Synchrony: Delayed inhibition is key

Period proportional to rise-time (linear fit plus offset); purple-mean interneuron period.

Period is twice the delay, which is the sum of two terms:

Rise-time's contribution is half of the rise-time.

Decay-constant's contribution is up to a quarter of the period.
Asynchronous state

The two steady-state curves’ intersection determines the asynchronous state.

In the fully-connected network, the neurons receives the same amount of inhibition, \( G \). In the absence of heterogeneity, they fire at the same rate \( f(G, r) \), where \( r \) is the common excitatory drive.

The network activity \( A \)—equal to \( Nf(G, r) \) for \( N \) neurons—determines the inhibitory conductance \( G(A) \) (similar to a single adaptive neuron), which in turn determines the network activity \( A(G) \).

In the *asynchronous state*, network activity remains constant, at a level \( c \) that satisfies:

\[
    c = N f[G[c], r]
\]

Delay destabilizes asynchronous state

![](image)
Inhibition impacts activity immediately; activity impacts inhibition with a delay \((d)\).

These deviations \((a(t) \text{ and } g(t))\) from the asynchronous-state \((c \text{ and } G(c))\) are related by:

\[
a[t] = -m \ g[t] \text{ and } g[t] = s \ a[t - d] \Rightarrow a[t] = -(m \ s) \ a[t - d]
\]

where \(m\) and \(s\) are the steady-state-curves' slopes at \(A = c\).

Thus, deviations grow if \(m \ s > 1\), destabilizing the asynchronous state.

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**Period and amplitude of network rhythm**

Inhibition overshoots and undershoots repeatedly.

We assume \(a(t) = A_0 \sin(2 \pi t / T)\) and solve for \(A_0\) and \(T\):

\[
A_0 \sin\left(\frac{2 \pi t}{T}\right) = -(m \ s) A_0 \sin\left(\frac{2 \pi (t - d)}{T}\right)
\]

\[
= \ (m \ s) A_0 \sin\left(\frac{2 \pi (t - d)}{T}\right) + \pi
\]

\[
\Rightarrow T = 2 \ d \ \text{and} \ m \ s = 1
\]

The second condition determines \(A_0\): the amplitude grows if \(m \ s > 1\) and shrinks if \(m \ s < 1\).
Both rise-time and decay-constant contribute delay

How long does it take for half the input to show up?

If the decay-constant is fast, the delay is half the rise-time (i.e., neurotransmitter pulse's width).

If the decay-constant is slow, the delay is longer, because the input is smeared out.

However, the rise-time's contribution is still $\frac{t_{\text{rise}}}{2}$; a frequency-domain analysis shows this.

Decay-constant's contribution

The maximum delay is a quarter-period when the rise-time is zero.

When the decay-constant is very slow, inhibition either reflects activity's integral:

$$\int \sin \left( \frac{2\pi t}{T} \right) dt \propto -\cos \left( \frac{2\pi t}{T} \right) = \sin \left( \frac{2\pi t}{T} - \frac{\pi}{2} \right) = \sin \left[ \frac{2\pi}{T} \left( t - \frac{T}{4} \right) \right]$$

Thus, the longest delay is a quarter of the period. Adding this to the rise-time's contributions yields:

$$\frac{t_{\text{rise}}}{2} < d < \frac{t_{\text{rise}}}{2} + \frac{T}{4}$$
Doubling the delay gives the period, which falls in the range:

\[ t_{\text{rise}} < T < 2 \ t_{\text{rise}} \]

How inhibition is phase-shifted by 180°

Delays due to rise-time (green), decay-constant (blue), and their sum (red)

The rise-time contributes \(2 \pi f d\) — the delay normalized by the period (in radians).

The decay-constant contributes \(\tan^{-1}(2 \pi f \tau_k)\) — which cannot exceed 90°.

There is a unique frequency \(f\) that makes these two contributions sum to 180°.

This is the frequency that the network synchronizes at.

Next week: Attention