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Dynamical relaying can yield zero time lag neuronal synchrony despite long conduction delays

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Computational & theoretical neuroscience

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2008 November 4

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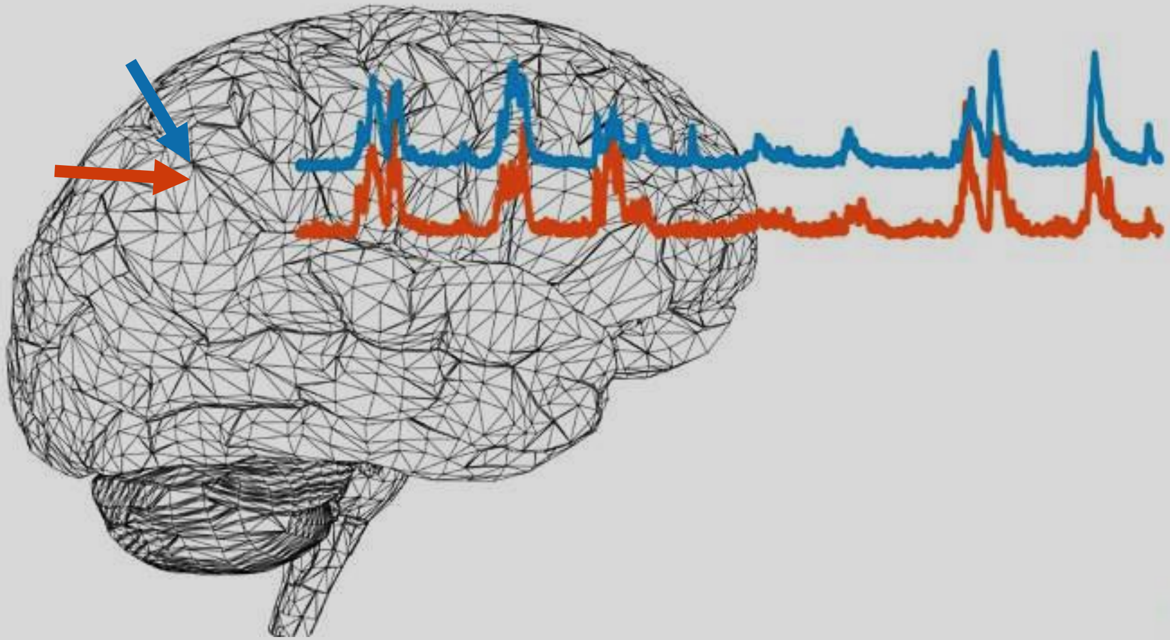
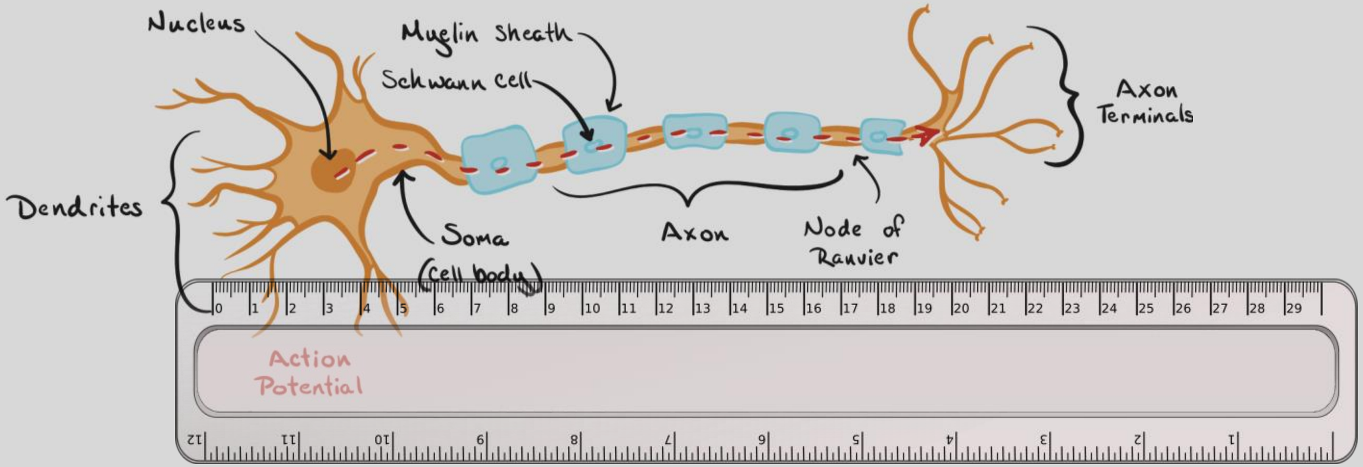
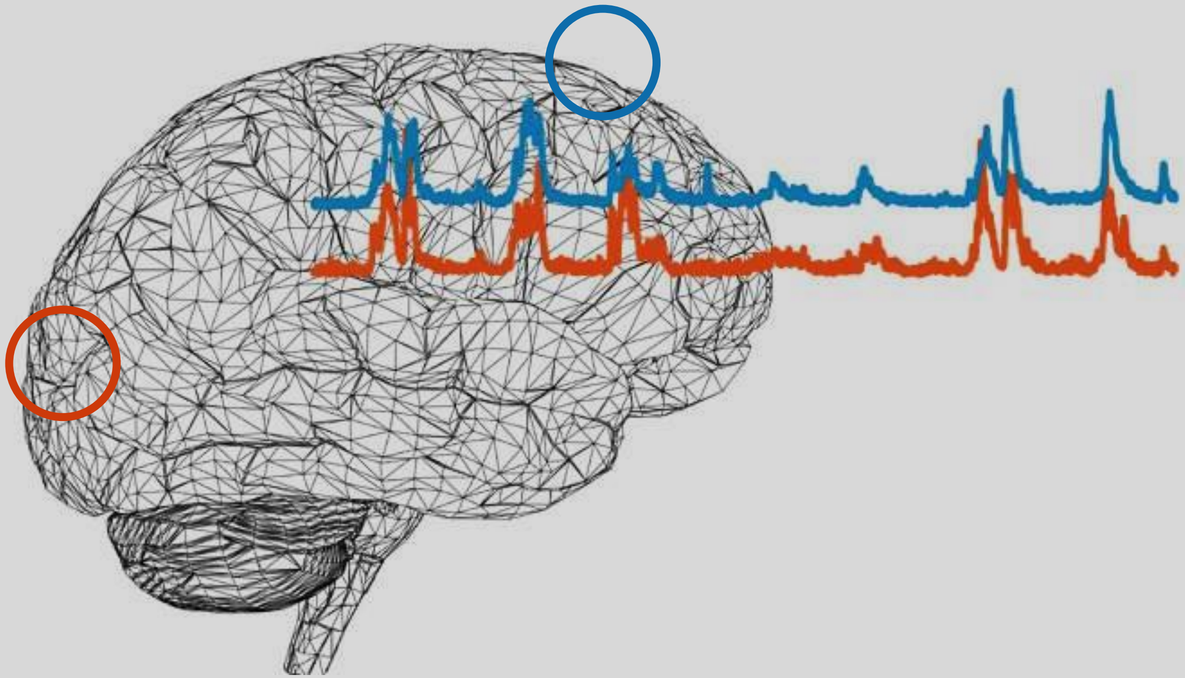
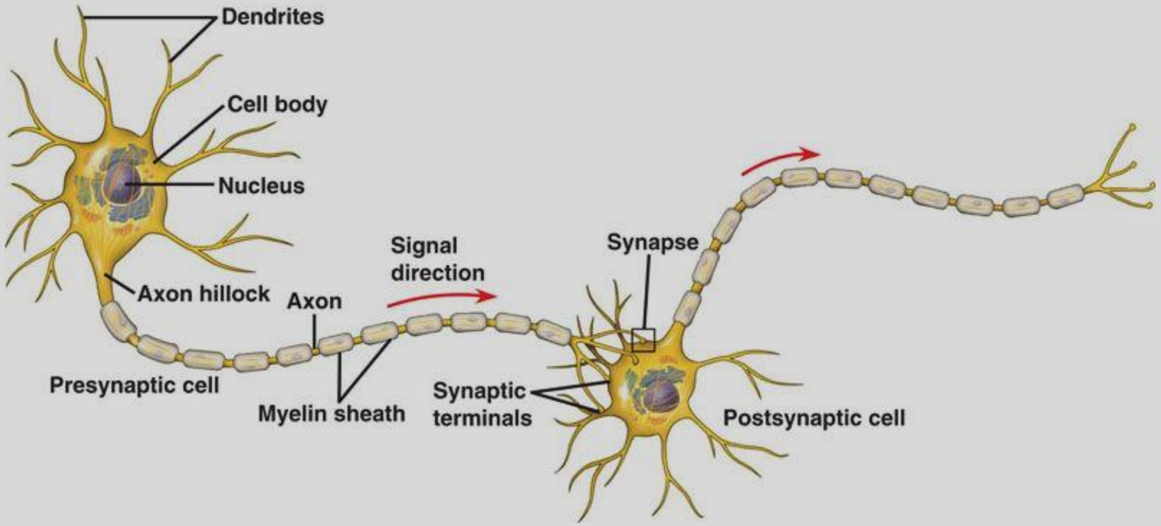
OUTLINE

- Abstract
- Introduction
- Results (3)
- Discussion
- Materials and Methods (Supporting Information)




Nucleus
Myelin sheath
Schwann cell
Axon Terminals
Dendrites
Soma
(cell body)
Axon
Node of Ranvier
Action Potential




Dendrites
Cell body
Nucleus
Axon hillock
Axon
Signal direction
Synapse
Presynaptic cell
Myelin sheath
Synaptic terminals
Postsynaptic cell

Zero Time Lag Synchronization of Individual Neurons as a Self Organization Process.

This mechanism of synchronization rests on the ability of an excitatory postsynaptic potential (EPSP) to modify the firing latencies of a postsynaptic neuron in a consistent manner.

It further relies on the symmetric relay that the central neuron provides for the indirect communication between the outer neurons.

The key idea is that this network motif allows for the outer neurons to exert an influence on each other via the intermediate relay cell. Thus, the reciprocal connections from the relay cell assure that the same influence that is propagating from one extreme end of the network to the other is also fed back into the neuron that originated the perturbation promoting the synchronous state.

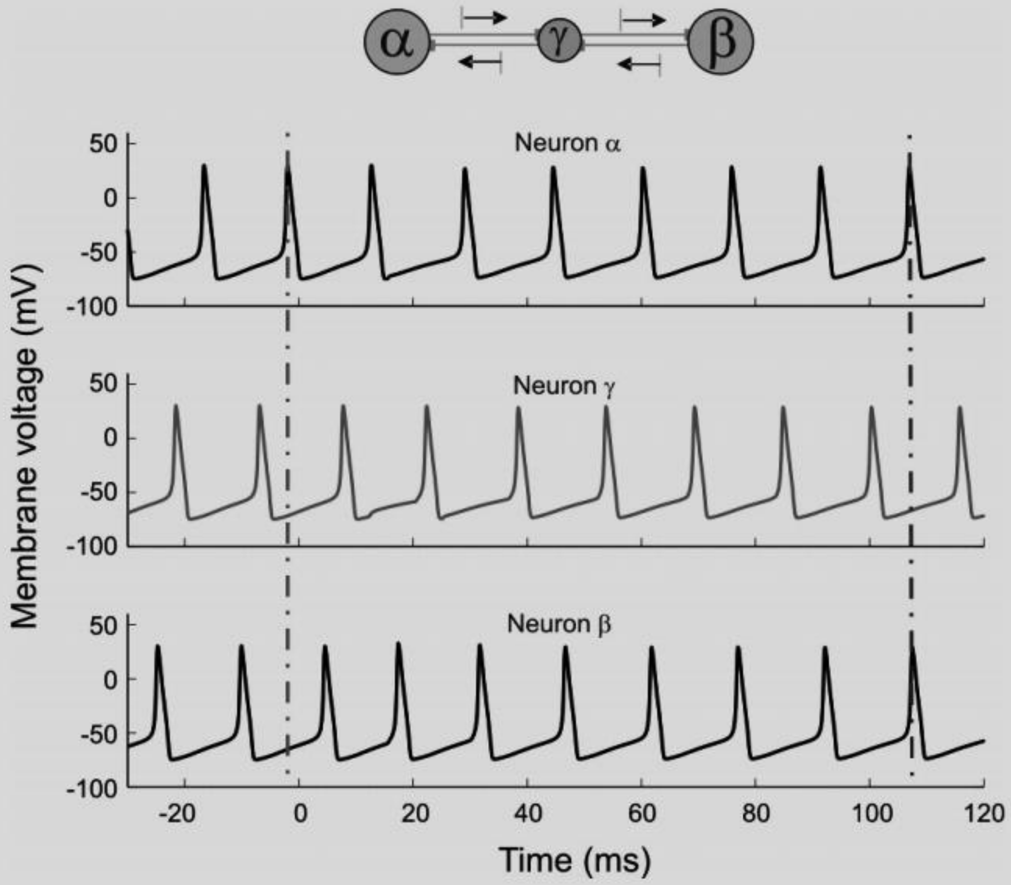


Fig. 1. Time series of the membrane voltage of 3 coupled HH cells N_α-N_β-N_γ. At time $t = 0$ the excitatory synapses were activated. Conduction delay $\tau = 8$ ms. Vertical lines help the eye to compare the spike synchrony before and after the interaction takes place.

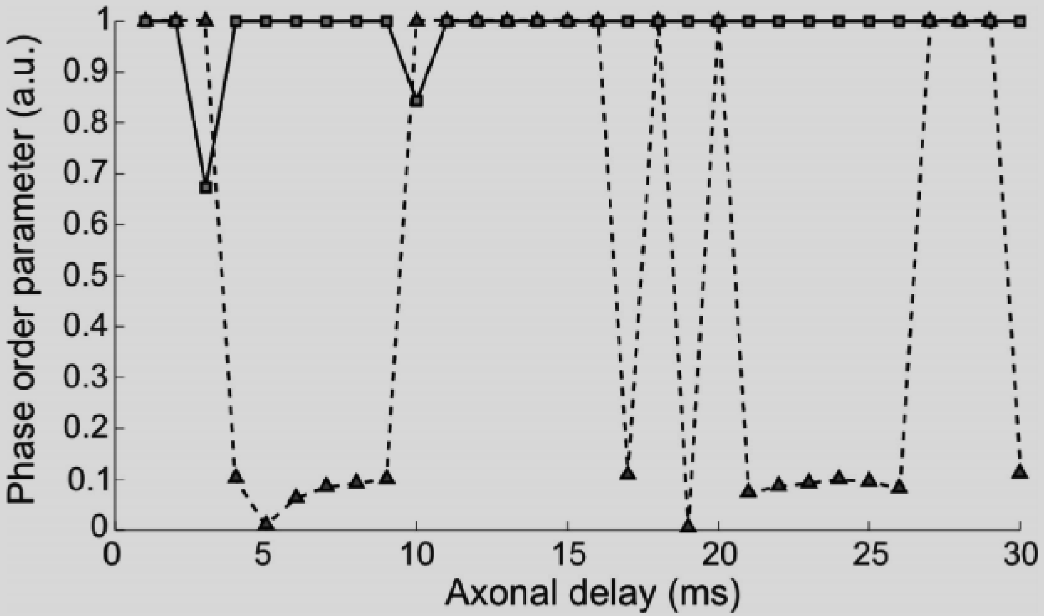


Fig. 2. Dependence of zero time lag synchronization as a function of the axonal delay between neighbor cells for a scheme of 2 coupled neurons (dashed line) and 3 coupled neurons (solid line). In the case of the 3 interacting cells, only the synchrony between the outer neurons is plotted here.

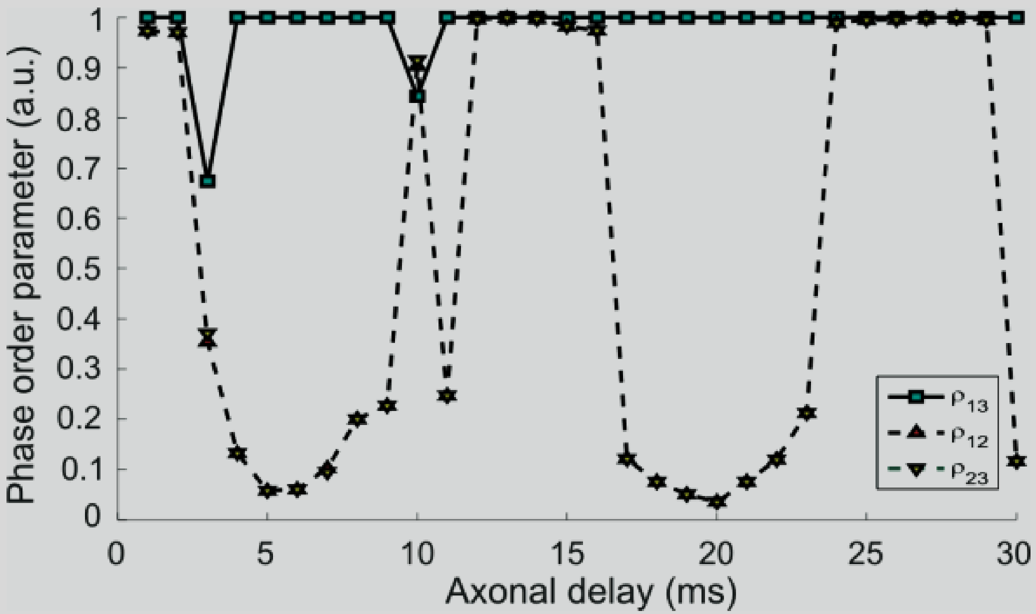


Fig. S1. Synchronization index at zero lag for pairs of HH neurons 1 and 3 (squares), 1 and 2 (upright triangles), and 2 and 3 (inverted triangles) as a function of the axonal delay. The coupling is excitatory, and the neurons are interacting according to the scheme in Fig. 1 *Top* in the main text. The sudden decays of the synchronization index between nearest neighbor neurons usually indicate the transitions to antiphase states. Notice that the zero-phase relation between neurons 1 and 3 is almost insensitive to such changes.

Hodgkin Huxley model

$$C\frac{dV}{dt} = -g_{Na}m^3h(V - E_{Na}) - g_K n^4(V - E_k) - g_L(V - E_L) + I_{ext} + I_{syn}, \quad \textbf{[S1]}$$

$$\frac{dm}{dt} = \alpha_m(V)(1 - m) - \beta_m(V)m, \quad \textbf{[S2]}$$

$$\frac{dh}{dt} = \alpha_h(V)(1 - h) - \beta_h(V)h, \quad \textbf{[S3]}$$

$$\frac{dn}{dt} = \alpha_n(V)(1 - n) - \beta_n(V)n, \quad \textbf{[S4]}$$

$$\alpha_m(V) = \frac{0.1(V + 40)}{1 - \exp(-(V + 40)/10)}, \quad \textbf{[S5]}$$

$$\beta_m(V) = 4\exp(-(V + 65)/18), \quad \textbf{[S6]}$$

$$\alpha_h(V) = 0.07\exp(-(V + 65)/20), \quad \textbf{[S7]}$$

$$\beta_h(V) = [1 + \exp(-(V + 35)/10)]^{-1}, \quad \textbf{[S8]}$$

$$\alpha_n(V) = \frac{(V + 55)/10}{1 - \exp(-0.1(V + 55))}, \quad \textbf{[S9]}$$

$$\beta_n(V) = 0.125\exp(-(V + 65)/80). \quad \textbf{[S10]}$$

$$\alpha(t) = \frac{1}{\tau_d - \tau_r}(\exp(-t/\tau_d) - \exp(-t/\tau_r)), \quad \textbf{[S11]}$$

Dynamical Relaying-Based Synchronization Is Robust to Broad Distributions of Axonal Delays

$$I_{syn}(t) = -\frac{g_{max}}{N} \sum_{\tau_l} \sum_{spikes} \alpha(t - t_{spike} - \tau_l)(V(t) - E_{syn}),$$

[S12]

$$f(\tau_l) = \tau_l^{k-1} \frac{\exp(-\tau_l/\theta)}{\theta^k \Gamma(k)}, \quad \text{[S13]}$$

Our numerical simulations indicate that for a large region of mean delays (between 3 and 10 ms), the outer neurons synchronize independently of the shape of the distribution. These results can be observed in Fig. 3 *Right*, where we plot the zero lag synchronization index of the outer neurons of the network motif as a function of the shape of the γ distribution of axonal delays and its mean value.

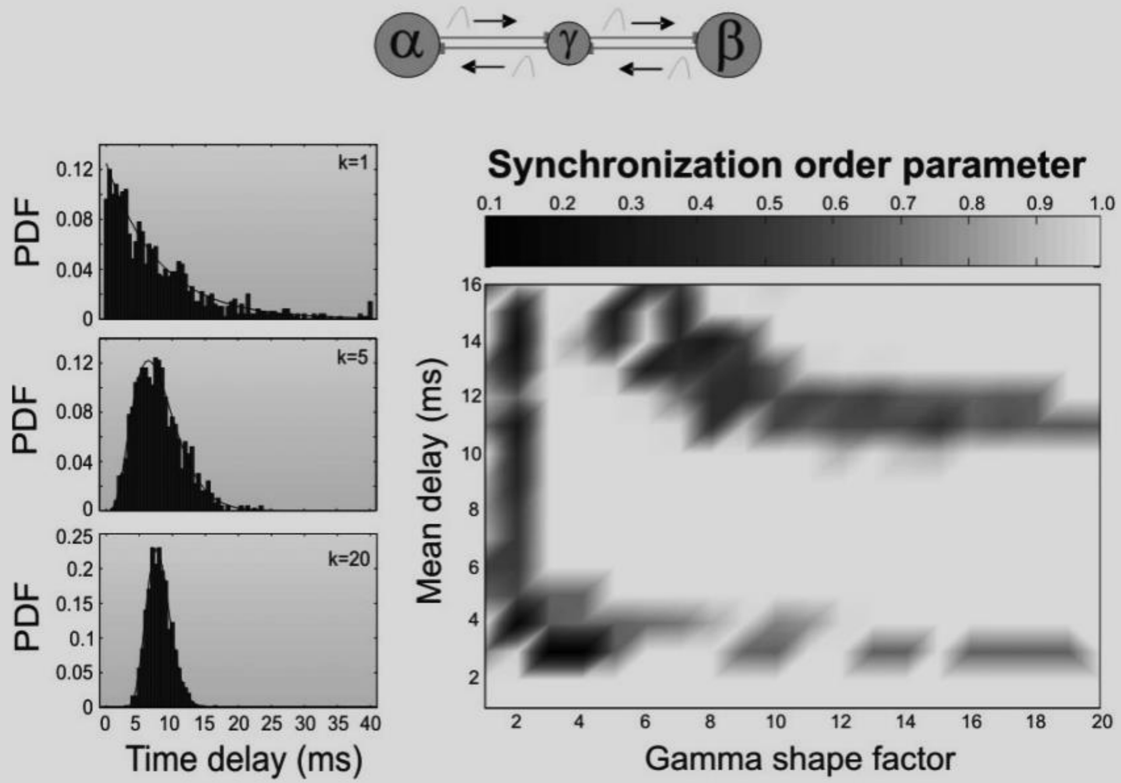


Fig. 3. Effects of broad distributions of axonal delays on synchrony. (*Left*) γ distribution of delays with different shape factors (k = 1, 5, and 20) and the same mean (τ = 8 ms). (*Right*) Synchronization index at zero lag of the outer neurons as a function of the shape factor and mean of the distribution of delays.

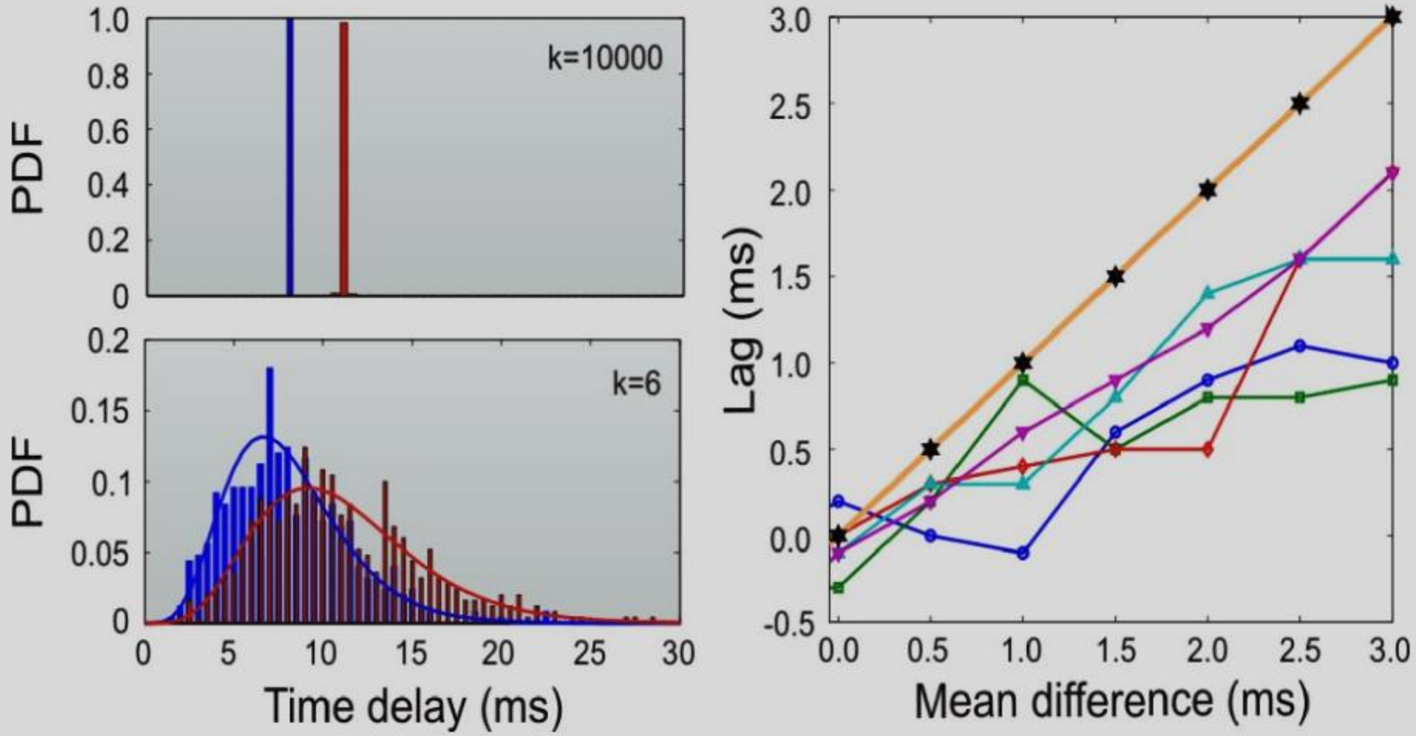


Fig. 4. Effects of dissimilar distributions of axonal delays on the lag of synchronization. (*Left*) different γ distributions of delays used for the 2 dissimilar branches of the network module. (*Upper*) Distributions with shape factor $k = 10{,}000$ (quasi-δ) and means of 8 and 11 ms. (*Lower*) Distributions with shape factor $k = 6$ and means of 8 and 11 ms. (*Right*) Lag between the discharges of the outer neurons as a function of the difference in the mean of the distributions of delays for the 2 branches. Shape factors $k = 6$ (squares), $k = 8$ (circles), $k = 10$ (diamonds), $k = 12$ (upright triangles), $k = 14$ (inverted triangles), and $k = 10{,}000$ (stars) were tested.

Populations of Distant Neurons Can Also Exhibit Zero Lag Synchrony via Dynamical Relaying

integrate and fire (IAF)

$$\tau_m \frac{dV_i}{dt} = -V_i(t) + RI_i(t), \qquad \textbf{[S14]}$$

$$RI_i(t) = \tau_m \sum_j J_j \sum_k \delta(t - t_j^k - \tau_l) + A\xi_i, \qquad \textbf{[S15]}$$

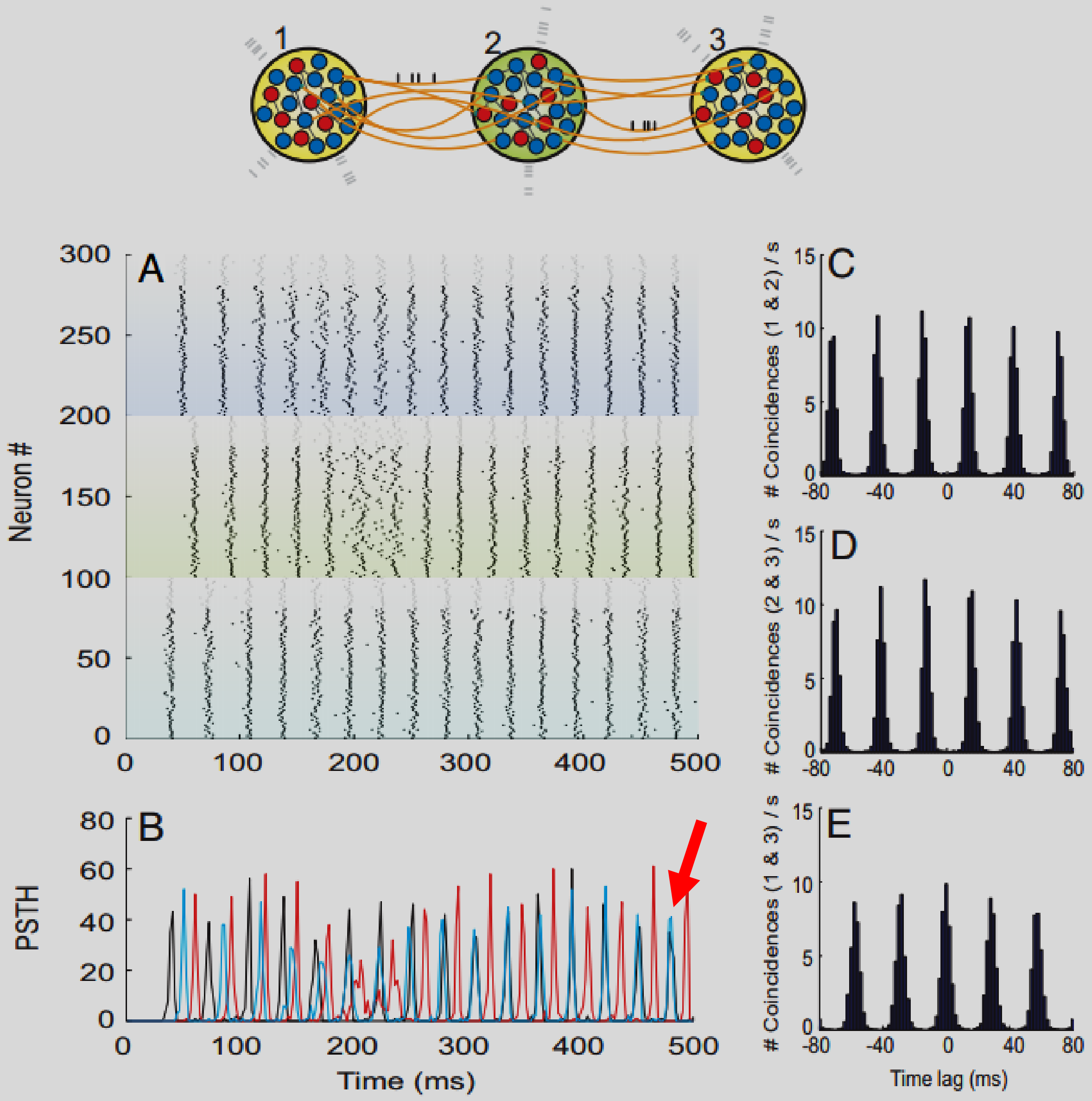


Fig. 5. Dynamics of 3 large-scale networks interacting through dynamical relaying. (*A*) Raster plot of 300 neurons randomly selected among the 3 populations (neurons 1–100 are from population 1, 101–200 from population 2, and 201–300 from population 3). The top 20 neurons of each subpopulation (plotted in gray) are inhibitory, and the rest are excitatory (black). (*B*) Firing histogram of each subpopulation of 100 randomly selected neurons (black, red, and blue colors code for populations 1, 2, and 3, respectively). (*C*) Averaged cross-correlogram between neurons of populations 1 and 2. (*D*) Averaged cross-correlogram between neurons of populations 2 and 3. (*E*) Averaged cross-correlogram between neurons of populations 1 and 3. At t = 100 ms, the external interpopulation synapses become active. Bin sizes for the histogram and correlograms are set to 2 ms. Interpopulation axonal delays are set to 12 ms.

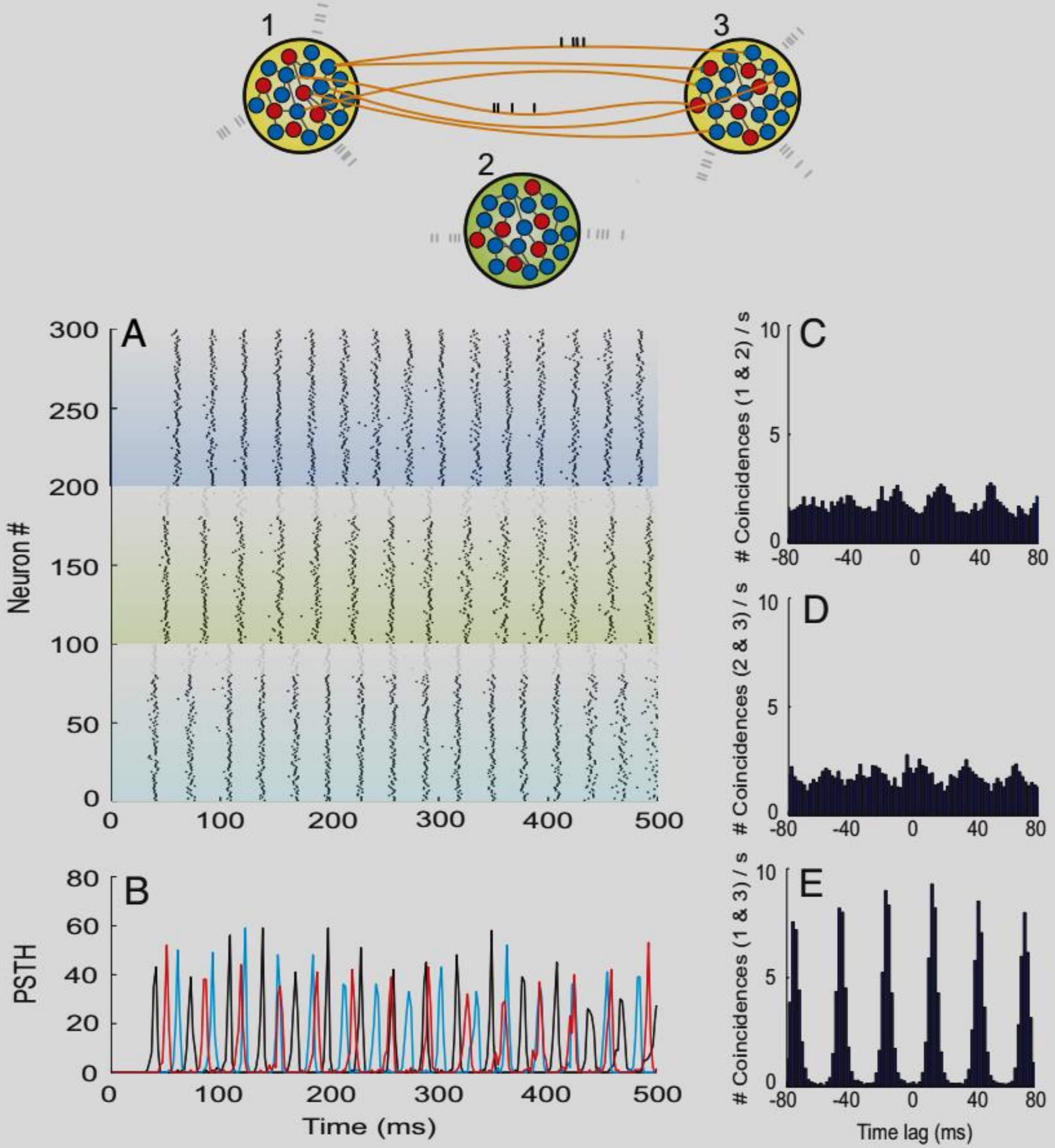


Fig. 6. Dynamics of 2 large-scale networks interacting directly. Population 2 is disconnected from other populations. Structure of the panels and parameters are otherwise as in Fig. 5.

Seizure First Aid

What to do in the event of a seizure

1. **STAY** with the person and start timing the seizure. Remain ***calm*** and check for medical ID.
2. Keep the person **SAFE**. Move or guide away from ***harmful objects***.
3. Turn the person onto their **SIDE** if they are not awake and aware. ***Don't block airway***, put something small and soft under the head, loosen tight clothes around neck.
4. Do **NOT** put ***anything*** in their mouth. Don't give water, pills or food until the person is awake.
5. Do **NOT** ***restrain.***
6. **STAY** with them until they are awake and alert after the seizure. ***Most seizures end in a few minutes.***

Call 911:

- ☒ Seizure lasts longer than 5 minutes
- ☒ Repeated seizures
- ☒ **Difficulty breathing**
- ☒ Seizure occurs in water
- ☒ Person is injured, pregnant, or sick
- ☒ Person does not return to their usual state
- ☒ First time seizure

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PURPLE DAY
FOR EPILEPSY
March 26/Farvardin 7