \epsilon P_1(V)e^{i\omega t}, & \text{(S.22)}\\
\nu(t) &= \nu_0 + \epsilon \nu_1 e^{i\omega t}. & \text{(S.23)}
\end{aligned}
$$

Plugging these formula into Eq.S.12 and the corresponding relevant boundary conditions yields at order ϵ

$$
\begin{aligned}
i\omega P_1 &= \frac{\sigma^2}{2\tau}\frac{\partial^2 P_1}{\partial V^2} + \frac{\partial}{\partial V}\Big(-I_1 P_0 + (V-\mu)P_1\Big), & \text{(S.24)}\\
p_1(V_t) &= p_1(V_r^+) - p_1(V_r^-) = 0, & \text{(S.25)}\\
\frac{\partial P_1(V_t)}{\partial V} &= \frac{\partial P_1(V_r^+)}{\partial V} - \frac{\partial P_1(V_r^-)}{\partial V} = -\frac{2\tau\nu_1}{\sigma^2}. & \text{(S.26)}
\end{aligned}
$$

To clean up the equations and make the algebra a bit easier we define $Q_1 = \frac{\sigma^2}{2\tau\nu_0}$ and $y = (V-\mu)/\sigma$. This leads to the following equations

$$
\frac{1}{2}Q_1'' + yQ_1' + (1 - i\tau\omega)Q_1 = \frac{I_1}{\sigma}Q_0', \qquad \text{(S.27)}
$$

where $Q_0 = \frac{\sigma^2}{2\tau\nu_0}P_0$ and $Q' = \frac{\partial Q}{\partial y}$. The boundary conditions are

$$
\begin{aligned}
Q_1(y_t) &= Q_1(y_r^+) - Q_1(y_r^-) = 0, & \text{(S.28)}\\
Q_1'(y_t) &= Q_1'(y_r^+) - Q_1'(y_r^-) = -\frac{\nu_1}{\nu_0}. & \text{(S.29)}
\end{aligned}
$$

Now we are faced with a second-order, non-homogeneous linear differential equation. The solution consists of a linear combination of two independent solutions to the homogeneous equation plus the particular solution. So we can write

$$
Q_1(y) = \begin{cases} \alpha_+ \phi_1(y) + \beta_+ \phi_2(y) + Q_p(y), & \text{if } y > y_r \\ \alpha_- \phi_1(y) + \beta_- \phi_2(y) + Q_p(y), & \text{if } y \le y_r. \end{cases} \qquad \text{(S.30)}
$$

Note also that the stationary solution given this normalization obeys

$$\frac{1}{2}Q_0'' + yQ_0' + Q_0 = 0, \tag{S.31}$$

which can be differentiated to yield

$$\frac{1}{2}Q_0^{(k)} + yQ_0^{(k-1)} + (k-1)Q_0^{(k-2)}, \tag{S.32}$$

where $k > 2$. This leads us to propose $Q_p = CQ_0'$. Plugging this particular solution into Eq.S.27 reveals $C = -I_1/\sigma$. The homogeneous solutions are known functions, called confluent hypergeometric functions, see [1, 5] for details. These are not the easiest functions to work with but luckily we will not need to use them directly. If fact, in the end we will solve the Fokker-Planck equation numerically. In any case, we are free to choose two linearly independent functions which satisfy the homogeneous equation. We will take them such that the function ϕ_2 decays rapidly (exponentially) to zero for $y \to -\infty$ while the other does not, therefore we take $\alpha_- = 0$. We could have chosen the opposite, it is arbitrary at this point.

Now we apply the boundary conditions at threshold y_t, which can be written as

$$\begin{pmatrix} \phi_1(y_t) & \phi_2(y_t) \\ \phi_1'(y_t) & \phi_2'(y_t) \end{pmatrix} \begin{pmatrix} \alpha_+ \\ \beta_+ \end{pmatrix} = \begin{pmatrix} -1 \\ 2y_t \end{pmatrix} \frac{I_1}{\sigma(1 + i\tau\omega)} - \begin{pmatrix} 0 \\ \frac{\nu_1}{\nu_0} \end{pmatrix} \tag{S.33}$$

This linear system can be solved simply by multiplying both sides by the inverse matrix of the homogeneous functions evaluated at threshold. In the end we only need the value of α_+, which is

$$\alpha_+ = -(\phi_2'(y_t) + 2y_t\phi_2(y_t))e^{y_t^2}\frac{I_1}{\sigma(1 + i\tau\omega)} + \phi_2(y_t)e^{y_t^2}\frac{\nu_1}{\nu_0}. \tag{S.34}$$

To get the terms that go like $e^{y_t^2}$ we have used the fact that $Wr(\phi_1, \phi_2)' = -2yWr(\phi_1, \phi_2)$ where the "Wronskian" $Wr(\phi_1, \phi_2) = \phi_1\phi_2' - \phi_1'\phi_2$. Now we are almost done. In fact, applying the other boundary condition at the reset voltage y_r leads to an identical system of equations to Eq.S.33 with y_t replaced by y_r and with α_+ replaced by $\alpha_+ - \alpha_-$ (and the same for the βs). But, noting that we have chosen the functions such that $\alpha_- = 0$ means the two conditions, Eq.S.34 and the equivalent one for y_r must be equal. This gives us the linear response as

$$\nu_1 = I_1A(\omega), \tag{S.35}$$

8

where

$$A(\omega) = \frac{1}{\sigma(1 + i\tau\omega)} \frac{U'(y_t, \omega) - U'(y_r, \omega)}{U(y_t, \omega) - U(y_r, \omega)}, \tag{S.36}$$

and $U(y, \omega) = \phi_2(y, \omega)e^{y^2}$. Oftentimes it is convenient to express the linear response explicitly in terms of an amplitude and phase response, i.e. $A(\omega) = R(\omega)e^{i\phi(\omega)}$.

1.1.4 Solution of steady state and linear response with Laplace Transforms

For completeness I'm including here an alternative method for solving the Fokker-Planck (FP) equation. Sometimes it might be easier to work with the resultant formulas using Laplace transforms. Now we will write the FP equation in a slightly different form.

$$\frac{\partial P}{\partial t} + \frac{\partial F}{\partial V} = \nu(t)\Big(\delta(V - V_r) - \delta(V - V_t)\Big), \tag{S.37}$$

$$\tau F(V, t) = -\frac{\sigma^2}{2}\frac{\partial P}{\partial V} - (V - \mu)P. \tag{S.38}$$

So the boundary conditions at threshold and reset are now expressed explicitly in the continuity equation. Again we take $I(t) = \mu + \epsilon I_1 e^{i\omega t}$ and so $P(V, t) = P_0(V) + \epsilon P_1(V)e^{i\omega t}$ and $\nu(t) = \nu_0 + \epsilon\nu_1 e^{i\omega t}$. Then, at order $\vartheta(1)$ we have

$$\frac{\partial F_0}{\partial V} = \nu_0\Big(\delta(V - V_r) - \delta(V - V_t)\Big), \tag{S.39}$$

$$\tau F_0 = -\frac{\sigma^2}{2}\frac{\partial P_0}{\partial V} - (V - \mu)P_0. \tag{S.40}$$

We introduce the generalized Laplace transform $\tilde{A}(s) = \int_{-\infty}^{\infty} dV\, e^{sv}A(v)$. Applying this operation to all terms in Eqs.S.39-S.40 yields

$$-s\tilde{F}_0 = \nu_0\Big(e^{V_r s} - e^{V_t s}\Big), \tag{S.41}$$

$$\tau\tilde{F}_0 = s\frac{\sigma^2}{2}\tilde{P}_0 - \frac{d\tilde{P}_0}{ds} + \mu\tilde{P}_0. \tag{S.42}$$

The advantage of the Laplace transform is it converts the PDE into an ODE, which we can solve for easily. After just a bit of algebra we find

$$\tilde{P}_0(s) = \tau\nu_0 e^{\mu s + \frac{\sigma^2 s^4}{4}} \int_s^{\infty} \frac{dx}{x} e^{-(\mu x + \frac{\sigma^2 x^4}{4})}\Big(e^{V_t x} - e^{V_r x}\Big). \tag{S.43}$$

Now we can take advantage of the fact that $\tilde{P}_0(s=0) = \int_{-\infty}^{\infty} dV P_0(V) = 1$ and so find that $\tau\nu_0 \int_0^{\infty} \frac{dx}{x} e^{-(\mu x + \frac{\sigma^2 x^4}{4})} \left(e^{V_t x} - e^{V_r x} \right)$. If we make the simple substitution $y = \sigma x/2$ then the steady-state firing rate is

$$\nu_0 = \left[\tau \int_0^{\infty} \frac{dy}{y} e^{-y^2} \left(e^{2y_t y} - e^{2y_r y} \right) \right]^{-1}, \tag{S.44}$$

where $y_t = (V_t - \mu)/\sigma$ and $y_r = (V_r - \mu)/\sigma$. Compare this with Eq.S.21 without the refractory period (you can always add it here if you want).

At order $\vartheta(\epsilon)$ we have

$$i\omega P_1 + \frac{dF_1}{dV} = \nu_1 \Big(\delta(V - V_r) - \delta(V - V_t) \Big), \tag{S.45}$$

$$\tau F_1 = -\frac{\sigma^2}{2} \frac{dP_1}{dV} - (V - \mu)P_1 + I_1 P_0. \tag{S.46}$$

Once again we can simply integrate the ODE directly. Then we apply the condition $\tilde{P}_1(s=0) = \int_{-\infty}^{\infty} dV P_1(V) = 0$ to find $\nu_1 = I_1 A(\omega)$

$$A(\omega) = \frac{2\nu_0}{\sigma(1 + i\tau\omega)} \frac{B_1(\omega)}{B_0(\omega)}, \tag{S.47}$$

where

$$B_n(\omega) = \int_0^{\infty} \frac{dy}{y} y^{n + i\tau\omega} e^{-y^2} \left(e^{y_t y} - e^{y_r y} \right). \tag{S.48}$$

Compare Eqs.S.47 and S.36.

1.1.5 The low frequency response

In the limit $\omega \to 0$ we can extract a simpler formula for the rate response. We set $\omega \to 0$ in Eq.S.27 and write

$$\frac{1}{2} Q_1'' + y Q_1' + Q_1 = \frac{I_1}{\sigma} Q_0', \tag{S.49}$$

The boundary conditions are

$$Q_1(y_t) = Q_1(y_r^+) - Q_1(y_r^-) = 0, \tag{S.50}$$

$$Q_1'(y_t) = Q_1'(y_r^+) - Q_1'(y_r^-) = -\frac{\nu_1}{\nu_0}, \tag{S.51}$$

$$\int_{-\infty}^{y_t} dy Q_1 = 0. \tag{S.52}$$

10

Again the solution to Eq.S.49 requires two independent homogeneous solutions and one particular solution. The particular solution is as before $Q_p = -I_1 Q_0'/\sigma$. The homogeneous solutions are now no longer confluent hypergeometric functions. Rather, one obvious choice is simply Q_0, the stationary solution. Another independent solution is e^{-y^2} (this is easy to check). Therefore we can write

$$Q_1 \begin{cases} \alpha_+ Q_0 + \beta_+ e^{-y^2} - \frac{I_1}{\sigma} Q_0', & \text{if } y > y_r \\ \alpha_- Q_0 + \beta_- e^{-y^2} - \frac{I_1}{\sigma} Q_0', & \text{if } y \leq y_r. \end{cases} \tag{S.53}$$

i. $Q_1(y_t) = 0$
   Applying this condition leads to $\beta_+ = -\frac{I_1}{\sigma} e^{y_t^2}$.
ii. $Q_1'(y_t) = -\frac{\nu_1}{\nu_0}$
   Applying this condition leads to $\alpha_+ = \nu_1/\nu_0$.
iii. $Q_1'(y_r^+) - Q_1'(y_r^-) = 0$
   Applying this condition leads to $(\alpha_+ - \alpha_-)Q_0(y_r) + (\beta_+ - \beta_-)e^{-y_r^2} + \frac{I_1}{\sigma} = 0$.
One solution to this equation is $\alpha_- = \alpha_+$ and hence $\beta_- = \beta_+ + \frac{I_1}{\sigma} e^{y_r^2}$. This is not a gratuitous choice, it is actually the only solution which also satisfies the condition $Q_1'(y_r^+) - Q_1'(y_r^-) = -\nu_1/\nu_0$.
iv. $\int_{-\infty}^{y_t} dy Q_1 = 0$
   This condition gives the linear response in the low-frequency limit. Namely

$$\nu_1 = \frac{\tau \sqrt{\pi} \nu_0^2 I_1}{\sigma} \left(e^{y_t^2} \text{erfc}(-y_t) - e^{y_r^2} \text{erfc}(-y_r) \right). \tag{S.54}$$

EXERCISE PROBLEM: Show that the linear response in this limit can be written

$$\nu_1 = I_1 \frac{\partial \nu_0}{\partial \mu}. \tag{S.55}$$

What does this mean? Also, show that the method using Laplace transforms also gives the same result. That is, show that Eq.S.47 can be reduced to Eq.S.54 in that limit.

1.1.6   The high frequency limit

Again, we can obtain a simplified formula for the linear response in the limit $\omega \to \infty$. We are looking for a solution to the equation

$$i\tau \omega P_1 = \frac{\sigma^2}{2} \frac{\partial^2 P_1}{\partial V^2} + (V - \mu) \frac{\partial P_1}{\partial V} + P_1 - I_1 \frac{\partial P_0}{\partial V}, \tag{S.56}$$

11

where $\omega \ll 1$. Again we consider a homogeneous and a particular solution, i.e. $P_1 = \phi + P_p$. The particular solution is, to leading order $P_p = -\frac{I_1}{i\tau\omega}\frac{\partial P_0}{\partial V}$. The homogeneous solution satisfies

$$i\tau\omega\phi = \frac{\sigma^2}{2}\frac{\partial^2 \phi}{\partial V^2} + (V - \mu)\frac{\partial \phi}{\partial V} + \phi. \tag{S.57}$$

We consider a solution of the form $\phi = ke^{X(V)-X(V_t)}$, where k is a constant. Note that the condition $P_1(V_t)$ immediately gives us that $k = -\frac{2\tau\nu_0}{\sigma^2}\frac{I_1}{i\tau\omega}$. In any case, plugging our ansatz for ϕ into Eq.S.57 yields to leading order that $(\frac{\partial X}{\partial V})^2 = 2i\frac{\tau\omega}{\sigma^2}$. Finally, we have that

$$\nu_1 = F_1(V_t) = -\frac{\sigma^2}{2\tau}\frac{\partial P_1}{\partial V}(V_t), \tag{S.58}$$

where

$$\frac{\partial P_1}{\partial V} = -\frac{I_1}{i\tau\omega}\frac{\partial^2 P_0}{\partial V^2}(V_t) + k\frac{\partial X}{\partial V}(V_t). \tag{S.59}$$

Putting these two equations together gives the linear response as $\nu_1 = I_1 A(\omega)$ where

$$A(\omega) = \nu_0 e^{i\pi/4}\sqrt{\frac{2}{\tau\omega\sigma^2}}. \tag{S.60}$$

1.1.7 Numerical Method for the Fokker-Planck Equation

We have seen that it is relatively straightforward to calculate the stationary state of an LIF neuron in the diffusion limit of many weak synaptic inputs. We have also seen that something as simple as the linear response is already a pretty daunting calculation although we can get some intuition by looking at the low and high-frequency responses. So how should we calculate the response? Should we evaluate the confluent hypergeometric functions in Eq.S.36 or the integrals in Eq.S.47? You can do this, but it is not easy. Luckily we have a much simpler alternative, described in detail in [16]. The crux of the method is to directly integrate the Fokker-Planck equation by separating the probability density and the probability flux and solving them independently.

Stationary Distribution Note that we will be solving for the stationary solution and the linear response. The stationary problem can be written

$$\frac{dF_0}{dV} = \nu_0\Big(\delta(V - V_r) - \delta(V - V_t)\Big), \tag{S.61}$$

$$-\tau F_0 = \frac{\sigma^2}{2}\frac{dP_0}{dV} + (V - \mu)P_0 \tag{S.62}$$

We now consider the flux and density normalized by the stationary firing rate, i.e. $F_0 = \nu_0 f_0$ and $P_0 = \nu_0 p_0$. The normalized functions satisfy

$$\frac{df_0}{dV} = \delta(V - V_r) - \delta(V - V_t), \tag{S.63}$$

$$-\tau f_0 = \frac{\sigma^2}{2}\frac{dp_0}{dV} + (V - \mu)p_0. \tag{S.64}$$

Now the idea is the following. The flux is equal to the firing rate at threshold. Therefore $j_0(V_t) = 1$. So if we discretize the voltage we can integrate the flux from threshold down to some lower bound, well below the reset voltage. At reset there is a downward jump in the flux. Specifically, the numerical method for the flux is

$$j_0^{k-1} = j_0^k - \delta_{k,k_r+1}, \tag{S.65}$$

$$j_0^N = 1, \tag{S.66}$$

where there are a total of N mesh points, $k \in [1, N]$ on the interval $V \in [V_{lb}, V_t]$ such that V_r also falls on a mesh point and $V_{lb} \ll V_r$.

Integrating Eq.S.64 requires a bit more work. First, we rewrite it as

$$-\frac{dp_0}{dV} = Gp_0 + H, \tag{S.67}$$

where $G = \frac{2}{\sigma^2}(V - \mu)$ and $H = \frac{2\tau}{\sigma^2}j_0$. We integrate this equation over an interval starting from a mesh point V_k, i.e. $\frac{d}{dV}\left(pe^{\int_{V_k}^V dxG(x)}\right) = -He^{\int_{V_k}^V dxG(x)}$, which gives

$$p(V) = p^k e^{-\int_{V_k}^V dxG(x)} - e^{-\int_{V_k}^V dxG(x)}\int_{V_k}^V du H(u)e^{\int_{V_k}^u dxG(x)}. \tag{S.68}$$

Note that $p(V_k) = p^k$. The value of the density at the next mesh point is

$$p^{k-1} = p^k e^{\int_{V_{k-1}}^{V_k} dxG(x)} + \int_{V_{k-1}}^{V_k} du H(u)e^{\int_{V_{k-1}}^u dxG(x)}. \tag{S.69}$$

13

Because the integrals are over a very small interval in voltage we can assume that the functions G and H are constant, i.e. $\int_{V_{k-1}}^{V_k} dx G(x) = (V_k - V_{k-1})G(V_k) = \Delta \cdot G(V_k)$, and $\int_{V_{k-1}}^{V_k} du H(u) e^{\int_{V_{k-1}}^{u} dx G(x)} = \int_{V_{k-1}}^{V_k} du H(u) e^{(u-V_{k-1})G(V_k)} = \frac{H(V_k)}{G(V_k)}\left(e^{\Delta \cdot G(V_k)} - 1\right)$. Finally we have

$$
\begin{aligned}
p^{k-1} &= p^k A^k + 2\tau j_0^k B^k, &\text{(S.70)}\\
p^N &= 0, &\text{(S.71)}
\end{aligned}
$$

where

$$
\begin{aligned}
A^k &= e^{\Delta \cdot G^k}, &\text{(S.72)}\\
B^k &= \frac{1}{\sigma^2}\frac{(e^{\Delta \cdot G^k} - 1)}{\Delta \cdot G^k}. &\text{(S.73)}
\end{aligned}
$$

Also note that if $G = 0$ we should set $B^k = \frac{1}{\sigma^2}$. So now we just need to integrate j_0 and p_0 from V_t down to V_{lb}. The stationary firing rate

$$
\nu_0 = \frac{1}{\sum_{k=1}^{N}\Delta \cdot p_0^k}. \qquad\qquad \text{(S.74)}
$$

Linear Response

Now we consider the linear response. The equations are

$$
\begin{aligned}
i\omega P_1 + \frac{dJ_1}{dV} &= \nu_1\left(\delta(V - V_r) - \delta(V - V_t)\right), &\text{(S.75)}\\
-\tau J_1 &= \frac{\sigma^2}{2}\frac{dP_1}{dV} + (V - \mu)P_1 - I_1 P_0. &\text{(S.76)}
\end{aligned}
$$

We consider distinct modulations of the probability density and flux due to changes in the input and in the firing rate response, respectively. That is, we write

$$
\begin{aligned}
P_1 &= \nu_1 P_\nu + I_1 P_I, &\text{(S.77)}\\
J_1 &= \nu_1 J_\nu + I_1 J_I. &\text{(S.78)}
\end{aligned}
$$

Now we consider the effect of each in turn. (We can do all of the this because the system is linear, of course.)

i. $\nu_1 = 1,\ I_1 = 0$

The equations are

$$-\frac{dJ_\nu}{dV} = i\omega P_\nu - \delta(V - V_r) + \delta(V - V_t), \qquad (\text{S.79})$$

$$-\frac{dP_\nu}{dV} = \frac{2}{\sigma^2}(V - \mu)P_\nu + \frac{2\tau}{\sigma^2}J_\nu. \qquad (\text{S.80})$$

for which the numerical method is

$$J_\nu^{k-1} = J_\nu^k + \Delta \cdot i\omega P_\nu^k - \delta_{k,k_r+1}, \qquad (\text{S.81})$$

$$P_\nu^{k-1} = P_\nu^k A^k + \Delta \cdot 2\tau J_\nu^k B^k, \qquad (\text{S.82})$$

with the "initial conditions" $J_\nu(V_t) = J_\nu^N = 1$ and $P_\nu^N = 0$.

ii. $\nu_1 = 0,\ I_1 = 1$

The equations are

$$-\frac{dJ_I}{dV} = i\omega P_I, \qquad (\text{S.83})$$

$$-\frac{dP_I}{dV} = \frac{2}{\sigma^2}(V - \mu)P_I + \frac{2\tau}{\sigma^2}J_I - \frac{2}{\sigma^2}P_0, \qquad (\text{S.84})$$

for which the numerical method is

$$J_I^{k-1} = J_I^k + \Delta \cdot i\omega P_I^k, \qquad (\text{S.85})$$

$$P_I^{k-1} = P_I^k A^k + \Delta \cdot 2\tau \left(J_I^k - P_0^k\right) B^k, \qquad (\text{S.86})$$

with the "initial conditions" $J_I^N = 0$ and $P_I^N = 0$. The final step is to realize that for low enough voltages (V_{lb} must really be low enough) the probability flux is zero. That is $J_1(V_{lb}) = 0$. This conditions yields the linear response

$$\nu_1 = -I_1 \frac{J_I(V_{lb})}{J_\nu(V_{lb})}. \qquad (\text{S.87})$$

So clearly $A(\omega) = -J_I(V_{lb})/J_\nu(V_{lb})$.

1.2 Recurrent Networks

We have spent a lot of time studying the response of a single neuron. As it turns out, meanfield theory for recurrent networks is precisely the same with

15

the twist that the input and output must be made self-consistent. In any case we will not need to go through all of the theory again, we will just need to alter the notation a bit. In order to apply the theory we will need to specify some details regarding the network connectivity, essentially whether or not it is "all-to-all" or sparsely connected. Also, to model neuronal circuits we will need excitation and inhibition. So we will have two FP equations etc., i.e. there's double the algebra but conceptually there is nothing new. What is new is how to calculate the meanfield input itself, namely the mean and variance of the input.

1.2.1 All-to-all Networks

Let's first consider a networks of all-to-all connected E and I LIF neurons. The membrane potential of an excitatory neuron i obeys

$$
\tau_E \frac{dV_i^E}{dt} = -V_i^E + \tau_E \Big(\frac{J_{EE}}{N} \sum_{j=1}^{N_E} \sum_k \delta(t - t_j^k - D_{EE}) - \frac{J_{EI}}{N} \sum_{j=1}^{N_i} \sum_k \delta(t - t_j^k - D_{EI}) \Big)
$$
$$
+ I(t) + \sigma_{ext} \xi_i(t). \tag{S.88}
$$

and there is an analogous equation for the inhibitory neurons. A couple of remarks: the sums of delta functions looks quite similar to the bombardment of inputs impinging on the single neuron model we considered previously. Here, however, these inputs come from other neurons in the network. Also, we assume the spikes arrive with some delay. Let's again assume that the input statistics (and hence the output spike trains as well!) are Poisson. Then the mean input to an excitatory neuron is

$$
\mu = \tau_E \Big(J_{EE} \nu_{E0} - J_{EI} \nu_{I0} \Big) + I_0, \tag{S.89}
$$

where $I(t) = I_{E0}$ is a constant and ν_{E0} and ν_{I0} are the stationary firing rates of the excitatory and inhibitory neurons. So we don't know these values a priori, we will need to determine them self-consistently. Now, the variance of the input is

$$
\sigma^2 = \tau_E \Big(\frac{J_{EE}}{N} \nu_{E0} - \frac{J_{EI}}{N} \nu_{I0} \Big) + \sigma_{ext}^2. \tag{S.90}
$$

So in the large system-size limit $N \to \infty$ the fluctuations due to recurrent inputs vanish. Note, however, that these finite-size fluctuations are the same from cell to cell, they're not uncorrelated noise sources. That is why, in

16

an all-to-all coupled network we need to drive the neurons with an external source of fluctuations (here a Gaussian white noise term proportional to σ_{ext}).

Now, the same theory applies as before. Let's consider the stationary state and linear response in turn.

BTW, synaptic kinetics are not actually instantaneous, i.e. cells do not receive a train of Dirac-delta functions. This is merely an idealization which we assume for simplicity. In the all-to-all network we can consider more realistic, finite synaptic kinetics and still apply the same theory. For example, we can model the neuronal dynamics as

$$\tau_E \frac{dV_i^E}{dt} = -V_i^E + \tau_E \Big(\frac{J_{EE}}{N} s_E(t) - \frac{J_{EI}}{N} s_I \Big)$$
$$+ I(t) + \sigma_{ext} \xi_i(t), \tag{S.91}$$

$$\tau_E^{\text{decay}} \dot{s}_E = -s_E + x_E, \tag{S.92}$$

$$\tau_E^{\text{rise}} \dot{x}_E = -x_E + \sum_{j=1}^{N_E} \sum_k \delta(t - t_j^k - D_{EE}), \tag{S.93}$$

$$\tau_I^{\text{decay}} \dot{s}_I = -s_I + x_I, \tag{S.94}$$

$$\tau_I^{\text{rise}} \dot{x}_I = -x_I + \sum_{j=1}^{N_i} \sum_k \delta(t - t_j^k - D_{EI}), \tag{S.95}$$

where the x and s variables model the rise and decay phases of the post-synaptic response. It turns out that the rise and decay time constants strongly affect the linear stability of the steady state.

Stationary State

The stationary firing rates are given by

$$\nu_{E0} = \phi_E(\mu(\nu_{E0}, \nu_{I0}), \sigma^2(\nu_{E0}, \nu_{I0})), \tag{S.96}$$

$$\nu_{I0} = \phi_I(\mu(\nu_{E0}, \nu_{I0}), \sigma^2(\nu_{E0}, \nu_{I0})), \tag{S.97}$$

where the function ϕ is given by Eq.S.21. How do we solve this set of nonlinear equations? Basically we will need to make a guess for ν_{E0} and for ν_{I0} and then evaluate ϕ using this guess. If the ϕs gives rates which are close enough to our guess then we stop, otherwise we should update our guess. We can evaluate ϕ using the numerical method we have learned.

Linear Stability

Let's consider the response of the system to small perturbations in the network activity. These might be due to external inputs, i.e. the linear

response, or due to intrinsic fluctuations. In fact, we cannot consider the linear response of the system if the stationary state is unstable, so first we need to consider the linear stability. The only difference with the single neuron is that whereas before the perturbation I_1 was external, now $I_{E1} = \tau_E(J_{EE}\nu_{E1} - J_{EI}\nu_{I1})$ and $I_{I1} = \tau_I(J_{IE}\nu_{E1} - J_{II}\nu_{I1})$. This means that Eq.S.35 becomes

$$\nu_{E1} = (J_{EE}\nu_{E1} - J_{EI}\nu_{I1})A_E(\omega), \tag{S.98}$$

$$\nu_{I1} = (J_{IE}\nu_{E1} - J_{II}\nu_{I1})A_I(\omega), \tag{S.99}$$

the solution of which is

$$(1 - J_{EE}A_E(\omega))(1 + J_{II}A_I(\omega)) + J_{EI}J_{IE}A_I(\omega)A_E(\omega) = 0. \tag{S.100}$$

So you calculate the linear response functions A separately for the excitatory and inhibitory populations and then search for the zeros of this complex function to find the eigenvalues. If the real part of the eigenvalue passes through zero as you vary some parameter then you have found an instability of the system. Alternatively you can set the real part identically equal to zero (as I have implicitly done here by writing just ω and then the zeros will give you the instability line for instabilities with frequency ω. There are oscillatory (Hopf) instabilities.

EXERCISE PROBLEM: Calculate the condition for a steady (saddle-node) bifurcation. What does this condition tell us about the synaptic properties in recurrent networks which favor bistability?

Linear Response

If the stationary state is stable, then one can calculate the linear response of the network to weak external drive. Specifically, if the external input to the excitatory cells is $I_{E,ext}(t) = I_{E0} + I_{E1}e^{i\omega t}$ and that to the inhibitory cells is $I_{I,ext}(t) = I_{I0} + I_{I1}e^{i\omega t}$, then the linear response is given by solving the system of equations

$$\begin{pmatrix} 1 - J_{EE}A_E(\omega) & J_{EI}A_E(\omega) \\ -J_{IE}A_I(\omega) & 1 + J_{II}A_I(\omega) \end{pmatrix} \begin{pmatrix} \nu_{E1} \\ \nu_{I1} \end{pmatrix} = \begin{pmatrix} I_{E1}A_E(\omega) \\ I_{I1}A_I(\omega) \end{pmatrix}. \tag{S.101}$$

Now it's clear mathematically why the stationary state must be stable to calculate the linear response. At an instability the lefthand matrix is not invertible!

1.2.2 Sparse Networks

Real cortical networks are sparse. How "promiscuously" cells couple with one another depends on the cell type (excitatory versus inhibitory), brain area, cortical layer, and probably animal species. The sparseness of connections between layer 2/3 pyramidal cells of mouse visual cortex is on the order of 10% [19] and in other cortical areas the sparseness is comparable. Inhibitory cells tend to be much close to the all-to-all limit in their connectivity, and between inhibitory interneurons of the same type one finds strong gap-junction (direct electrical coupling, a.k.a. a channel) coupling. But we won't model that here. Sparse networks pose the problem of how exactly to construct the network to begin with. The easiest solution, and the one that makes the fewest assumptions is to make a directed connection between any two cells independently with a probability p; this is known as the Erdös-Rényi (ER) network [1]. When you do this in a network of N neurons then each cell receives (and projects) on average $C = pN$ connections. However, the number of incoming (and outgoing) connections for any given cell will deviate from this. The distribution of these inputs and outputs, called "degrees", is binomial and has variance $\sigma^2 = p(1-p)N$. This distribution is very close to Gaussian for large N.

We can use, once again, precisely the same meanfield theory for sparse networks that we have used up to now. The only difference is once again in the mean and variance of the inputs. For the sake of simplicity let's assume for the meantime that each cell receives *exactly* $C = pN$ inputs, and we'll put the additional quenched variability back in in the next section. Then, the mean and variance to an excitatory neuron are

$$\mu_E = \tau_E\Big(J_{EE}C_{EE}\nu_{E0} - J_{EI}C_{EI}\nu_{I0}\Big), \qquad (\text{S.102})$$

$$\sigma_E^2 = \tau_E\Big(J_{EE}^2 C_{EE}\nu_{E0} + J_{EI}^2 C_{EI}\nu_{I0}\Big). \qquad (\text{S.103})$$

Note now that the fluctuations need not vanish as $N \to \infty$. In particular, we can keep the number of inputs constant, in which case, e.g. $\lim\limits_{N\to\infty} p_{EE} = \lim\limits_{N\to\infty} \frac{C_{EE}}{N_E} = 0$. This is known as the sparse limit. There another well known scaling for sparse networks for which, in the large system-size limit, the

---

[1] As it turns out real cortical networks are not ER networks [19, 14], but hey, you've got to start somewhere.

19

connectivity does not vanish. This is known as the "balanced network" limit, but we will not address it here [20]

1.3 Variability in Neuronal Networks

This has all been a lot of work just to calculate some fixed points and their linear stability. Why not use some low-dimensional model of neuronal activity, such as the Wilson-Cowan equations [21], which are much easier to analyze? Surely spiking networks must give us some additional insight into neuronal activity beyond fixed points and stability to be worth the effort. Indeed. The great success of the meanfield theory of spiking networks has been to describe a certain aspect of, in particular, cortical activity, which cannot be addressed otherwise, namely: variability [2]. In-vivo cortical activity is highly variable, that is one of its starkest features. Here we will focus on two types of variability: irregular spiking activity and broad firing rate distributions.

1.3.1 Irregular Spiking

If you crank the external noise level up in an all-to-all network you can get highly irregular spiking activity. However, this is not a particularly satisfying situation conceptually. Where is all that noise coming from? It turns out that in a sparsely connected network you get irregularity for free even with no noise injection, as well as broad firing rate distributions. We can already see that from Eq.S.103 which gives the noise level. That's funny, we inject no noise but the neurons are driven by i.i.d white noise processes. This arises from the assumption that spiking activity is Poisson and that p is small enough so that any two neurons only share a very small fraction of inputs.

A measure of the irregularity of the spiking activity in the coefficient of variation of the inter-spike interval (CV of ISI). Note that the mean ISI is just the average time it takes for the neuron to make it past threshold. This is known as the first passage time. For the LIF neuron, which is an Ornstein-Uhlenbeck process, the moments of the first passage time distribution follow a recursive relationship

$$\frac{\sigma^2}{2}\frac{d^2\mu_k}{dx^2} - (x-\mu)\frac{d\mu_k}{dx} = -k\mu_{k-1} \qquad \text{(S.104)}$$

---

[2]This is not to disparage the contribution of the theory for finding stationary states, and oscillatory instabilities!

When using this relation just remember once you integrate that x should be evaluated from V_r to V_t. The mean ISI itself is just $\mu_1 = 1/(\tau\nu_0)$. The coefficient of variation is

$$
\begin{aligned}
CV &= \frac{\mu_2 - \mu_1^2}{\mu_1}, \\
&= 2\pi\tau^2\nu_0^2 \int_{\frac{V_r-\mu}{\sigma}}^{\frac{V_t-\mu}{\sigma}} du\, e^{u^2} \int_{-\infty}^{u} dv\, e^{v^2} \left[\operatorname{erfc}(-v)\right]^2. \quad \text{(S.105)}
\end{aligned}
$$

For low firing rates the CV approaches a value of 1, which is precisely what one would obtain for a Poisson process which was our assumption for the meanfield theory. For high firing rates the CV approaches 0 which means very regular activity [4]. Despite this, the theory still works! For other types of neuron models there is no closed-form expression for the CV.

1.3.2 Distributions of firing rates

We can study the effect of quenched variability in sparse networks in a very general way and then see how that plays out specifically in the context of the meanfield theory we have developed so far. In a network in which each neuron receives a different *mean* input we can write

$$
I_{rec}(t) = \mu + \Delta z_i + \sigma\xi_i(t), \quad \text{(S.106)}
$$

where the μ and σ are just the mean and amplitude of temporal fluctuations from before ($\xi_i(t)$ is a white-noise process). But now z_i is a random variable with variance Δ^2 which does not change in time. This means that for a neuron with a given value of z, the steady state firing rate will be $\nu_0(z) = \phi(z)$. Let's assume that the distribution of z is written $f(z)$. Then the steady state firing rate distribution of the network as a whole is

$$
P(\nu_0) = \int_{-\infty}^{\infty} dz\, f(z)\delta(\nu_0 - \phi(z)), \quad \text{(S.107)}
$$

where $\delta(x)$ is the Dirac delta function. We can evaluate the integral explicitly with the change of variable $w = \phi(z)$, which leads to

$$
\begin{aligned}
P(\nu_0) &= \int_{0}^{\infty} dw \frac{f(\phi^{-1}(w))}{\phi'(\phi^{-1}(w))}\delta(\nu_0 - w), \\
&= \frac{f(\phi^{-1}(\nu_0))}{\phi'(\phi^{-1}(\nu_0))}. \quad \text{(S.108)}
\end{aligned}
$$

21

So we have the firing rate distribution in terms of the steady-state fI curve and the distribution of quenched variability. If the quenched variability is Gaussian with mean μ and variance Δ^2 we have

$$P(\nu_0) = \frac{1}{\sqrt{2\pi}\Delta}\exp\Big(-\frac{[\phi^{-1}(\nu_0) - \mu]^2}{2\Delta^2}\Big)[\phi'(\phi^{-1}(\nu_0))]^{-1}. \qquad \text{(S.109)}$$

Now consider two cases: i. The fI-curve is linear, i.e. $\phi(\mu) = A\mu + B$ and ii. The fI-curve is exponential, i.e. $\phi(\mu) = \bar{\nu}e^{\gamma\mu}$. It is straightforward to show that in the first case the firing rate distribution is Gaussian, while in the second case it is log-normal. *EXERCISE: Show this!* In real cortical networks in-vivo the firing rate distributions are, in fact, close to log-normal [17]. This means, according to this theory, that the fI curve smut be close to exponential, and this is the case only when the mean input is below threshold, hence mean firing rates are low, and spiking occurs due to fluctuations. This is known as the fluctuation-driven regime, where the CV is close to 1, and is precisely the operating point of cortical networks. This particular network mechanism linking the fluctuation-driven regime to broad firing-rate distributions through the skewing of Gaussian input distributions has actually been convincingly shown to hold in the turtle spinal cord [15].

How do we take this heterogeneity into account in the LIF network? The variance of the quenched variability in Eq.S.106 for an excitatory neuron in an EI network is

$$\Delta_E^2 = \tau_E^2[(1-p)(C_E\bar{\nu_E}^2 J_{EE}^2 + C_I\bar{\nu_I}^2 J_{EI}^2) + C_E\Delta\nu_E^2 J_{EE}^2 + C_I\Delta\nu_I^2 J_{EI}^2], \quad \text{(S.110)}$$

where $\bar{\nu_E}$ and $\Delta\nu_E^2$ are the mean and the variance of the excitatory rates respectively. The first term in Eq.S.110 arises due to the variability in the number of inputs to each cell (e.g. the variance of the excitatory in-degree is $p(1-p)N_E = (1-p)C_E$). It is this variability in inputs which gives rise to the distribution of firing rates. Once the firing rates broaden, they also, in turn, add to the variability. This is reflected in the second term in Eq.S.110. Now, we must calculate the mean and variance of the firing rate distribution self-consistently. For the excitatory rates this is

$$\bar{\nu_E} = \frac{1}{\sqrt{2\pi}}\int_{-\infty}^{\infty} dz\, e^{-z^2/2}\phi_E(z), \qquad \text{(S.111)}$$

$$\Delta\nu_E^2 = \frac{1}{\sqrt{2\pi}}\int_{-\infty}^{\infty} dz\, e^{-z^2/2}\phi_E^2(z) - \bar{\nu_E}^2. \qquad \text{(S.112)}$$

22

Finally, the full distribution is given by

$$P(\nu_E) = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{\infty} dz e^{-z^2/2} \delta(\nu_E - \phi_E(z)). \qquad \text{(S.113)}$$

The resulting distribution in the fluctuation-driven regime fits in-vivo cortical data quite well [17].

2 Networks of heterogeneous Quadratic Integrate-and-Fire neurons

In this second lecture we will switch gears considerably. The theory for E-I networks which we reviewed in the previous section was inspired by the type of activity seen *in-vivo* in cortical networks. The great success of the theory is that it not only captures some key macroscopic dynamical features of cortical circuits, such as oscillatory activity and persistent elevated activity (bistability), but also the fact that the microscopic states display the types of variability, such as irregular spiking, and broad firing rate distributions, which are canonical features of cortical circuits. Nonetheless the mean-field theory is technically challenging, and can only take us as far as the stationary states and their linear stability and response to small-amplitude inputs. We have no dynamical mean-field theory for arbitrary time-varying inputs for these networks. What is the non-stationary behavior of recurrent networks of spiking neurons? This is the most relevant dynamical regime because the brain is constantly subjected to non-stationary sensory inputs.

In this section we will consider a very specific type of spiking network: an all-to-all network of quadratic integrate-and-fire (QIF) neurons with quenched variability (no noise!). The microscopic state in such networks is not cortical-like, spiking is not irregular and fluctuation-driven. However, this network has the distinct advantage that we can derive an *exact* mean-field model which is valid for any arbitrary external input. The resulting mean-field model is a low-dimensional system of ODEs, which is simple and intuitive. We can bring standard mathematics tools to bear for the analysis of ODEs which means that stationary states and linear stability are trivial and we can go well beyond this in principle. Let's begin!

2.1 QIF neurons and Theta neurons

The QIF can be written

$$\tau \dot{V} = V^2 + I. \qquad (S.114)$$

A spike occurs at a time t_{spike} whenever $V(t_{\text{spike}}) = V_{\text{peak}}$ and the voltage is then reset to $V(t_{\text{spike}}^+) = V_{\text{reset}}$. This model can be seen simply as a phenomenological spiking model, but it also has a more rigorous mathematical pedigree. In fact, most biophysically "realistic" conductance-based spiking models can be formally reduced to Eq.S.114 close to threshold for spiking [8]. The reason is that the transition to spiking generically occurs via a saddle-node bifurcation of the voltage rest state, e.g. as the cell is depolarized. Once the rest state vanishes a large voltage excursion occurs (the spike), but the spiking currents conspire to reset the voltage and the system therefore once again finds itself in the vicinity of the threshold to spiking. The spike and reset, which is a global phenomenon in phase-space, is therefore dominated by the dynamics near threshold, which can be described locally by the normal form for a saddle-node bifurcation, Eq.S.114. This bifurcation has been called the "saddle-node bifurcation on an invariant circle" (SNIC).

Any one-dimensional spiking model can be recast as a phase oscillator. In the case of the QIF this can be done via the transformation $V = \tan(\theta/2)$, where $\theta \in [-\pi, \pi)$, as long as we take $V_{\text{peak}} = \infty$ and $V_{\text{reset}} = -\infty$. Taking the peak spike voltage and reset voltage to infinity is not a problem because the voltage reaches infinity in finite time in the QIF. Plugging this into Eq.S.114, and after a bit of algebra one finds

$$\tau \dot{\theta} = I + 1 + (I - 1)\cos(\theta). \qquad (S.115)$$

This form of the QIF is known as the "theta" model. What is gained by looking at the dynamics of the QIF in this form? For one, it eliminates the infinities from the dynamics, so they are now completely smooth; whenever the phase crosses π there is a spike. But the real advantage is to be had once we consider a network of coupled neurons. In that case it just turns out that being able to use periodic basis functions is very convenient.

2.2 An exact meanfield model for theta neurons [11, 18, 7]

Now we consider a large assembly of theta neurons. The dynamics of neuron i obeys

$$\tau\dot{\theta}_i = I_i + 1 + (I_i - 1)\cos(\theta_i), \qquad (S.116)$$

so we allow for the input current to be different from neuron to neuron, and it may depend on the activity of all other neurons as well. However, there is no noise. The probability of finding an oscillatory in an interval of phases $(\theta, \theta + d\theta)$ is $P(\theta, t)d\theta$ where

$$\frac{\partial P}{\partial t} + \frac{\partial F}{\partial \theta} = 0, \qquad (S.117)$$

which is just the continuity equation we considered for spiking networks in the preceding section, and the flux $F = \dot{\theta}P$. As before the firing rate is $r(t) = F(\pi, t)$, where I'm using r now instead of ν; this way whenever I use r you'll know I'm talking about the network of QIF neurons. The probability distribution can be expressed as a Fourier series,

$$P(\theta, t) = \frac{1}{2\pi}\left[1 + \sum_{n=1}^{\infty} z_n e^{in\theta} + \sum_{n=1}^{\infty} \bar{z}_n e^{-in\theta}\right], \qquad (S.118)$$

so that $\int_{-\pi}^{\pi} d\theta P(\theta, t) = 1$. We can plug this ansatz into Eq.S.117 and project the dynamics onto each Fourier mode. When you do this, the coefficients of all the Fourier modes become coupled; to make any progress one must truncate the Fourier series, which can be done rigorously e.g. if the amplitudes are small. This was the approach taken originally by Kuramoto in his classical work on collective synchronization [10] (the approach was the same but the model was not). However, in 2008, Ed Ott and Thomas Antonsen discovered one could make much more progress by considering a particular functional form for the different Fourier coefficients z_n [13], namely

$$P(\theta, t) = \frac{1}{2\pi}\left[1 + \sum_{n=1}^{\infty} z^n e^{in\theta} + \sum_{n=1}^{\infty} \bar{z}^n e^{-in\theta}\right]. \qquad (S.119)$$

Now when we plug this ansatz into Eq.S.117 we find that upon projecting onto each Fourier mode the coefficients decouple. Specifically, if we

project onto the kth Fourier mode by integrating the continuity equation as $\int_{-\pi}^{\pi} d\theta e^{-ik\theta} \cdot (...)$ we find that

$$\tau \dot{z} = -i(I+1)z - \frac{i}{2}(I-1)(1+z^2),\qquad\text{(S.120)}$$

which does not depend on k at all (if $k = 0$ we get identically zero). Therefore if the complex variable z, known as the Kuramoto order parameter, satisfies Eq.S.120, this is an exact solution to the continuity equation. Furthermore, the probability distribution Eq.S.119 can be simplified by noting that $\sum_{n=1}^{\infty} z^n e^{in\theta} = \sum_{n=1}^{\infty} \alpha^n = \frac{\alpha}{1-\alpha}$ as long as $|\alpha| < 1$. Then we have

$$P(\theta, t) = \frac{1}{2\pi}\Big[\frac{1-|z|^2}{(1-ze^{i\theta})(1-\bar{z}e^{-i\theta})}\Big].\qquad\text{(S.121)}$$

This is known as the Poisson kernel. So far, so good. Now we must deal with the currents in Eq.S.120. We will take them to be of the form $I_{total} = I_i + J\tau r(t) + I_{ext}(t)$, so that there is quenched variability, a meanfield input with weight J and a global external input. Because the inputs are distributed we must integrate over the probability distribution, which we call $f(I)$. Therefore we write

$$\tau \int_{-\infty}^{\infty} dx f(x)\dot{z} = -i\int_{-\infty}^{\infty} dx f(x)(x + Jr + I_{ext} + 1)z$$
$$-\frac{i}{2}\int_{-\infty}^{\infty} dx f(x)(x + Jr + I_{ext} - 1)(1 + z^2).$$

This is the full model and can be analyzed via typical methods. It does, however, simplify if we allow the function $f(x)$ to have a simple pole in the complex plane. In that case the integrals are all equal to the argument evaluated at the pole by the Cauchy residue theorem. Magic! Unfortunately there aren't any "normal" distributions with simple poles but for mathematical convenience we will take one anyway, the Cauchy or Lorentz distribution, which looks like $f(x) = \frac{1}{\pi}\frac{\Delta}{(x-\bar{I})^2+\Delta^2}$ and has very broad tails. Then, the macroscopic equations simplify to

$$\tau \dot{z} = -i(\bar{I} + Jr + I_{ext} + 1)z - \frac{i}{2}(\bar{I} + Jr + I_{ext} - 1)(1 + z^2).\qquad\text{(S.122)}$$

This ODE is really two coupled ODEs because the Kuramoto order parameter z is a complex variable. It is typical to consider the amplitude and phase of

26

$z = Re^{i\psi}$. This makes a lot of sense in studies of coupled oscillators because oftentimes one is interested in synchronized states for which $R = 1$. Here we should also note that the firing rate can be written as

$$
\begin{aligned}
r(t) &= F(\pi, t), \\
&= 2P(\pi, t) \\
&= \frac{1}{\pi\tau} \frac{1 - |z|^2}{(1+z)(1+\bar{z})} \\
&= \frac{1}{\pi\tau} \frac{1 - R^2}{1 + 2R\cos\psi + R^2}.
\end{aligned}
\tag{S.123}
$$

Finally we can write

$$
\begin{aligned}
\tau\dot{R} &= -\Delta R - \frac{\Delta}{2}(1 + R^2)\cos\psi \\
&\quad - \left(\bar{I} + \frac{J\tau}{\pi}\frac{1 - R^2}{1 + 2R\cos\psi + R^2}\right)\frac{(1 - R^2)}{2}\sin\psi, \tag{S.124} \\
\tau\dot{\psi} &= -\frac{1}{2R}\left(\bar{I} + \frac{J\tau}{\pi}\frac{1 - R^2}{1 + 2R\cos\psi + R^2}\right)(2 + (1 + R^2)\cos\psi) \\
&\quad + \frac{\Delta}{2R}(1 - R^2)\sin\psi. \tag{S.125}
\end{aligned}
$$

There might be some typos in there, it's a long formula.

2.3 An exact meanfield model for QIF neurons

Of course, Eqs.S.124-S.125 *are* an exact meanfield model for QIF neurons since theta neurons are just QIF neurons transformed. But how do I relate R and ψ to firing rates and voltages? Do I just use the relationship $V = \tan\theta/2$ somehow? No, that's for single cells, here I'm talking about transforming some macroscopic variable to some other macroscopic variables. The answer is not obvious [12]. The solution is to realize that the firing rate (which is what we care about) can be written as the real part of a complex variable which is a particular transformation of the Kuramoto order

parameter. Specifically, consider the variable w, where

$$\begin{aligned}
w &= \frac{1-z}{1+z}, \\
&= \frac{1-|z|^2}{(1+z)(1+\bar{z})} - \frac{z-\bar{z}}{(1+z)(1+\bar{z})}, \\
&= \pi\tau r(t) + iy,
\end{aligned} \tag{S.126}$$

where we don't yet know what y is, but we can find out! Now that we have the relationship between w and z and between θ and V we can transform the Poisson kernel Eq.S.121 to find

$$P(V,t) = \frac{\tau r(t)}{(V-y)^2 + \pi^2\tau^2 r^2(t)}, \tag{S.127}$$

which we can all the "Lorentz" kernel. It's now clear that y is the mean (actually the mid-point or mean in the Cauchy Principal Value sense) of the distribution of voltages. So $w = \pi\tau r(t) + iv(t)$. We can transform Eq.S.120 to find

$$\tau\dot{w} = i(I - w^2). \tag{S.128}$$

And finally, using the same trick of taking the distributed inputs from a Lorenz distribution we can write Eq.S.128 as two coupled ODEs

$$\begin{aligned}
\tau\dot{r} &= \frac{\Delta}{\tau\pi} + 2rv, \tag{S.129} \\
\tau\dot{v} &= v^2 + \bar{I} + J\tau r + I_{ext} - \pi^2\tau^2 r^2. \tag{S.130}
\end{aligned}$$

Let me emphasize that Eqs.S.129-S.130 and Eqs.S.124-S.125 describe *identical* dynamics. Nonetheless it is my opinion that Eqs.S.129-S.130 are much more helpful and intuitive when investigating neuronal activity as I will illustrate in the next section. They certainly are simpler! BTW, what does the transformation $w = (1-z)/(1+z)$ mean? It is a conformal mapping which maps the unit circle (amplitude and phase) onto the half plane (voltage and firing rate).

2.3.1 Alternative derivation of the meanfield model for QIF neurons

Let's analyze the continuity equation for a networks of QIF neurons directly.

$$\frac{\partial P}{\partial t} + \frac{\partial F}{\partial V} = 0, \tag{S.131}$$

28

where $F = P(V,t)\frac{(V^2+I)}{\tau}$. Now, the steady state distribution is given by $F_0(V) = C$ where C is a constant. There can only be the case if $P_0(V) = \frac{C}{V^2+I}$. This is a Lorentz distribution with $C = \sqrt{I}/\pi$. Note also that the steady state firing rate

$$r_0 = \lim_{V \to \infty} F_0(V) = \frac{\sqrt{I}}{\pi\tau}. \tag{S.132}$$

This means we can write

$$P_0(V) = \frac{\tau r_0}{(V - v_0)^2 + \pi^2 \tau^2 r_0^2}, \tag{S.133}$$

where $v_0 = 0$ is the "mean" membrane potential. This really is the stationary distribution of voltages. However, it suggests that we might want to take it as an ansatz for the full, time-dependent solution with $r(t)$ and $v(t)$ as time-varying parameters. In hindsight we know that this is the correct solution. We can plug this ansatz into the continuity equation, which will lead to two coupled ODEs, Eqs.S.129-S.130.

2.3.2 Analysis of the meanfield model for QIF

You may be familiar with so-called firing rate models (or Wilson-Cowan equations) or neural mass models. These generally have the form

$$\tau \dot{r} = -r + \phi(I), \tag{S.134}$$

where the steady state is given by $r_0 = \phi(I)$. Note that this is precisely the form of the steady state from the meanfield theory of spiking networks. So such heuristic firing rate models assume some simple first order relaxation to a steady state which can be made to match the true steady state of any given spiking network. What about Eqs.S.129-S.130 (I'll call this the QIF meanfield)? They look quite different from Eq.S.134. But this is actually just a consequence of the explicit dependence on the subthreshold variable v. In fact, if we solve for the fixed points in the QIF meanfield, we find that

$$v_0 = -\frac{\Delta}{2\pi\tau r_0}, \tag{S.135}$$

$$r_0 = \phi(I), \tag{S.136}$$

where $\phi(I) = \frac{1}{\sqrt{2\pi}}\sqrt{I + \sqrt{I^2 + \Delta^2}}$, and $I = \bar{I} + J\tau r_0 + I_{ext,0}$. This ϕ looks similar to typical choices for fI curves: it has an expansive nonlinearity for

29

low rates and a compressive one for high rates. But note that as opposed to your typical firing rate model for a single neuronal population, the QIF meanfield model has two independent dynamical variables. So while the fixed point structure is similar the dynamics need not be.

One advantage of the simplicity of the QIF meanfield model is that we can gain some insight into the dynamics just by looking at the equations. For example, imagine that a large depolarizing current arrives all at once to the whole network. This means I_{ext} is increasing suddenly. This will drive up v, to positive values if the input is large enough. Once v is positive, we see from Eq.S.129 that the term $2rv$ will be positive and so the mean rate will also grow rapidly. However, once r is large enough, the negative feedback term in Eq.S.130, proportional to r^2 will drive v back down. Once v is negative r will go down once again, allowing v to increase...This is a mechanism for generating oscillations. Also note that the key terms in the QIF meanfield model are the quadratic ones, the ones related to spike generation and reset. This means that spike generation mechanism in the networks of QIF neurons will result in oscillatory behavior in response to a sudden depolarizing input: these oscillations reflect an underlying network-wide spike synchrony. This is what makes the QIF meanfield model so different from other meanfield models, namely it captures spike synchrony.

Let's back up this intuition with some mathematics. We can study the linear stability of the fixed point solution by taking the ansatz $(r, v) = (r_0, v_0) + (\delta r, \delta v)e^{\lambda t}$, where δr, $\delta v \ll 1$. Plugging this into Eqs.S.129-S.130 yields

$$\begin{pmatrix} \tau\lambda - 2v_0 & -2r_0 \\ -J\tau + 2\pi^2\tau^2 r_0 & \tau\lambda - 2v_0 \end{pmatrix} \begin{pmatrix} \delta r \\ \delta v \end{pmatrix} = 0. \tag{S.137}$$

This system has a solution if and only if

$$\tau\lambda = -2|v_0| \pm \sqrt{2\tau r_0(2\pi^2\tau r_0 - J)}. \tag{S.138}$$

Note that for strong enough coupling $J > 2\pi^2 r_0$ this becomes

$$\tau\lambda = -2|v_0| \pm i\sqrt{2\tau r_0(J - 2\pi^2\tau r_0)}. \tag{S.139}$$

and so the fixed point is a stable focus. The damped oscillations that occur when this focus is perturbed are precisely those we described heuristically in the preceding paragraph.

2.3.3 Variants

I will not go into any details in these notes, I just wanted to mention that this methodology works for several populations of neurons, e.g. E and I, in the continuum limit, e.g. neural fields, for conductances as synaptic inputs and for gap junctions. Also, we can distribute the synaptic weights instead of the input currents.

References

[1] M. Abramowitz and I. A. Stegun. *Tables of mathematical functions.* Dover Publications, NY, 1970.

[2] D. J. Amit and N. Brunel. Dynamics of a recurrent network of spiking neurons before and following learning. *Network*, 8:373–404, 1997.

[3] D. J. Amit and N. Brunel. Model of global spontaneous activity and local structured activity during delay periods in the cerebral cortex. *Cerebral Cortex*, 7:237–252, 1997.

[4] N. Brunel. Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons. *J. Comput. Neurosci.*, 8:183–208, 2000.

[5] N. Brunel and V. Hakim. Fast global oscillations in networks of integrate-and-fire neurons with low firing rates. *Neural Comp.*, 11:1621–1671, 1999.

[6] N. Brunel and P. Latham. Firing rate of noisy quadratic integrate-and-fire neurons. *Neural Computation*, 15:2281,2306, 2003.

[7] S. Coombes and A. Byrne. Next generation neural mass models. In *Lecture notes in nonlinear dynamics in computational neuroscience: from physics and biology*, 2016.

[8] G. B. Ermentrout and N. Kopell. Parabolic bursting in an excitable system coupled with a slow oscillation. *SIAM J. Appl. Math.*, 46:233–253, 1986.

[9] S. Fusi and M. Mattia. Collective behavior of networks with linear (VLSI) integrate and fire neurons. *Neural Computation*, 11:633–652, 1999.

[10] Y. Kuramoto. *Chemical Oscillations, Waves and Turbulence.* Springer-Verlag, New York, 1984.

[11] T. B. Luke, E. Barreto, and P. So. Complete classification of the macroscopic behavior of a heterogeneous network of theta neurons. *Neural Comput.*, 25:3207, 2013.

[12] E. Montbrió, D. Pazó, and A. Roxin. Macroscopic description for networks of spiking neurons. *Phys. Rev. X*, 5, 2015.

[13] E. Ott and T. M. Antonsen. Low-dimensional behavior of large systems of globally coupled oscillators. *Chaos*, 18:037113, 2008.

[14] R. Perin, T. K. Berger, and H. Markram. A synaptic organizing principle for cortical neuronal groups. *PNAS*, 108:5419–5424, 2011.

[15] P. C. Petersen and R. W. Berg. Lognormal firing rate distribution reveals prominent fluctuation-driven regime in spinal motor networks. *eLife*, 5:e22735, 2016.

[16] M. J. E. Richardson. Firing-rate response of linear and nonlinear integrate-and-fire neurons to modulated current-based and conductance-based synaptic drive. *Phys. Rev. E*, 76:021919, 2007.

[17] A. Roxin, N. Brunel, D. Hansel, G. Mongillo, and C. van Vreeswijk. On the distribution of firing rates in networks of cortical neurons. *J. Neurosci.*, 31:16217–16226, 2011.

[18] P. So, T. B. Luke, and E. Barreto. Networks of theta neurons with time-varying excitability: Macroscopic chaos, multistability, and final-state uncertainty. *Physica D*, 296:16–26, 2014.

[19] S. Song, P. J. Sjöström, M. Reigl, S. Nelson, and D. B. Chklovski. Highly nonrandom features of synaptic connectivity in local cortical circuits. *PLoS Biology*, 3:507–519, 2005.

[20] C. van Vreeswijk and H. Sompolinsky. Chaotic balanced state in a model of cortical circuits. *Neural Computation*, 10:1321–1371, 1998.

[21] H. R. Wilson and J. D. Cowan. Excitatory and inhibitory interactions in localized populations of model neurons. *Biophys. J.*, 12:1–24, 1972.