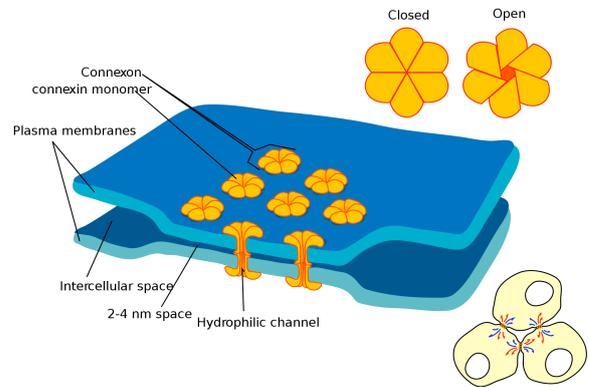


Weak gap junction coupling

Gap junctions allow for direct communication between cells. They are typically formed from the juxtaposition of two hemichannels (connexin proteins) and allow the free movement of ions or molecules across the intercellular space separating the plasma membrane of one cell from another. Gap junction coupling is known to occur between many cell types, including, for example, pancreatic- β cells, heart cells, and astrocytes. It is no understatement to say that they are now believed to be ubiquitous throughout the central nervous system. Indeed it has been



appreciated for some time that they exist between inhibitory neurons of the neocortex. As well as being found in the neocortex, they occur in many other brain regions, including the hippocampus, inferior olivary nucleus in the brain stem, the spinal cord, and the thalamus, and have recently been shown to form axo-axonic connections between *excitatory* cells in the hippocampus (on mossy fibers). Without the need for receptors to recognize chemical messengers, gap junctions are much faster than chemical synapses at relaying signals. The synaptic delay for a chemical synapse is typically in the range 1–100 ms, while the synaptic delay for an electrical synapse may be only about 0.2 ms. There is now little doubt that gap junctions play a substantial role in the generation of neural rhythms, both functional and pathological, and that they may subserve system level computations.

For a recent discussion see

S Coombes 2008 Neuronal networks with gap junctions: A study of piece-wise linear planar neuron models, SIAM Journal on Applied Dynamical Systems, Vol 7, 1101-1129

It is common to view the gap-junction as nothing more than a channel that conducts current according to a simple ohmic model. For two neurons with voltages v_i and v_j the current flowing into cell i from cell j is given by

$$I_{\text{gap}}(v_i, v_j) = g(v_j - v_i).$$

Here g is the constant strength of the gap junction conductance.

As an example consider two QIF neuron models with gap-junction coupling:

$$\begin{aligned}\dot{v}_1 &= 1 + v_1^2 + I_{\text{gap}}(v_1, v_2), \\ \dot{v}_2 &= 1 + v_2^2 + I_{\text{gap}}(v_2, v_1).\end{aligned}$$

For simplicity we shall take the threshold to be at $+\infty$ and the reset to be at $-\infty$.

The uncoupled orbit (obtained using $g = 0$) can easily be obtained in closed form (see notes on nonlinear IF models):

$$t = \int_{-\infty}^{v(t)} \frac{du}{1 + u^2} = \tan^{-1} v(t) + \frac{\pi}{2}.$$

The period of oscillation, Δ , is calculated from the threshold condition $v(T) = \infty$, giving $T = \pi$. Introducing a phase $\theta = t/\Delta \equiv \Psi(v)$ means that we can rewrite the voltage dynamics in an equivalent set of phase variables as

$$\begin{aligned}\dot{\theta}_1 &= \frac{1}{\Delta} + gR(\theta_1)(v_1(\theta_1) - v_2(\theta_2)), \\ \dot{\theta}_2 &= \frac{1}{\Delta} + gR(\theta_2)(v_2(\theta_2) - v_1(\theta_1)),\end{aligned}$$

where $R(\theta)$ is the PRC and we are careful to write the voltage trajectories in terms of the phase variables. The trajectory takes the form

$$v = \tan(\theta\Delta - \pi/2) = -\cot(\theta\Delta) \equiv \Psi^{-1}(\theta),$$

and the PRC (see notes on PRC for nonlinear IF models) is given by

$$\begin{aligned}R(\theta) &= \frac{1}{\Delta} \frac{1}{F \circ \Psi^{-1}(\theta)}, \quad F(v) = 1 + v^2. \\ &= \frac{1}{\Delta} \frac{1}{1 + \cot^2(\theta\Delta)} \\ &= \frac{1}{\Delta} \sin^2(\theta\Delta).\end{aligned}$$

The gap-junction current between two neurons phase shifted by an amount χ can be written $g(v(t + \chi\Delta) - v(t))$ (with a common trajectory $v(t)$) and so the phase-interaction function (see notes on weakly coupled oscillators) is

$$H(\chi) = \frac{1}{\pi} \int_0^\pi \sin^2(t) [\cot(t) - \cot(t + \chi\Delta)] dt = \frac{1}{2} \sin 2\chi\Delta.$$

Hence the phase difference $\phi = \theta_2 - \theta_1$ satisfies

$$\dot{\phi} = g[H(-\phi) - H(\phi)] = -g \sin(2\phi\Delta).$$

Hence the synchronised state ($\phi = 0$) is always stable while the anti-phase state ($\phi = 1/2$) is not.

$$\begin{aligned}\int_0^\pi \sin^2(t) \cot(t) dt &= \int_0^\pi \sin(t) \cos(t) dt = \frac{1}{2} \int_0^\pi \sin(2t) dt = 0. \\ \int_0^\pi \sin^2(t) \cot(t + a) dt &= \int_a^{\pi+a} \sin^2(t - a) \cot(t) dt = \int_a^{\pi+a} [\sin(t) \cos(a) - \sin(a) \cos(t)]^2 \cot(t) dt \\ &= 0 - 2 \sin(a) \cos(a) \int_a^{\pi+a} \sin(t) \cos(t) \cot(t) dt + 0 = -\sin(2a) \int_a^{\pi+a} \cos^2(t) dt = -\frac{\pi}{2} \sin(2a).\end{aligned}$$