# Pool-size invariance requires inverse synaptic scaling

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## Abstract

The apparent invariance of pyramidal–interneuron network gamma (PING) firing rate to inhibitory-pool size is a consequence of the simulator's fan-in normalization, not independence from interneuron count. Excitatory cells are denoted E and inhibitory cells I. The nominal I→E parameter $G_{IE}$ is divided by the presynaptic pool size, so the realised mean synaptic conductance is $j_{IE} = \frac{G_{IE}}{N_I}$. We vary $N_I$ under paired controls. Rates remain flat when $G_{IE}$ is fixed and $j_{IE} \propto \frac{1}{N_I}$; when the realised synapse $j_{IE}$ is fixed, rates change strongly with $N_I$. Pool-size invariance therefore requires compensatory inverse scaling of individual synapses.

## Methods

**1. Weight convention.** For a dense I→E matrix with shape $N_I \times N_E$, the simulator draws a non-negative weight with nominal mean $G_{IE}$ and then divides every entry by its presynaptic fan-in $N_I$. Thus

$$\mathcal{E}\left[W_{kj}^{IE}\right] \approx j_{IE} = \frac{G_{IE}}{N_I}, \quad \mathcal{E}\left[\sum_{k=1}^{N_I} W_{kj}^{IE}\right] \approx G_{IE}. \quad (1)$$

$G_{IE}$ is consequently an expected *summed coupling*, not the conductance of one synapse. An inhibitory volley with active set $\mathcal{A}$ gives E cell $j$ the conductance increment

$$\Delta g_j^I = \sum_{k \in \mathcal{A}} W_{kj}^{IE}, \quad (2)$$

where:

- $\mathcal{E}$ denotes expectation over random weight initialization;
- $W_{kj}^{IE}$ is the realised conductance from inhibitory cell $k$ to excitatory cell $j$;
- $j_{IE}$ is the mean realised conductance of one I→E synapse;
- $G_{IE}$ is the expected sum of all I→E weights arriving at one E cell;
- $N_I$ and $N_E$ are the inhibitory and excitatory pool sizes;
- $\mathcal{A}$ is the set of inhibitory cells active in a volley; and
- $\Delta g_j^I$ is the resulting inhibitory-conductance increment at excitatory cell $j$.

Equation 2 therefore depends on both the realised synapses and the number of participating inhibitory cells.

**2. Paired controls.** We sweep $N_I \in \{16, 64, 256\}$ under two conventions. In the fixed-summed-coupling arm, $G_{IE} \in \{1, 2, 4\}$ µS is held fixed, forcing $j_{IE} \propto \frac{1}{N_I}$. In the fixed-synapse

arm, $j_{IE} \in \{3.91, 7.81, 15.63\}$ nS is held fixed and the nominal parameter is set to $G_{IE} = N_I j_{IE}$. The two arms coincide at the reference pool $N_I = 256$.

**3. Simulation.** Untrained dense PING network, $N_E = 1024$, $\Delta t = 0.1$ ms, $T = 500$ ms, 8 independent Poisson-input trials per network and 3 network/input seeds per condition. E→I summed coupling is fixed at 1 μS; input comprises 784 independent 25 Hz Poisson channels. Markers show seed means and error bars ±1 standard deviation (SD).

## Results

Fixed summed coupling produces overlapping rate curves. At $G_{IE} = 2$ μS, the E rate is 6.01 Hz for $N_I = 16$ and 6 Hz for $N_I = 256$. This is the expected consequence of holding the summed I→E coupling fixed while shrinking each realised synapse as $\frac{1}{N_I}$.

Fixed realised synaptic strength gives the opposite result. At $j_{IE} = 7.81$ nS, increasing $N_I$ from 16 to 256 changes the E rate from 16.62 to 6 Hz and the I rate from 120.41 to 34.41 Hz. Every tested synaptic strength shows the same pool-size dependence. The direction is mechanistically consistent: more inhibitory cells at unchanged individual strength produce greater population-level inhibition, while the recurrent feedback reduces both E activity and the I activity it recruits.

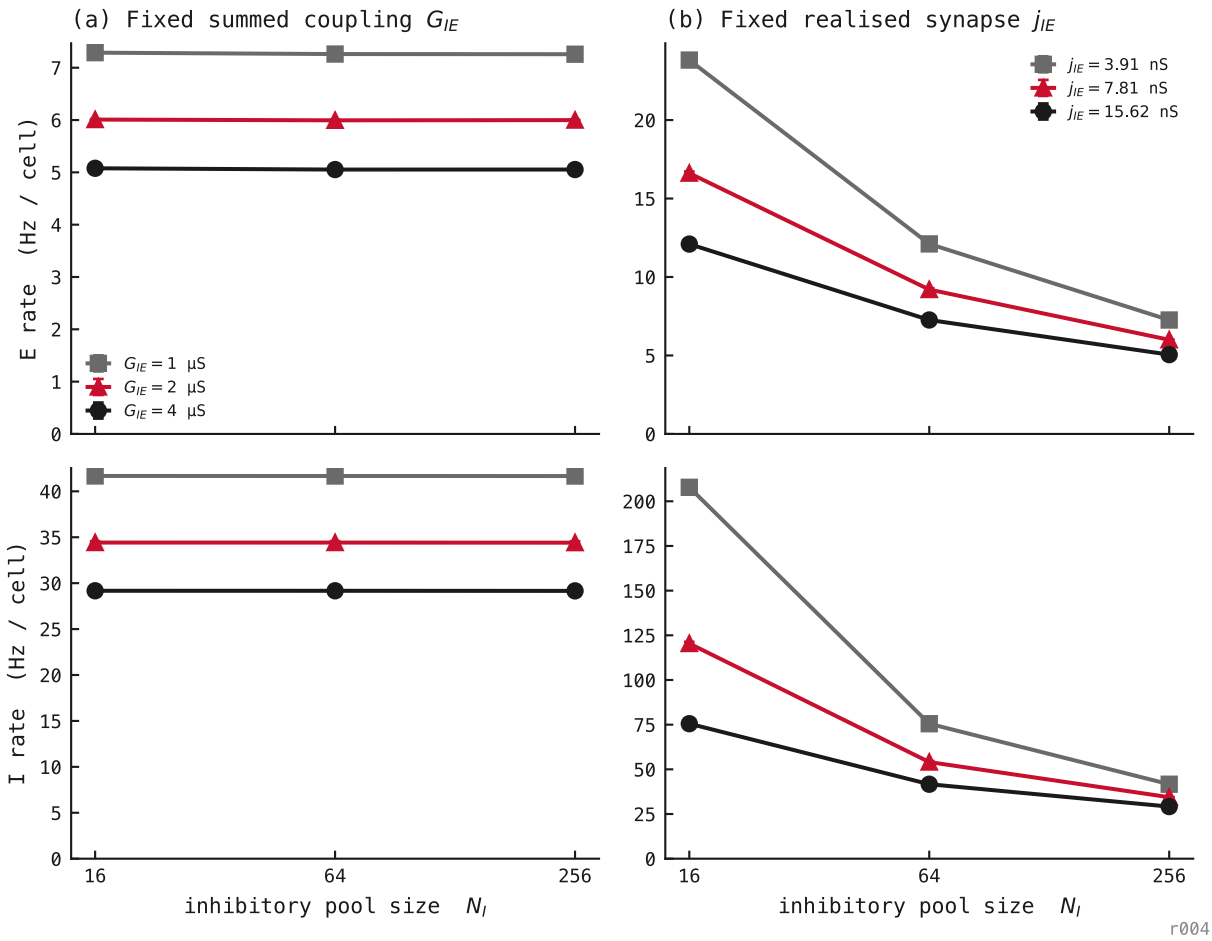


Figure 1: **Excitatory and inhibitory firing rates across inhibitory-pool size under two I→E scaling controls.** Top: excitatory-cell rate in Hz per cell. Bottom: inhibitory-cell rate in Hz per cell. **(a)** Holding the expected summed I→E coupling $G_{IE}$ fixed makes the realised synapse $j_{IE} = \frac{G_{IE}}{N_I}$ shrink as the pool grows. **(b)** Holding $j_{IE}$ fixed makes summed coupling grow with $N_I$. Each curve is one coupling level; markers are means and error bars ±1 standard deviation (SD) over 3 seeds, with 8 trials per seed. The controls coincide at the reference pool $N_I = 256$. Rates are invariant to pool size only under inverse scaling of individual synapses.

## Conclusion

The network is not intrinsically insensitive to inhibitory-pool size. It is insensitive only along a specific normalization path: individual I→E synapses must scale approximately as $\frac{1}{N_I}$ so that expected summed coupling stays fixed. The operative control variable in this dense model is population-level inhibitory drive, jointly determined by pool size, realised synaptic strength, and volley participation. This experiment does not establish that the same inverse scaling holds biologically; it identifies the compensation required for invariance in the model.