Synchrony: Inhibition's rise-time is key

Period is proportional to rise-time (linear fit includes offset); purple, mean interneuron period
Inhibition's strength & decay-constant

These parameters do not change the period as much as the rise-time

Amount of inhibition \( (g_K(t_\infty)) \)

The rate \( (1/\Delta) \) at which a neuron receives spikes determines its inhibition

If a spike arrives every \( \Delta \) seconds, the inhibition in steady-state is:

\[
g_K [t_\infty, \Delta] = \frac{\Delta g_K}{g_0/\tau_k - 1} = \frac{\Delta g_K}{(\Delta/\tau_k + 1) - 1} \quad \text{for} \quad \Delta \ll \tau_k
\]

(identical to adaptive neuron's \( K^+ \) conductance). Hence

\[
g_K [t_\infty, A] = \Delta g_K \tau_K A
\]

where \( A = 1/\Delta \) is the spike rate; \( A \) equals the total network activity when connectivity is all-to-all.
Asynchronous state

The network rate $c$ produces inhibition $g(c)$ that makes each neuron fire at $c/N$.

The network activity $A$ gives us the conductance, $g(A)$.

The conductance $g(A)$ gives us each neuron's firing rate, $f(g, r)$.

Multiplying $f(g, r)$ by the population size $N$ gives us the activity $A$.

The $A$ we get is the same as the one we started with if

$$A = N \, f[g(A), r]$$

This is the activity level (labeled $c$ above) in the asynchronous state—neurons distribute their spikes uniformly in time.

Inhibition's rise ($t_p$)

The rise time $t_p$ of $g_K(t)$ is the time it takes for the inhibition to reach its steady-state value $g_K(t_{\text{inf}})$.
A \( t_p \)-second synaptic rise-time is like a \( t_p / 2 \)-second delay (dashed line).
The synaptic rise-time delays the effect of inhibition by about half the rise-time.

In this approximation, \( g_K(t) \) is just a delayed version of \( A(t) \):

\[
g_K[t] = \Delta g_K \tau_K A[t - d]
\]

where \( d \approx t_p / 2 \).

### Delay destabilizes asynchrony

Inhibition overshoots and undershoots repeatedly.

Due to \( f(g, r) \)'s negative slope, excitation falls as inhibition rises—and vise versa—they are exactly half-a-cycle (180°) out-of-phase.

Therefore, the network activity oscillates with a period:

\[
T = 2d \approx t_p
\]
The decay-constant $\tau_K$ smears inhibition in time

By smearing inhibition is time, the decay-constant delays inhibition further—it peaks later.

This makes the period longer than it is without the decay-constant.

But this effect can only increase the period by a factor of two:

$$t_p < T < 2t_p$$

Phase-shifting inhibition

Adding the phase-shifts due to the rise-time and decay-constant yields the actual phase-shift. Both contributions change with the network frequency $f$.

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

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

Thus, the rise-time must contribute at least 90 of the 180°s required.