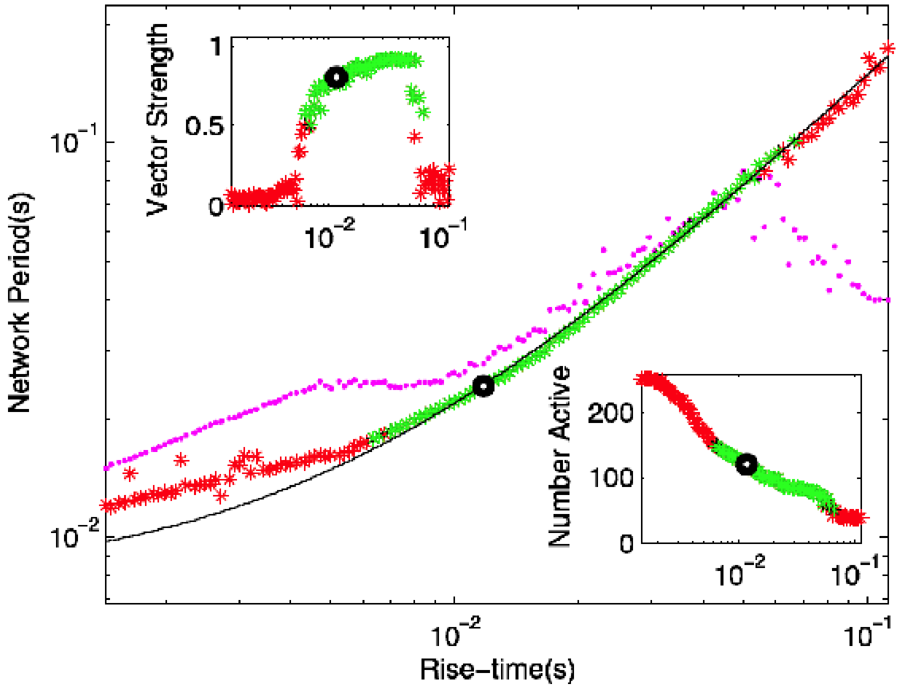
Synchrony: Delayed inhibition is key



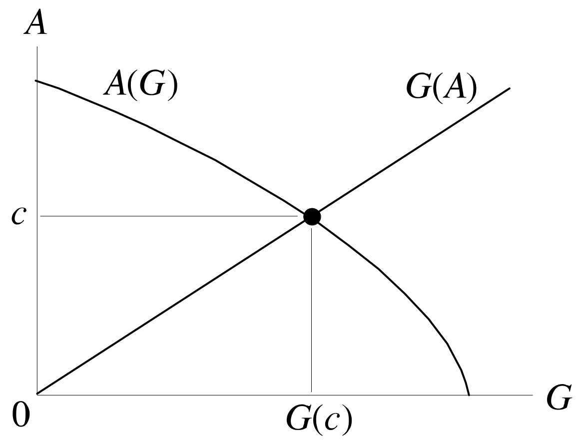
Period proportional to rise-time (linear fit plus offset); *purple*–mean interneuron period [Arthur07].

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

Rise-time contributes half of the rise-time.

Decay-constant contributes 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, and fire at the rate $A(G) = N\, f(G, r)$, where N is the number of neurons, $f(G, r)$ is their individual firing-rate curve, and r is their common excitatory drive (no heterogeneity).

The network activity A 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:

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c = N f[G[c], r]
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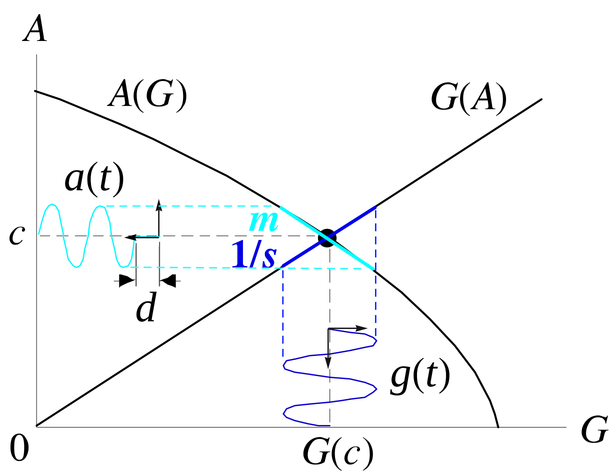
Delay destabilizes asynchronous state



Changes in inhibition ($g(t)$) impact activity ($a(t)$) immediately; changes in activity impact inhibition with a delay (d).

These deviations ($a(t)$ and $g(t)$) from the asynchronous-state (c and $G(c)$) are related by:

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

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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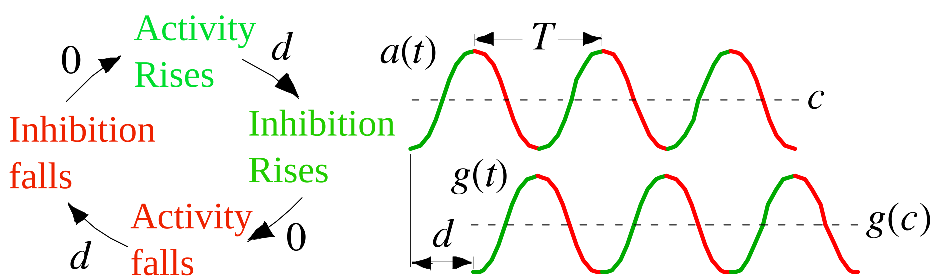
Period and amplitude of network rhythm



Inhibition overshoots and undershoots repeatedly.

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

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

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

$$\Longrightarrow -\frac{2\pi d}{T} + \pi = 0 \Longrightarrow T = 2d$$

$$\text{and } m\, s = 1$$

The second condition determines A_0: the amplitude grows if $m\, s > 1$ and shrinks if $m\, s < 1$.

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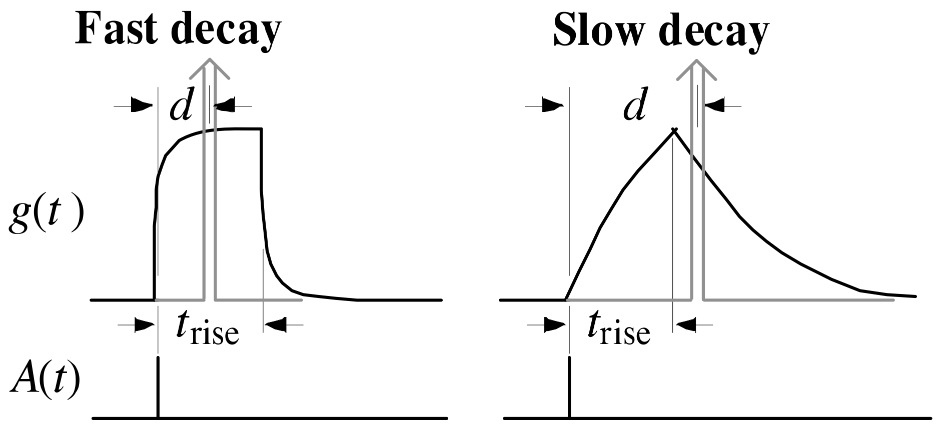
Both rise-time and decay-constant contribute delay



How long does it take for half the inhibition 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 $t_{rise}/2$; a frequency-domain analysis shows this.

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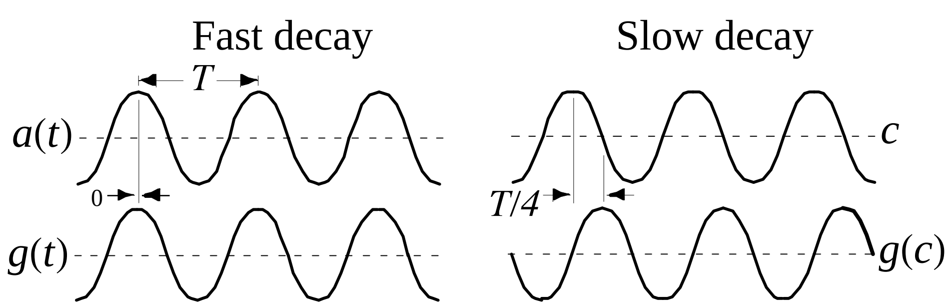
Decay-constant's contribution ($t_{rise} = 0$)



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

When the decay-constant is very slow, inhibition is the integral of activity:

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

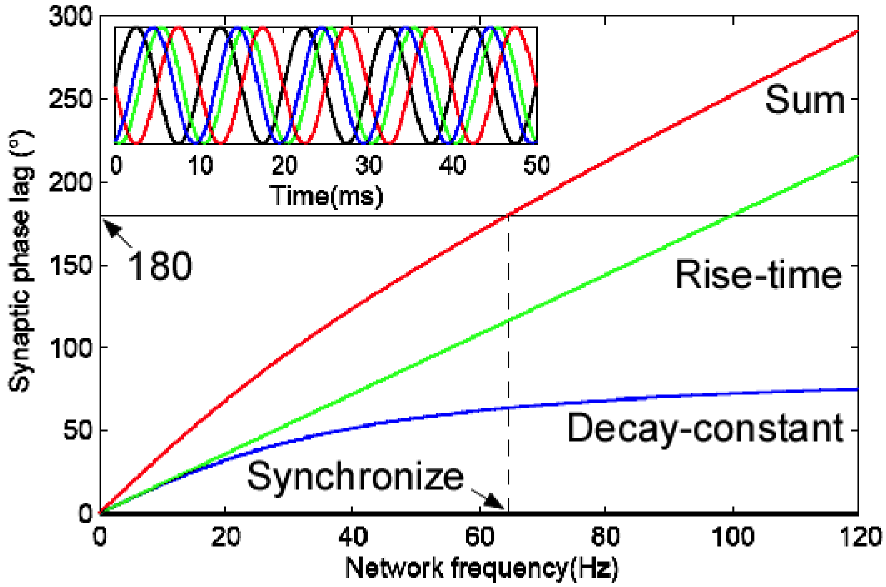
Thus, the longest delay is a quarter of the period. Adding this to the rise-time's contributions yields:

$$\frac{t_{rise}}{2} < d < \frac{t_{rise}}{2} + \frac{T}{4}$$

Doubling the delay gives the period, which falls in the range:

$t_{rise} < T < 2\, t_{rise}$

How inhibition is delayed by $T/2$ (180° lag)



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

The rise-time contributes $2\pi\left(\frac{t_{rise}}{2}\middle/T\right)$—the delay normalized by the period (in radians).

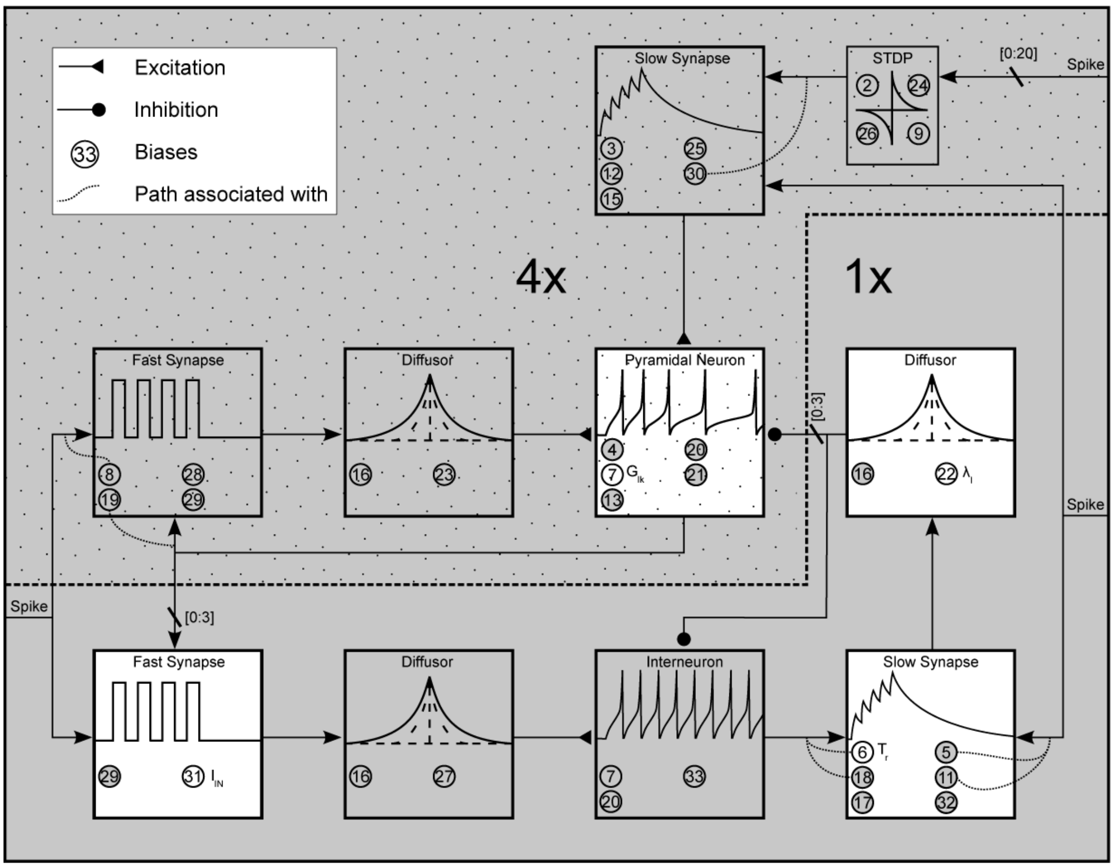
The decay-constant contributes $\tan^{-1}(2\pi\,\tau_{decay}/T)$—which cannot exceed 90 °.

There is a unique frequency $f = 1/T$ that makes these two contributions sum to 180°.

This is the frequency that the network synchronizes at.

Lab 5: Set-up

1	Vrefd2a	reference voltage - leave at 2.530
2	VQAPRE	(-) increases LTP-side of STDP curve's height
3	VLEAKDNMDA	(+) increases slow excitatory synapse rise-time
4	VMAGK	(-) increases m-type potassium channel strength
5	VLEAKDGABA2	(+) increases inhibitory synapse rise-time (external input)
6	VLEAKDGABA	(+) increases inhibitory synapse rise-time (inhibitory interneuron input)
7	VLEAKSOMA	(+) increases somatic leak current
8	VQAAMPA	(-) increases fast excitatory synapse strength pulse-width (external input)
9	VQAPOST	(-) increases LTD-side of STDP curve's height
10	VLEAKLTP	not used
11	VMAGGABA2	(-) increases inhibitory synapse strength (external input)
12	VQADNMDA2	(+) increases slow excitatory synapse rise-time
13	VANP	(+) increases pyramidal neuron sodium threshold
14	VLEAKLTD	not used
15	VLEAKNMDA	(+) increases slow excitatory synapse decay-constant (and strength)
16	VG	leave at 1.250
17	VLEAKGABA	(+) increases inhibitory synapse decay-constant (and strength)
18	VMAGGABA	(-) increases inhibitory synapse strength (inhibitory interneuron input)
19	VQAAMPA2	(-) increases fast excitatory synapse strength pulse-width (pyramidal neuron input)
20	VLEAKREFRACT	(+) increases absolute refractory period
21	VLEAKK	(+) increases m-type potassium decay-constant (and strength)
22	VRI	(+) increases spread of inhibition
23	VRRC	(+) increases spread of fast excitation to pyramidal neurons
24	VLEAKPOST	(+) increases LTP-side of STDP curve's decay
25	VMAGNMDA	(-) increases slow excitatory synapse strength
26	VLEAKPRE	(+) increases LTD-side of STDP curve's decay
27	VRE	(+) increases spread of fast excitation to interneurons
28	VMAGAMPARC	(+) increases fast excitatory synapse strength to pyramidal neurons
29	VLEAKAMPA	(-) increases fast excitatory synapse strength pulse-width
30	VQANMDA	(+) increases slow excitatory synapse rise-time
31	VMAGAMPAINT	(+) increases fast excitatory synapse strength to interneurons
32	VQADGABA	(+) increases inhibitory synapse rise-time
33	VANI	(+) increases interneuron sodium threshold



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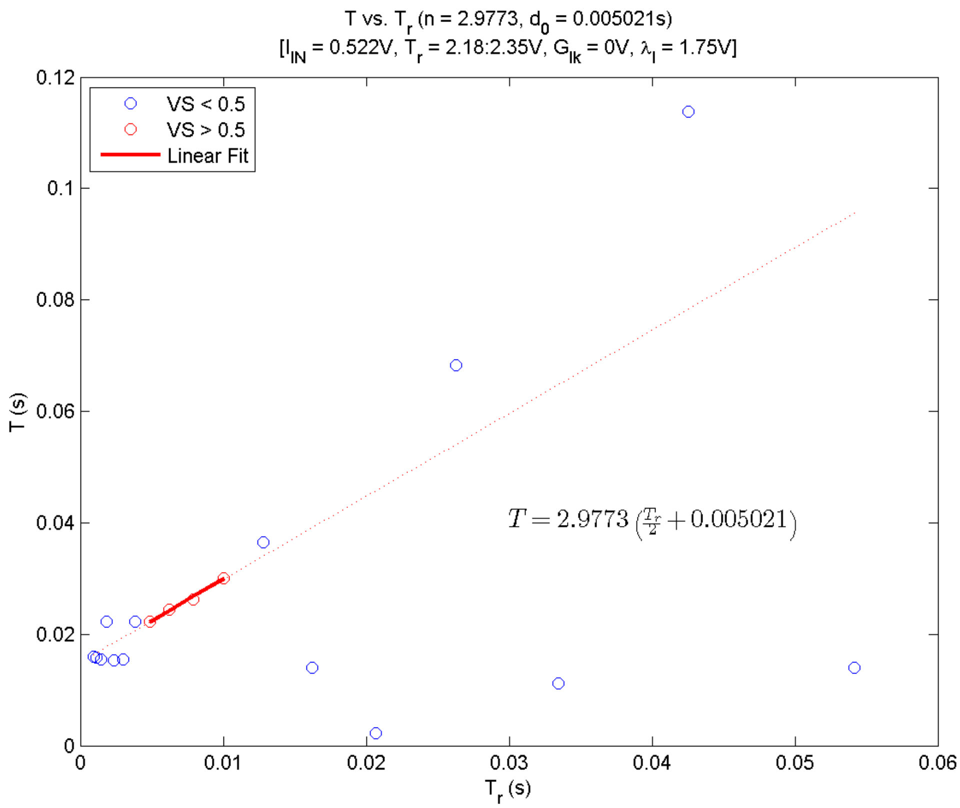
Lab 5: Data



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Next week: Attention