

# Computational Neuroscience

*Project No. 2, Report*

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## Introduction

The report aims to explore the workings of synapses, the interactions within neuronal populations, and the neural basis of decision-making in response to diverse stimuli. By delving into synaptic functions, neuronal connectivity, and simulating decision processes, this investigation seeks to enhance our understanding of fundamental neuroscience mechanisms and cognitive functions.

I have utilized several libraries, including PyTorch for its neural network capabilities, Matplotlib for plotting, and the Math and Random libraries for numerical operations and stochastic processes, respectively. The project outlines include:

1. **Synapse Implementation:** Using a specific function known as the Dirac delta function to simulate how synapses can vary in their functions.
2. **Connectivity Schemes:** Exploring three designs of how neurons connect to each other:
  - i. *Full Connectivity:* Every neuron is connected to every other neuron.
  - ii. *Random Coupling with Fixed Probability:* Connections are made randomly based on a set chance.
  - iii. *Random Coupling with a Fixed Number of Presynaptic Partners:* Each neuron connects to a specific number of other neurons randomly chosen.
3. **Simulation of Neuronal Populations:** Creating two homogenous groups of neurons – one excitatory and the other inhibitory – in a 2:8 size ratio to study their interactions.
4. **Decision Making:** Setting up three groups of neurons – two excitatory and one inhibitory – all of equal size. The connections among these groups will be detailed in the report. The decision process depends on the input to the excitatory populations; the group receiving more input is expected to show higher activity levels, illustrating the decision-making process.

## Synapse Implementation

The implementation of synapses in neuronal models can be effectively represented using the **Dirac delta function** ( $\delta$ ), a mathematical function that models the transmission of signals between neurons at precise moments. The Dirac delta function is utilized to simulate the instantaneous signal transmission from a presynaptic neuron to a postsynaptic neuron.

The basic principle of synapse implementation using the Dirac delta function can be expressed as follows:

When a presynaptic neuron fires an action potential at time  $t_0$ , the synaptic input to the postsynaptic neuron can be represented as  $I(t) = W \cdot \delta(t - t_0)$ , where  $I(t)$  denotes the synaptic input at time  $t$ ,  $W$  represents the synaptic weight or strength, and  $\delta(t - t_0)$  is the Dirac delta function centered at the spike time  $t_0$ .

The Dirac delta function is defined such that  $\delta(t - t_0) = 0$  for all  $t \neq t_0$ , and its integral over the entire time domain is equal to 1, mathematically represented as

$$\int_{-\infty}^{\infty} \delta(t - t_0) dt = 1.$$

This characteristic ensures that the total synaptic input is concentrated at the precise moment of the action potential, mimicking the real-life instantaneous effect of synaptic transmission.

By adjusting the synaptic weight  $W$  and the timing of action potentials  $t_0$ , different synaptic behaviors can be simulated to understand their roles in neural computations, such as the timing-dependent plasticity and the integration of excitatory and inhibitory inputs by neurons.

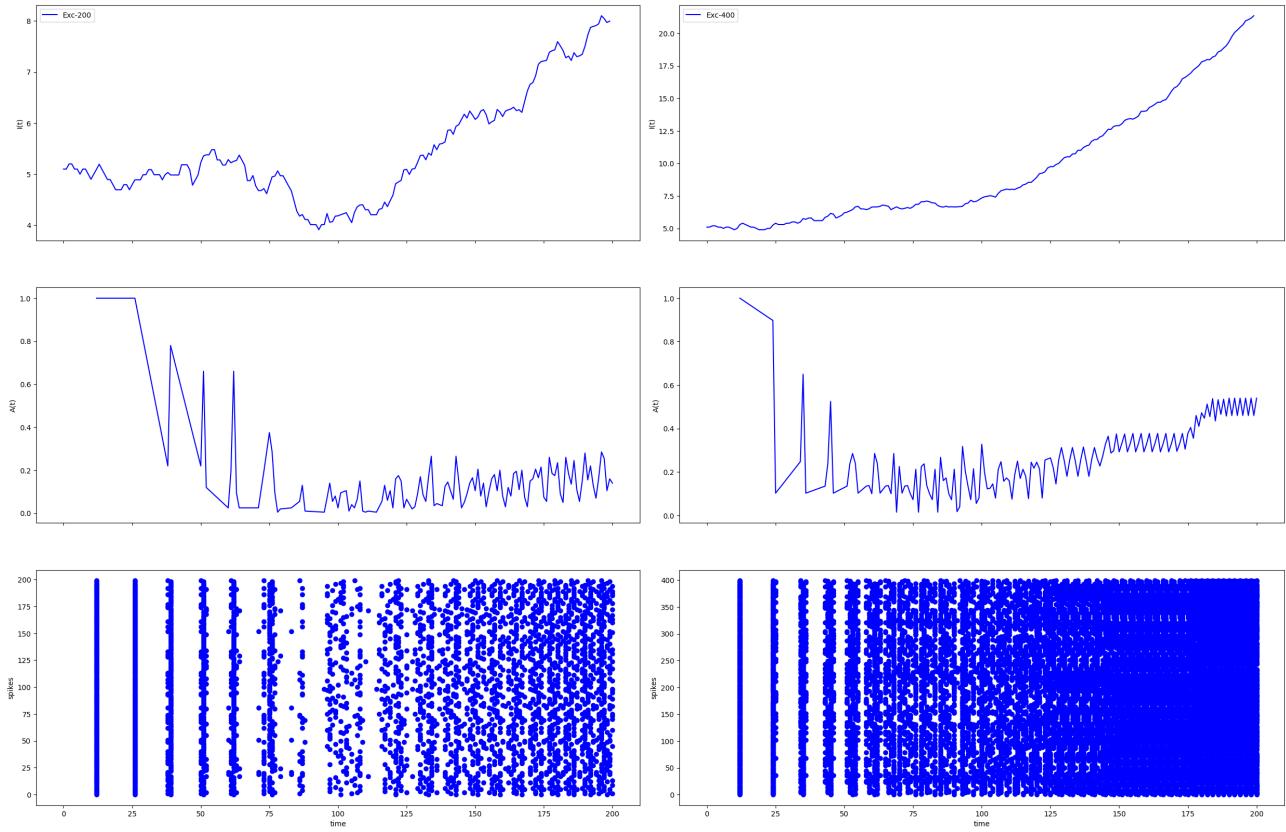
Two classes, `SynFunc()` and `DendriteDirac()`, inherit from the `Behavior` class to manage how synapses work. `SynFunc()` is designed for use with `SynapseGroup`, and `DendriteDirac()` is for `NeuronGroup`.

`SynFunc()` simplifies synapse behavior through two functions: `initialize()` and `forward()`. In `initialize()`, it sets up needed attributes based on the type of connection and creates a weight matrix with a size of SxD. This matrix initially gets filled with values following a normal distribution without any scaling. The `forward()` method introduces `synapse.I`, representing the input to the neurons receiving the signal. It is calculated by adding up the weights of all the neurons that have fired, multiplied by a variable named `coef`. This `coef` variable decides whether the sending neuron enhances (excitatory, value of 1) or suppresses (inhibitory, value of -1) the receiving neuron's activity, affecting all types of synaptic functions similarly. The calculated `synapse.I` is then used by the `DendriteDirac()` class.

`DendriteDirac()` contains a single method, `forward()`, which processes input for each neuron in a group. It does this by adding together the `synapse.I` values from all incoming synapses, contributing to the overall current of the receiving neuron.

Without a proper scaling approach, `SynFunc()`'s performance becomes sensitive to changes in the population size. As the number of neurons in the population increases, this lack of scaling can lead to significant issues ([Figure 1](#)). The observation underscores the direct relation between population size and increases in synaptic function activity, notably in the absence of a scaling mechanism to moderate this effect.

Namely, the root of the problem lies in the way the weight matrix is generated. When the population size ( $N$ ) grows, the total input to any given neuron can escalate dramatically due to the increased number of connections. Without any scaling, the elements of the weight matrix are filled based on a Gaussian distribution, causing the variance and the mean of the inputs to scale with  $N$ . This escalation can overwhelm neurons, leading to either excessive firing rates or the opposite effect, where neurons become less likely to fire due to overly inhibitory input.

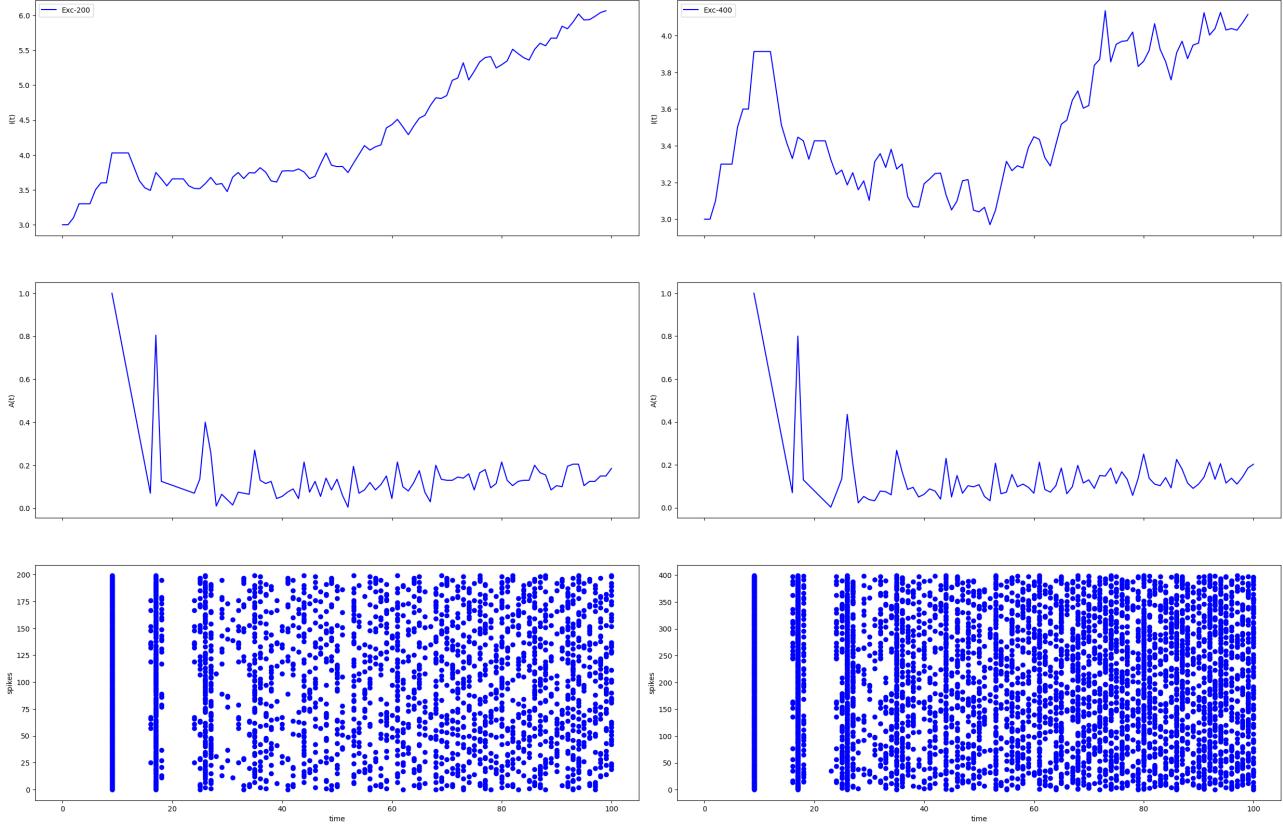


**Figure 1** The impact of population size on synapse function without a scaling approach, within a homogenous group of excitatory neurons. The left column presents a population of 200 neurons, and the right column shows a population double that size, at 400 neurons. Over 200 iterations, it is observed that the activity level—and correspondingly, the current ( $I$ )—for the larger group ( $N=400$ ) increases significantly. The current sees a sharp rise, reaching a value of  $I=20$ , which is 2.5 times higher than that of the smaller population ( $N=200$ ). These neurons are interconnected through synapses exhibiting a normal distribution with mean ( $\mu$ ) of 0.001 and standard deviation ( $\sigma$ ) of 0.001. Also the current is initialized at 5, attributed to consistent noise factors controlled by a set random seed, ensuring reproducibility across various simulations. All neurons in the study share uniform parameters, and the LIF model underpins all simulations conducted.

Furthermore, without scaling, the network's dynamic range narrows. This means that as  $N$  increases, the network becomes less capable of distinguishing between different patterns of activity because the inputs to each neuron become more similar (in terms of their statistical properties), reducing the network's overall computational power and flexibility.

## Full connectivity scheme

The most fundamental connectivity pattern is the all-to-all or full connectivity scheme within a neuronal network, where each neuron connects to every other neuron with uniform synaptic strength. To accommodate changes in the number of neurons,  $N$ , within a population while conducting simulations, a scaling law is employed:



**Figure 2** The impact of population size on synapse function with **full connectivity scaling method**, within a homogenous group of excitatory neurons. On the left, we see a population containing 200 neurons, while on the right, there's a group twice as large, with 400 neurons. Despite the difference in size, the  $A(t)$  across both populations remain remarkably consistent, without significant deviations. While, the current displays more variation across iterations, the smaller population reaches a peak current ( $I$ ) of 6 after 100 iterations, in contrast, the larger population peaks at a lower current value of 4. The neurons are linked through synapses that follow a normal distribution, characterized by a mean ( $\mu$ ) of 0.2 and a standard deviation ( $\sigma$ ) of 0.1.

$$w_{ij} = \frac{J_0}{N}$$

This scaling law serves as a mathematical tool that allows for the exploration of scenarios where  $N \rightarrow \infty$ , ensuring that the expected input a neuron receives from its counterparts in the network remains constant.

A more nuanced approach to the all-to-all connectivity model introduces variability in synaptic strengths. In this model, synaptic weights,  $w_{ij}$ , are determined by a Gaussian distribution with a mean of  $J_0/N$  and a standard deviation of  $\sigma_0/\sqrt{N}$ . This adjustment results in each neuron receiving a slightly different synaptic input from the others in the network, leading to fluctuations in their membrane potentials. These fluctuations are characterized by a magnitude of  $\sigma_0$ , even as the network grows. This variation introduces an element of realism into the model by acknowledging the inherent diversity in synaptic strength found in biological neural networks.

## Implementation

To implement the described functionality, the class `SynFuncFull()` receives attributes `coef`, `mu`, `sigma`, and `N` from the input and constructs a matrix following a Gaussian normal distribution, with the mean adjusted to `mu/N` and the standard deviation modified to `sigma/sqrt(N)`. This approach differs significantly from the initial method `SynFunc()`, which does not incorporate scaling.

### Scaling Effect

As a result of this scaling approach in `SynFuncFull()`, variations in population size do not affect the activity level; furthermore, the current does not experience a notable increase despite the increase in population size (indicating a higher input from the stimulation of more neurons) as it can be seen in [Figure 2](#).

As  $N$  approaches infinity, variance in the input tends to zero, making the expected synaptic input indistinguishable from the exact input received by any neuron within the population. Despite this theoretical construct, real neural networks always contain a finite number of neurons, implying that input fluctuations are inevitable, although increasing  $N$  tends to minimize these variations.

## Random Coupling: Fixed Coupling Probability

When modeling such connections in simulations, the connection probability  $p$  is fixed, and connections are established randomly with this probability among all possible pairs of neurons, leading to  $N^2$  possible connections. For any given postsynaptic neuron  $j$ , the expected number of presynaptic inputs, denoted by  $\langle C_j \rangle$ , averages out to  $pN$ , but actual values can fluctuate between individual neurons, featuring a variance of  $p(1 - p)N$ .

The total number of input connections per neuron scales linearly with the population size  $N$ . To maintain consistent mean synaptic input across variations in population size, the strength of each synaptic connection  $w_{ij}$  is scaled as:

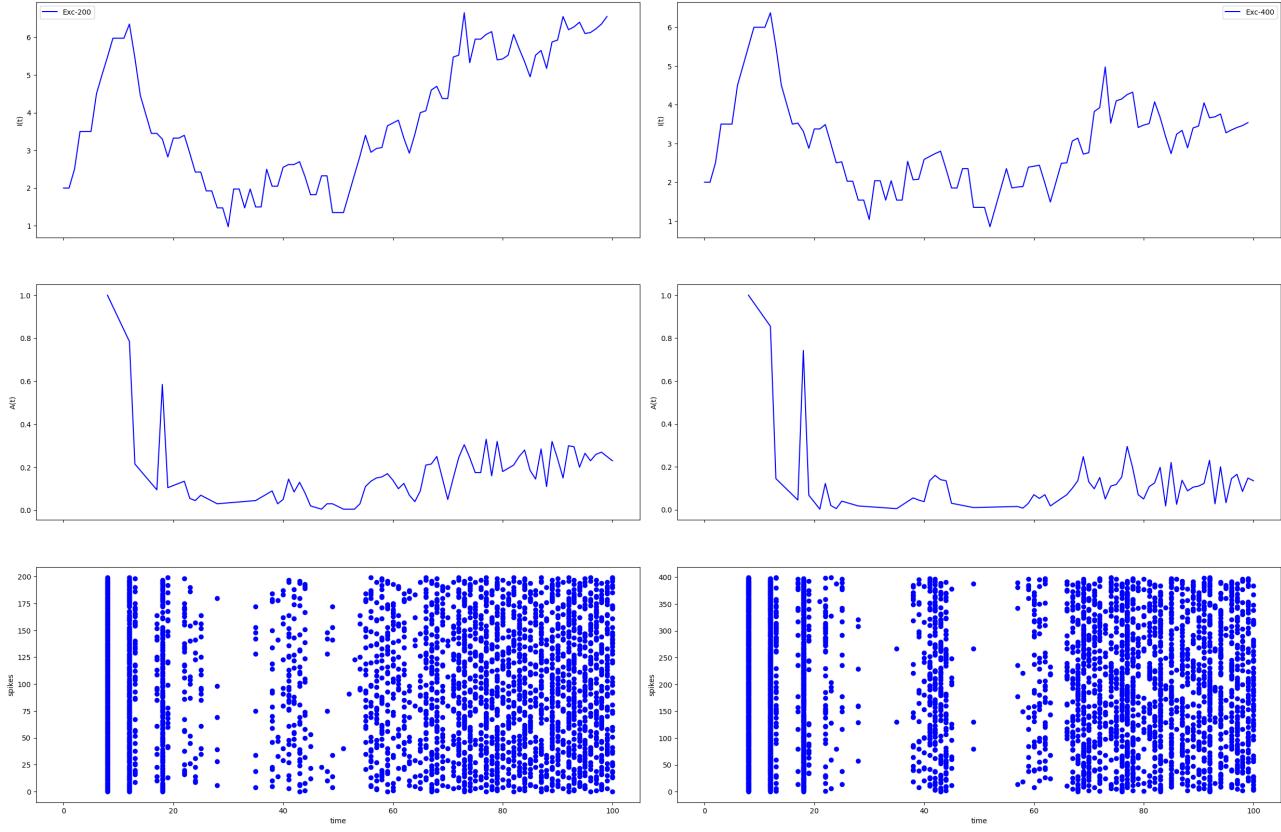
$$w_{ij} = \frac{J_0}{C} = \frac{J_0}{pN}$$

## Implementation

To implement the described functionality, the class `SynFuncRandFCP()` takes in attributes `coef`, `J`, `p`, and `N` as input. It begins by creating a weight matrix where each element is assigned the value  $J/(N*p)$ . Following this, a mask tensor is generated: initially, a new tensor is filled with the probability  $p$  and shaped identically to the weight matrix. This is then processed using the built-in `torch.bernoulli` function, which transforms it into a mask of 0s and 1s—each element is set to 1 with probability  $p$  and to 0 otherwise, reflecting a Bernoulli distribution. Finally, this mask is used to modify the weight matrix through multiplication, effectively applying the probabilistic pattern to the weights.

### Scaling Effect

This scaling scheme ensures that as the size of the network increases, despite individual synaptic inputs having a diminished effect, the overall mean input to a typical neuron remains stable.



**Figure 3** The impact of population size on synapse function with **fixed coupling probability of  $p=0.1$** , within a homogenous group of excitatory neurons. On the left, we see a population containing 200 neurons, while on the right, there's a group twice as large, with 400 neurons. Despite the difference in size, the  $A(t)$  across both populations remain remarkably consistent, without significant deviations. The current shows a reduced amount of fluctuation due to noise across iterations, meaning the influence of these fluctuations on the input received by any given neuron is decreased.

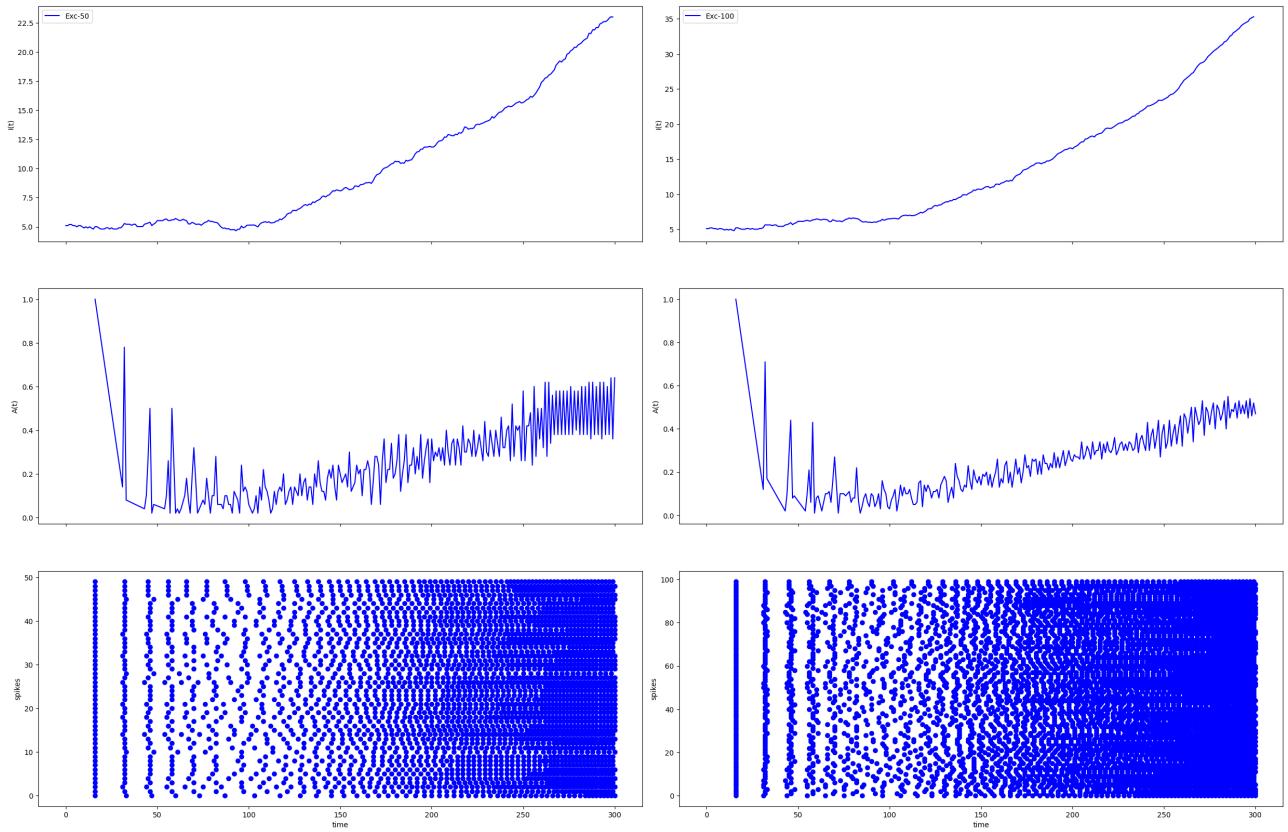
Consequently, as the network grows, the impact of fluctuations on the input received by any given neuron decreases, facilitating more consistent simulation outcomes irrespective of the simulated population size ([Figure 3](#)).

## Random Coupling: Fixed Number of Presynaptic Partners

The strategy for constructing a network with this connectivity scheme involves assigning a fixed number  $C$  of presynaptic inputs to each neuron within a network. This is done sequentially for each neuron by randomly selecting its  $C$  presynaptic partners.

### Implementation

To implement the described function, the class `SynFuncRandFNPP()` receives attributes `coef`, `mu`, `sigma`, `C` and `N` from the input and constructs a matrix following a Gaussian normal distribution, with the mean adjusted to `mu` and the standard deviation modified to `sigma`.



**Figure 4** The impact of population size on synapse function with fixed number of presynaptic partners  $C=5$ , within a homogenous group of excitatory neurons. On the left, we see a population with  $N=200$  neurons, while on the right, the group size is  $N=400$ . We can see a sharp increase in current from  $I=30$  to  $I=80$ . Intriguingly, in the graph representing the larger population size of 400, the variable  $A(t)$  exhibited fewer fluctuations in comparison to the graph for the population of 200.

We then iterate over each column in the weight matrix  $W$ . For each column (representing a postsynaptic neuron's connection to its presynaptic counterparts), The `torch.randperm` first outputs a permutation of numbers from 0 to  $\text{len}(W)$ , Then a mask is created by choose the first  $\text{len}(W) - C$  of the permutation. This mask is representing a proportion of the total presynaptic connections to be nullified. The corresponding element from the  $W$  matrix is set zero to apply the effect.

### Scaling Effect

In large networks, where the total number of neurons  $N$  greatly exceeds  $C$ , the inputs to any given neuron can be considered as random samples from the broader network activity without requiring adjustment or scaling based on  $N$ 's size. In other words, in larger networks, the effects of individual fluctuations are more diffuse due to the increased number of interactions. Essentially, the larger population acts as a buffer, diluting individual variances and leading to a more stable overall response ([Figure 4](#)).

This method ensures that each neuron has a specific, predetermined number of connections, reflecting a more realistic approach to modeling neural circuits, especially under conditions that simulate real biological activity and neuronal dynamics.

## **Homogenous 2:8 Inhibitory/Excitatory Population**

In neural networks, be they biological or artificial, the balance between excitatory and inhibitory neurons plays a crucial role in shaping the network's function, stability, and dynamics. A commonly cited ratio in many parts of the brain is that of 80% excitatory to 20% inhibitory neurons. This balance is critical for preventing runaway excitation, which can lead to epileptic seizures, or excessive inhibition, which can dampen neural processing and responsiveness.

A 2:8 ratio implies a significantly higher proportion of excitatory neurons compared to inhibitory neurons. This ratio suggests several key dynamics and characteristics of such a neural network:

1. **Network Excitability:** With a dominant population of excitatory neurons, the network would inherently have a higher baseline of excitability. This configuration could be beneficial for rapid signal propagation and heightened sensitivity to stimuli. However, it also risks hyperexcitability.
2. **Plasticity and Learning:** The higher proportion of excitatory neurons could facilitate more robust synaptic plasticity mechanisms, such as long-term potentiation (LTP), crucial for learning and memory.
3. **Regulatory Mechanisms:** To prevent dysfunction, such a network would require highly efficient and responsive inhibitory mechanisms. This might involve not just direct inhibition by GABAergic neurons but also modulatory influences, such as neuromodulators or feedback mechanisms, to finely tune the excitatory activity.
4. **Vulnerability to Disruption:** With the excitatory population so prominently outnumbering the inhibitory one, the network might be particularly sensitive to disruptions in the balance between excitation and inhibition.

## **Implementation**

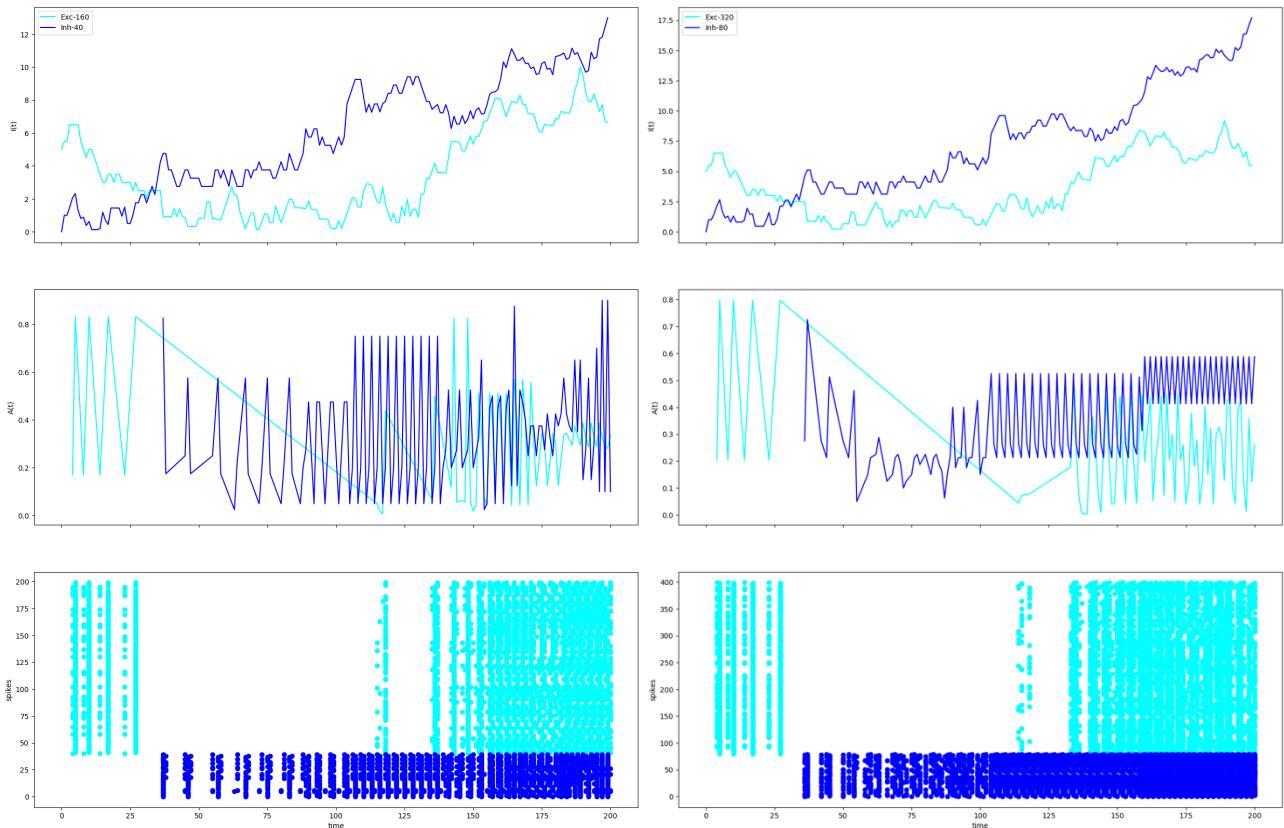
The network consists of  $N$  neurons, divided according to a 2:8 ratio into two groups: excitatory and inhibitory. For this ratio, we set the variable  $p$  to 0.8. This gives us two neuron groups: one excitatory group with  $N \cdot p$  neurons and one inhibitory group with  $N \cdot (1-p)$  neurons. All neurons follow the Leaky Integrate-and-Fire (LIF) model, sharing identical parameters such as a threshold voltage of -50 mV, a resting potential of -70 mV, a reset potential of -70 mV, and a membrane time constant of 10 ms. The difference between groups lies in their input current: the excitatory group ( $ng1$ ) receives 5 units of current, whereas the inhibitory group ( $ng2$ ) receives none. Additionally, both groups are subject to a noisy current with a rate of 0.5 and a noise amplitude on the order of 0.1.

To illustrate the interactions between the two groups, two types of synapses are defined: one from the excitatory group to the inhibitory group with a coefficient ( $coef$ ) of 1, and another from

the inhibitory group to the excitatory group with a coefficient of -1. This setup showcases the inhibitory and excitatory behaviors between the groups.

### Scaling Effect

Observations from simulations with  $N=200$  and  $N=400$  are provided without any scaling methods applied ([Figure 5](#)). The dynamics depicted in Figure 5 illustrate that spikes in the excitatory population activate the inhibitory population. In turn, as the firing rate of the inhibitory group increases, the activity in the excitatory group tends to decrease. Furthermore, the input current peaks at 12 units in the smaller network ( $N=200$ ) and rises to 17.5 units in the larger network ( $N=400$ ), affecting both excitatory and inhibitory groups despite the input pattern remaining the same.



**Figure 5** The behavior of neural networks with a 2:8 ratio of excitatory (cyan) to inhibitory (blue) neurons across two sizes:  $N=200$  (left column) and  $N=400$  neurons (right column). Through three key visuals; namely current fluctuations over 200 iterations (first row), neuron activation levels (second row), and timing of neuron firings (raster plots in the last row), it reveals how network size impacts the dynamics between excitatory and inhibitory neurons. This link between size and network activity highlights the importance of scale in neural interactions.

### Simulation Analysis (Full Connectivity Scheme)

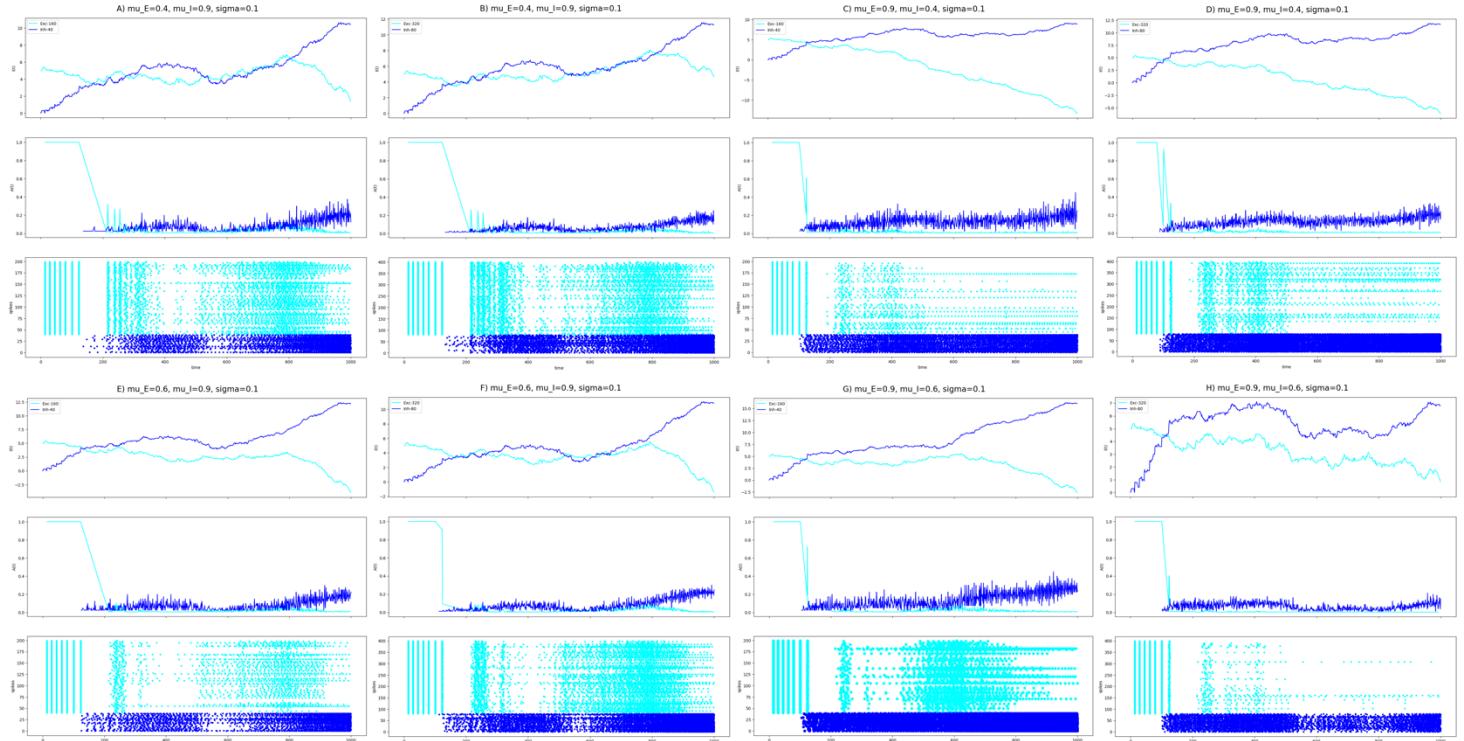
From the analysis of the plots in [Figure 6](#), it's evident that the activity levels of inhibitory and excitatory neurons decrease slightly as the population size doubles; however, the overall pattern remains consistent. Specifically, the fluctuation shapes of the current over 100 iterations are largely

unchanged, except in **6H**, where an increased population size of  $N=400$  alters the shape. Generally, the peak values of the currents increase modestly, about 2-2.5 times higher with larger populations. This increase suggests a slight upward shift in the model's plot with larger populations, where even the minimum current values are higher.

Interestingly, an increase in the current of inhibitory neurons typically leads to a decrease in the excitatory neurons' current, highlighting the inhibitory effect. Yet, [Figure 6H](#) deviates from this pattern by showing a lowered current and altered shape compared to its  $N=200$  counterpart.

In a full connectivity model, where every neuron is connected to every other neuron, the decrease in both the currents' values and overall activity levels as the size of the network increases can be attributed to several factors:

1. Dilution of Excitatory Input: As the network size increases, the excitatory input each neuron receives is distributed across more neurons. This dilution can lower the individual impact of excitatory inputs, leading to a decrease in overall activity levels.
2. Increased Inhibitory Influence: If the model includes inhibitory neurons, their influence becomes more pronounced in a fully connected network as it scales. The increased inhibitory signaling can effectively suppress excitatory activity, leading to lower current values and reduced network activity.



**Figure 6** **A)** Homogenous population of excitatory  $E$  (cyan) and inhibitory  $I$  (blue) neurons of size 200. **B)** Same  $\mu$ (mean) and  $\sigma$ (sigma) as simulation (A) with population size of  $N=400$ . **C)** Homogenous population of  $E$  and  $I$  neurons of  $N=200$ . **D)** Same  $\mu$  and  $\sigma$  as simulation (C) with  $N=400$ . **E)** Homogenous population of  $E$  and  $I$  neurons  $N=200$ . **F)** Same  $\mu$  and  $\sigma$  as simulation (E) with  $N=400$ . **G)** Homogenous population of  $E$  and  $I$  neurons of  $N=200$ . **H)** Same  $\mu$  and  $\sigma$  as simulation (G) with  $N=400$ .

3. Synaptic Scaling Rule: Some neural networks employ mechanisms like synaptic scaling, where synaptic strengths are normalized based on the size of the network. In larger networks, this normalization can reduce the effective strength of excitatory synapses, lowering the overall excitability of the network.
4. Network Homeostasis: Neural networks often have homeostatic mechanisms that maintain activity within a functional range. As network size increases, these mechanisms might work to prevent hyperactivity by reducing the effectiveness of synaptic inputs, thus lowering overall activity.
5. Signal Noise and Propagation Delay: In larger networks, the increased number of connections can introduce more noise and delay in signal propagation. These factors can disrupt the timing of neuronal firing, making coordinated activity more difficult and leading to a reduction in observable currents and activity levels.

In figures [6A and 6E](#), the inhibitory synapses' weight is approximately double that of excitatory synapses. This higher weight compensates for the relatively fewer inhibitory neurons, balancing the network by enabling these neurons to more effectively control excitatory neuron activity. This is contrasted in [6C and 6G](#), where excitatory synaptic weights are higher, leading to reduced excitatory neuron current and activity.

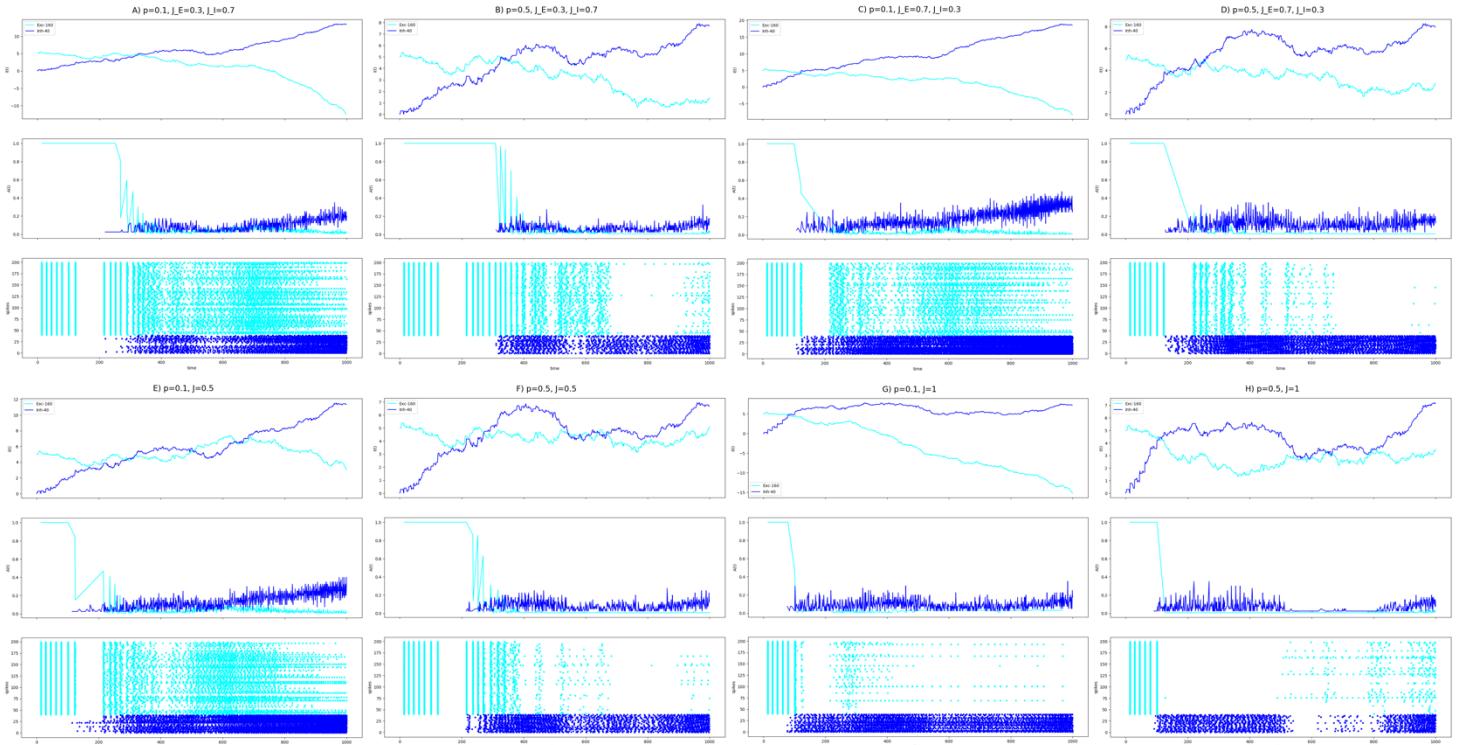
Additionally, the comparison between figures [6A and 6E](#) reveals that despite both having higher inhibitory than excitatory synaptic weights, the inhibitory synapses in [6E](#) exhibit a higher mean value. This results in more frequent spikes from inhibitory neurons around the 200th timestep and more substantial inhibition, thus a decreased activity in excitatory neurons at the 400th timestep in [6E](#) compared to [6A](#). Therefore, inhibitory neuron activation seems more dependent on excitatory neuron firings.

In [6C and 6G](#), the increased mean weight of excitatory synapses causes the inhibitory activity to rise as the current and firing rate of the excitatory population decrease. Notably, even minor excitatory activities can trigger inhibitory synapses to activate and subsequently dampen excitatory functions. This phenomenon is observed in columns 3 and 4, where despite sparse excitatory firing rates, inhibitory neuron activation remains high.

### **Simulation Analysis (Random coupling: Fixed coupling probability)**

[Figure 7](#) illustrates contrasting outcomes when increasing synaptic strength  $J$  for inhibitory versus excitatory neurons ([7A, 7C](#)): enhancing  $J$  in inhibitory neurons leads to reduced activation across the network, whereas increasing  $J$  for excitatory neurons results in heightened activity. The reason can be explained biologically.

Inhibitory neurons primarily secrete neurotransmitters like GABA in the mammalian brain, which decreases the likelihood that target neurons will fire. Increasing  $J$  for inhibitory neurons enhances their ability to suppress postsynaptic target neurons, leading to overall decreased network activation. The stronger the inhibitory signal, the more effectively it can prevent excitatory neurons from reaching the threshold needed to trigger an action potential.



**Figure 7** Homogenous population of excitatory  $E$  (cyan) and inhibitory  $I$  (blue) neurons of size 200.

Excitatory neurons, on the other hand, release neurotransmitters like glutamate, which increase the likelihood of firing in the postsynaptic neuron by depolarizing it, bringing it closer to the threshold for generating an action potential. When  $J$  increases for excitatory neurons, the efficacy of their connections improves, meaning they are better able to excite their postsynaptic partners. This leads to an overall increase in network activity because more neurons are pushed over the threshold needed to fire.

Inhibitory signals lead to reduced activity because they push neurons away from the action potential threshold, while excitatory signals increase activity by pushing neurons towards or over this threshold. While a uniform increase in  $J$  amplifies both excitatory and inhibitory signaling,

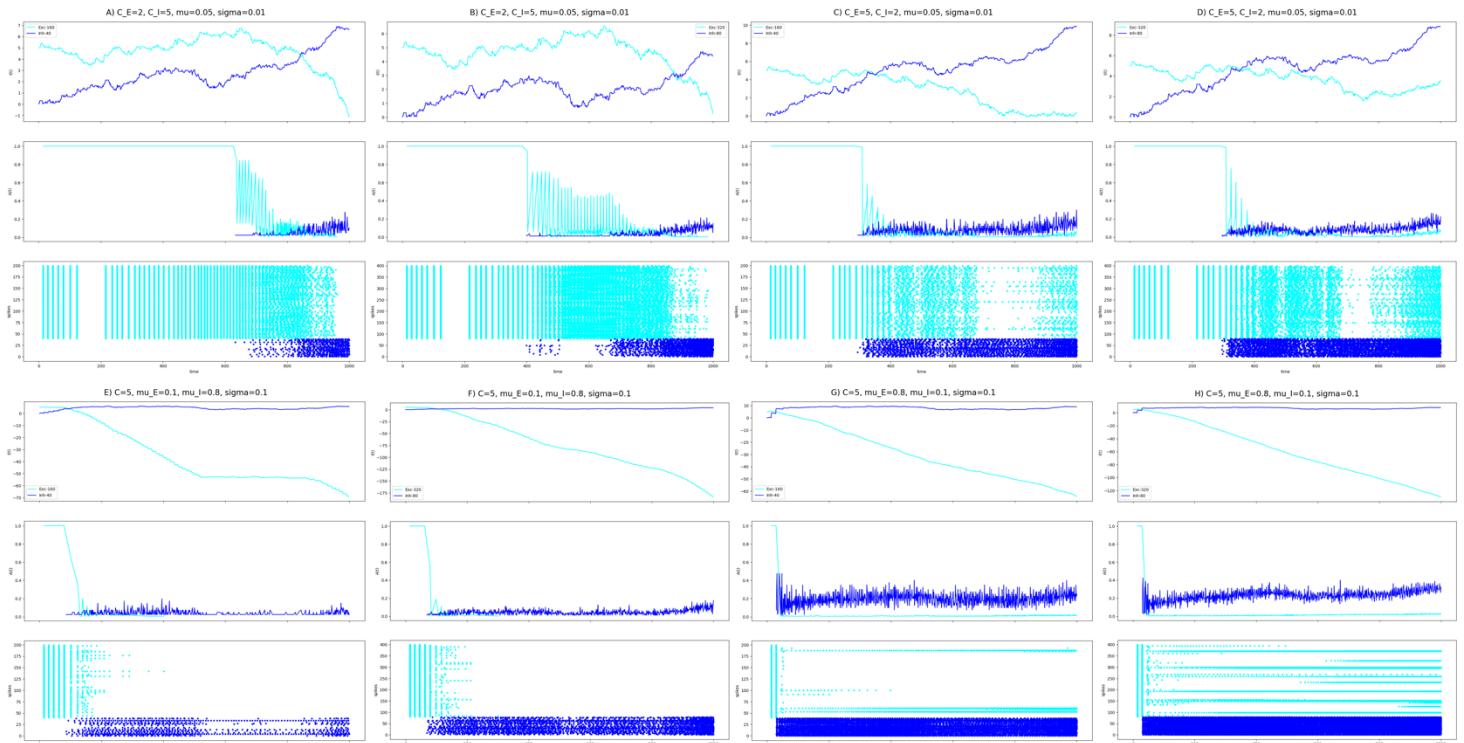
the disproportionate number of excitatory neurons means that their overall influence is significantly magnified. This could make it harder for the network to maintain a stable operating point, leading to periods of excessive activation or, conversely, suppression if the inhibitory response temporarily overtakes excitatory influences as it is seen in Figure 7E and 7G. An initial increase in the activity of excitatory neurons, due to stronger synaptic weights, could lead to a stronger activation of inhibitory neurons. Once activated, these inhibitory neurons can effectively suppress the activity of the excitatory neurons more than before, leading to an overall decrease in network activity. The stronger  $J$  means that even though inhibitory neurons are fewer, their influence is amplified, leading to a state where the excitatory neurons' influence is effectively repressed. In the figures 7C, 7E, and 7G an increase in coupling probability ( $p$ ) leads to decreased activity (7D, 7E, and 7H). This decrease can be attributed to several factors:

- Enhanced Network Inhibition: In cases [7E](#) and [7G](#), where  $J$  is increased particularly for excitatory neurons or both populations, the network might experience saturation or a state of balanced excitation and inhibition that reduces the overall activity due to a more efficient spreading of inhibition or excitation meeting its limits.
- Saturation and Feedback Inhibition: For excitatory populations with high  $J$ , increasing  $p$  may lead to a point where additional excitatory input doesn't further increase activity because neurons reach a firing rate ceiling or because inhibitory feedback mechanisms are activated to counteract further excitation and maintain network stability.

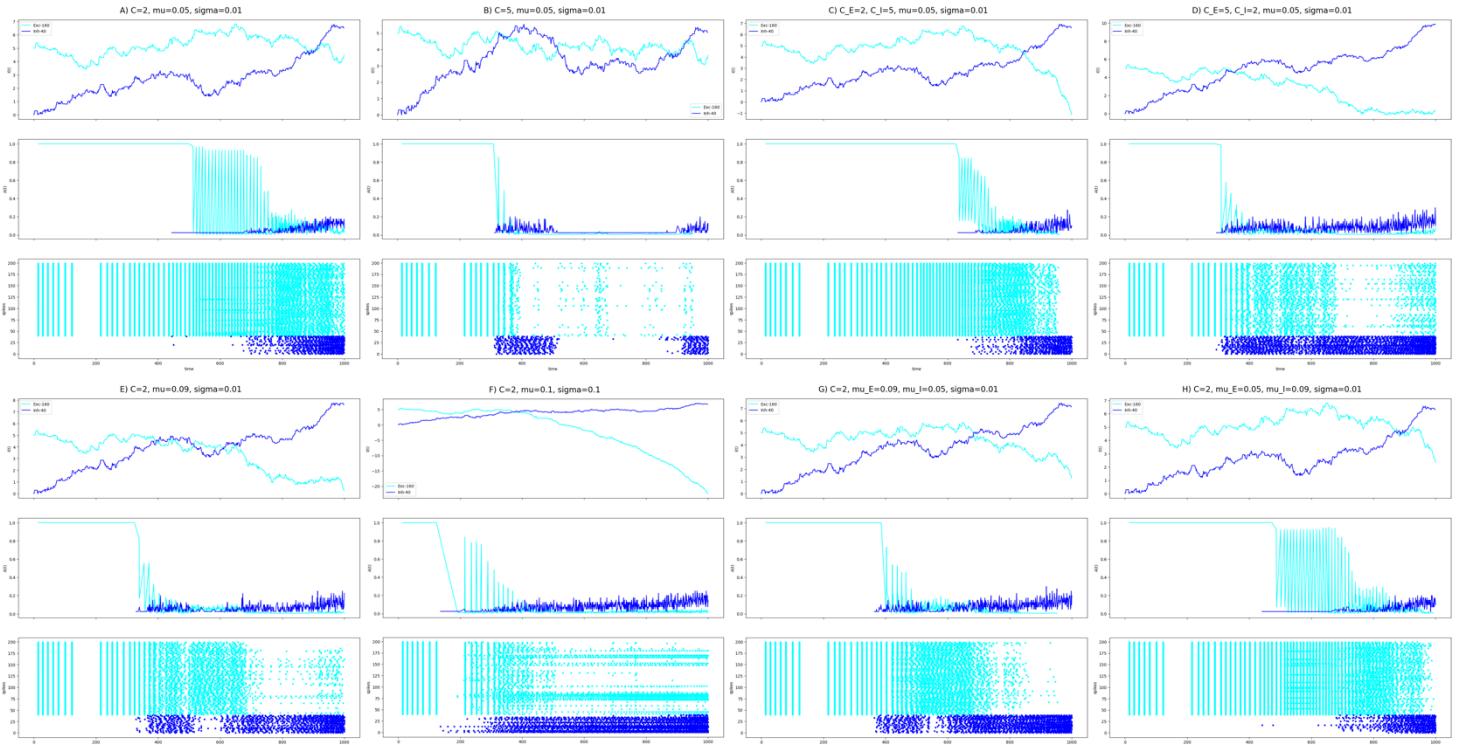
However, when  $J=0.7$  is higher solely for inhibitory neurons ([7A](#) and [7B](#)), increasing  $p$  seems to increase the activity. The significantly increased inhibitory strength might lead to a reorganization of network dynamics that favors conditions under which activity can propagate more efficiently. This could involve complex interactions where the increased precision of inhibitory control allows excitatory activity to flourish in a more regulated manner, thus overall increasing network activation under higher synaptic connectivity.

### Simulation Analysis (Random coupling: Fixed number of presynaptic partners)

Observations from [Figure 8](#) indicate that, as anticipated by this connectivity scheme, activity fluctuation levels across all simulations have decreased compared to equivalents with a larger population size of  $N=400$ .



**Figure 8** Homogenous population of excitatory  $E$  (cyan) and inhibitory  $I$  (blue) neurons with Random coupling: Fixed number of presynaptic partners scheme. First and third column (A, C, E, G) have population size of  $N=200$ , Second and forth column (B, D, F, H) have population size of  $N=400$ .



**Figure 9** Homogenous population of excitatory E (cyan) and inhibitory I (blue) neurons of size 200.

In this connectivity scheme, increasing the C value, as the simulations of [Figure 9](#) suggests, results in lower population activity ([9A](#), [9B](#)). This could be due to the fact that individual neurons receive inputs from a larger and potentially more diverse set of presynaptic partners. This can lead to a dilution effect where the intensity or efficiency of excitatory input to any single neuron is reduced, as inputs are spread out over a larger network. Another reason could be that with more connections per neuron, there's a higher chance that a proportion of these connections are inhibitory. The increased inhibitory input can suppress activity in the network, counteracting the effects of excitatory inputs.

In [9A](#) and [9C](#), the input remains largely unchanged; however, there is a noticeable decrease in activity, this trend is also seen when comparing [9B](#) and [9D](#). This reduction is attributed to an increase in inhibitory connections, which leads to diminished activation of excitatory neurons.

When the mean value across all neurons increases ([9E](#)), an increase in activity among inhibitory neurons is observed. the primary reason is tied to the integral role inhibitory neurons play in the neural network's balance and dynamics. An overall increase in the mean neuronal activity implies more excitatory signals are being generated. In response, inhibitory neurons become more active as part of a feedback mechanism to prevent excessive excitation and maintain network stability. This heightened activity in inhibitory neurons helps balance the excitatory inputs, ensuring the network doesn't become hyperactive, which could lead to dysfunctional signaling or excitotoxicity. As excitatory activity rises across the network, inhibitory neurons receive more synaptic input. Since these neurons are sensitive to the overall level of excitation in the network,

their activity increases in proportion to the heightened excitatory signals. This natural response helps modulate and control the network's excitation levels, maintaining a functional state of neural dynamics.

In 9F where excitatory input current decreases while overall neural activity still remains, the network may also engage in compensatory activity amongst different neuronal populations. Regions or clusters of neurons that receive stronger excitatory input might compensate for areas experiencing reduced excitatory currents, maintaining an overall stable level of network activity. In addition, as excitatory input currents decrease, the efficiency of inhibitory neurons in modulating neural activity could increase. This means that even with reduced excitatory signals, inhibitory neurons adapt by fine-tuning their responses or by increasing their synaptic efficacy. This adaptation helps maintain a balance between excitation and inhibition, preserving neural network activity.

In 9H, when the mean value for inhibitory neurons increases there seems to be no significant change in the overall network activity. This can be attributed to the network's inherent balancing mechanisms and the dynamic interplay between excitatory and inhibitory processes. If this increase in inhibitory activity matches the level of excitatory activity, the overall activity level of the network can remain relatively unchanged, illustrating the network's ability to adjust and maintain equilibrium.

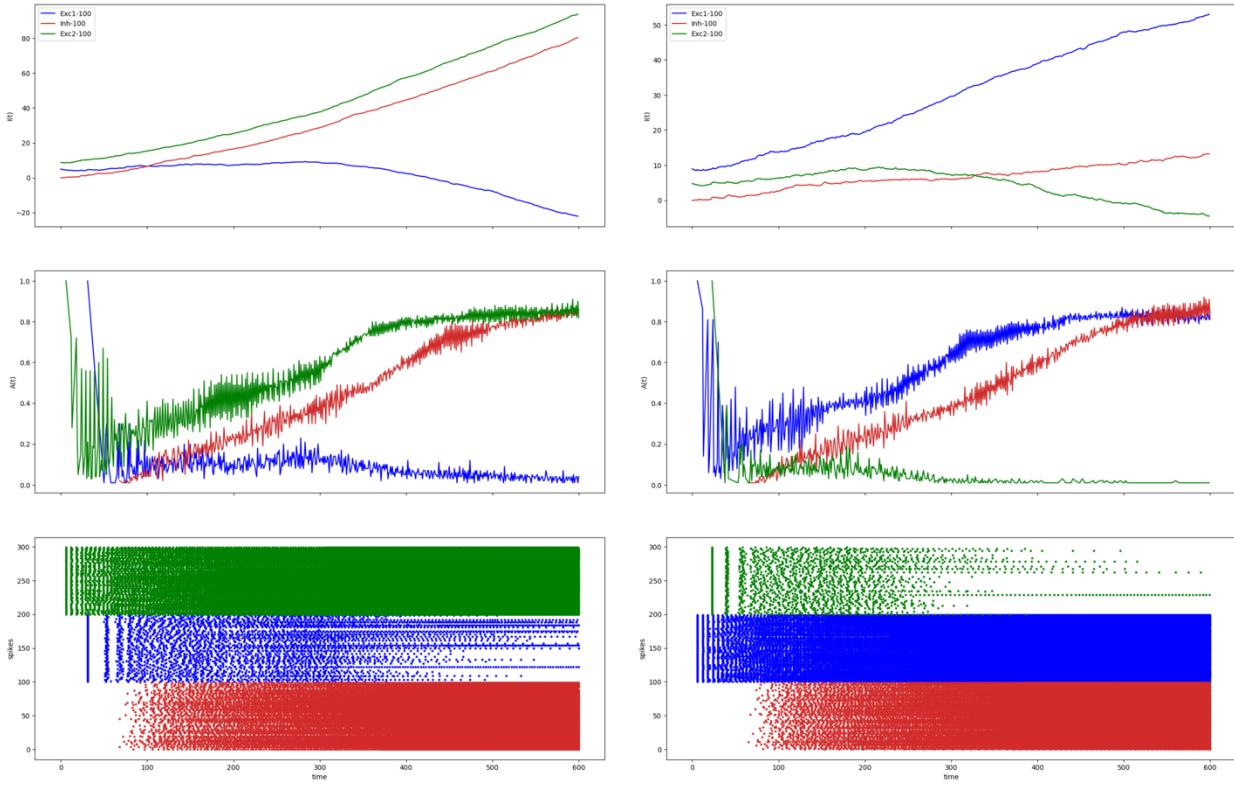
Also, The architecture of neural networks includes feedback and feedforward inhibitory circuits that modulate neuronal activity. An increase in the inhibitory mean value would enhance these circuits' role in fine-tuning and regulating the flow of neural information ensuring that any potential disruptions in activity levels are quickly addressed and normalized, contributing to the stability of overall network activity.

Another reason could be due to the fact that C=2, which is relatively small. Therefore, the increased mean value of inhibitory neurons may not effectively alter the network's activity.

## Decision-Making via Shared Inhibitory Mechanism

This setup includes two population of excitatory neurons linked to a shared population of inhibitory neurons. Excitatory neurons within the same group are interconnected with a specific connection weight, denoted as  $w_{EE}$ . The inhibitory group receives connections from and sends connections back to the excitatory groups with weights  $w_{IE}$  and  $w_{EI}$ , respectively. The network is maintaining a state of asynchronous, irregular activity.

When an external stimulus is applied and favors one of the excitatory groups by enhancing its coherence and input, that group's activity  $A_E$ , <sub>1</sub> intensifies. This increased activity then activates the inhibitory pool, which in turn releases inhibition back onto both excitatory groups. However, the group that received the initial stimulus is able to withstand this inhibition due to its higher stimulation level, leading to the suppression of activity in the other excitatory group. This dynamic ensures that at any given moment, only one of the two excitatory populations is active, creating a competitive interaction between them that is mediated by the common inhibitory neurons. The *winner* of this competition is the group receiving the stronger or more favorable external stimulus. In situations where external stimulation is absent or neutrally weak, both excitatory populations revert to a state of low activity.



**Figure 10** Homogenous population of excitatory E1 (blue), E2 (green) and inhibitory I (red) neurons of size 100. In the left column, we see the simulation where excitatory group E1 receives an input of value 5, and excitatory group E2 receives a higher input of value 9. The right column shows the opposite scenario: E1 receives an input of value 9, while E2 gets a lower input of value 5. In both simulations, the inhibitory group (I) does not receive any input. The results illustrate that the group with the higher input consistently demonstrates increased activity levels.

## Implementation

The network consists of three neuron groups, all having size of  $N=100$ . All neurons follow the Leaky Integrate-and-Fire (LIF) model, sharing identical parameters such as a threshold voltage of -50 mV, a resting potential of -70 mV, a reset potential of -70 mV, and a membrane time constant of 10 ms. The difference between groups lies in their input current: the first group (ng1) consists of excitatory neurons and receives 5 units of current, the inhibitory group (ng2) receives 0 and the third group (ng3), which is also excitatory, receives 9 units of input. Naturally we expect the population with  $I=9$  to have higher activity level ('winner' population). Additionally, all groups are subject to a noisy current with a rate of 0.9 and a noise amplitude on the order of 0.1. To illustrate the interactions between the groups, six types of *synapse groups* (*sg*) are defined:

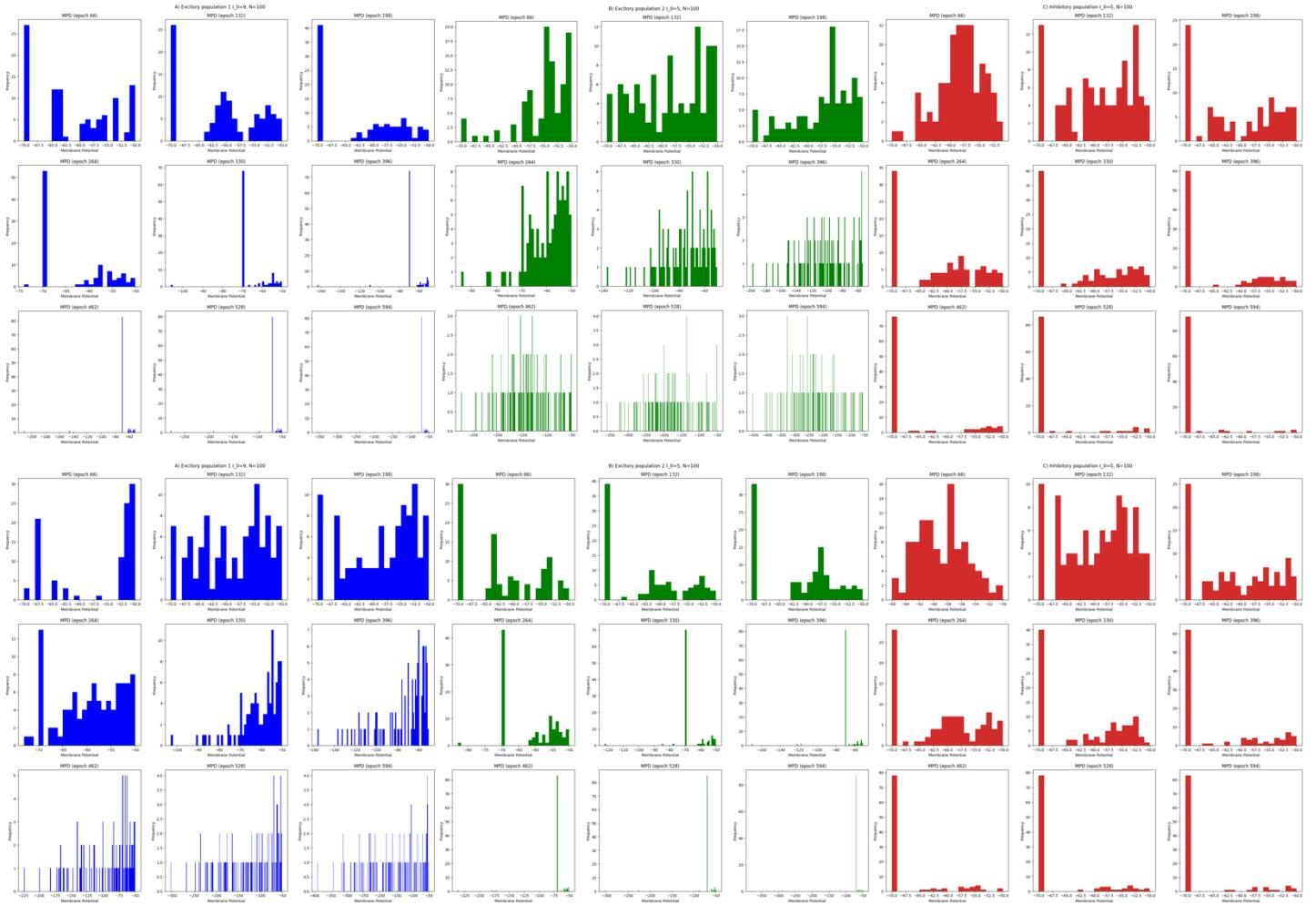
	<i>Type</i>	<i>Source</i>	<i>Destination</i>	<i>Size</i>	<i>Coefficient</i>
<i>sg1</i>	E	ng1	ng1	ng1.size	1
<i>sg2</i>	E	ng1	ng2	ng1.size + ng2.size	1
<i>sg3</i>	E	ng3	ng3	ng3.size	1
<i>sg4</i>	E	ng3	ng2	ng3.size + ng2.size	1
<i>sg5</i>	I	ng2	ng1	ng1.size + ng2.size	-1
<i>g6</i>	I	ng2	ng3	ng3.size + ng2.size	-1

## Simulation Result

[Figure 10](#) illustrates that when excitatory neuron group 3 (depicted in green) receives a higher input value than excitatory neuron group 1 (depicted in blue), it exhibits higher activity levels. Conversely, if excitatory neuron group 3 receives a lower input value than excitatory neuron group 1, its activity levels decrease, as shown in the right column. Thus, the neuron group receiving the larger input emerges as the “winner” in terms of activity. Meanwhile, the inhibitory neuron groups, shown in red, play a role in maintaining the balance between the two excitatory groups, with their current and activity levels remaining relatively consistent across both simulations.

[Figure 11](#) shows the membrane potential distribution across 9 specific time points during the overall iteration period of 600 epochs for the three populations. The membrane potential distribution within the inhibitory group remains relatively stable throughout the simulations. This stability reflects their role in maintaining network balance. Despite the competitive dynamics between the excitatory populations, the inhibitory neurons do not significantly alter their membrane potential, which indicates their function in providing a consistent regulatory influence over the activity of excitatory neurons, rather than directly participating in the competition. The excitatory population that receives the higher input (and thus “wins” the competition), their neurons tend to converge towards a single membrane potential value near -50 mV. This convergence suggests a high level of synchronization within the population as it becomes more active. The convergence around a somewhat depolarized value (-50 mV) compared to a resting potential indicates increased neuronal activity and a tendency towards a more uniform state among the neurons in this group.

The excitatory population that receives lower input shows a significantly different membrane potential distribution. Their potentials spread across a broad range, from -350 to -50 mV, following a normal Gaussian distribution. This diversity in membrane potentials suggests that, despite losing the competition for inputs, this population still maintains a wide range of activity levels among its neurons. The spread resembles the variability one might expect in a less activated or resting state, where individual neurons are not synchronized or driven towards a specific action potential threshold collectively.

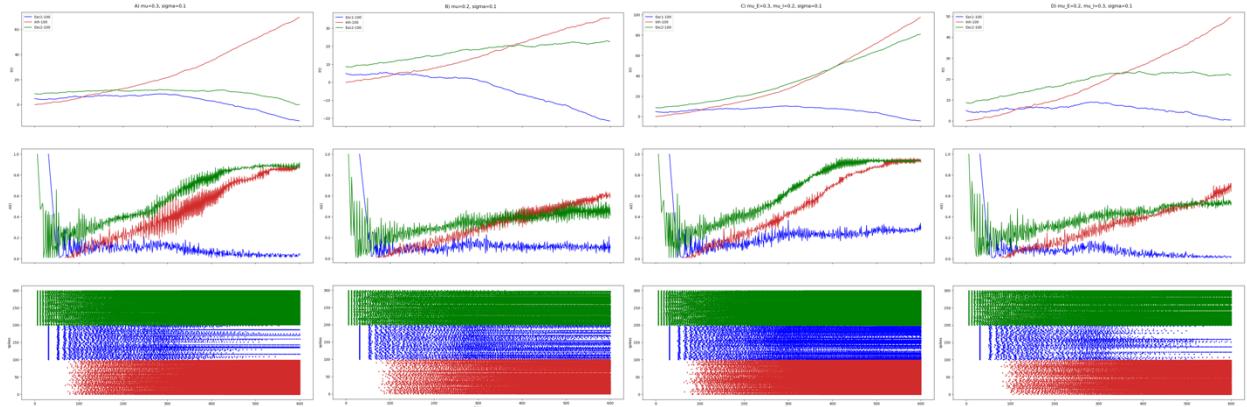


**Figure 11** Membrane potential distribution across nine specific time points during the overall iteration period for the three populations. In the first row, excitatory population 1 (blue) receives an input of value 9, while excitatory population 2 receives an input of value 5. The second row reverses this arrangement.

### Simulation Analysis (Full Connectivity Scheme)

As the average strength of the connections (mean value) in the network goes up, both the activity of the inhibitory neurons and the leading excitatory group (winner) rise ([Figure 12](#)). However, the activity of the trailing excitatory group (loser) does not change, and it might even decrease.

This may be because of the ‘winner-takes-all’ dynamics; In networks with competitive dynamics such as a winner-takes-all mechanism, the population that initially has a slight advantage in activity (either through stronger input or due to randomness in initial conditions) suppresses the activity of the other excitatory population indirectly through the inhibitory population. As the mean synaptic strength increases, this dominant (“winner”) group’s activity further amplifies, owing to stronger input-output gain.



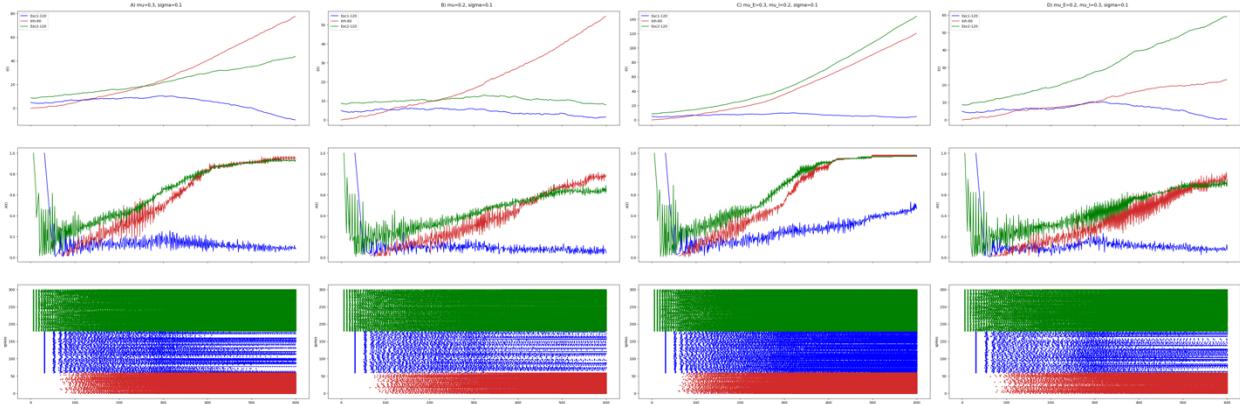
**Figure 12** Variation in the mean ( $\mu$ ) of Gaussian weight distribution of **full connectivity scheme** across three populations. Excitatory neuron group 2 (green) receives an input of  $I=9$ , while excitatory neuron group 1 (blue) receives an input of  $I=5$ . All three populations are of size 100.

In addition, The inhibitory population, responds to the overall increase in network activity by becoming more active. This activity increase in the inhibitory neurons acts to suppress the activity in both excitatory populations. However, because the "winner" excitatory population has a higher activity level, it can sustain its activity against inhibitory feedback better than the "loser" population. The inhibitory feedback effectively stabilizes the network by preventing runaway excitation and maintaining the suppression of the less active excitatory population.

Furthermore, since the interaction is mediated through the inhibitory population. The increase in mean synaptic strength primarily boosts the coupling efficiency between the populations, rather than changing the dynamics directly between the two excitatory populations. Given that the "winner" population has stronger or more effective input (directly or through network dynamics), its activity is enhanced, further suppressing the "loser" through the inhibitory feedback loop.

The above ‘*winner-takes-all*’ dynamics can also account for observation made from [12C](#) and [12D](#); When only the strength of connections for excitatory neurons increases, both the winner group and the inhibitory neurons experience a big jump in both their incoming signals and their overall activity, to the point where they might hit their highest possible levels of activity. However, the loser group shows a smaller increase in activity. Also in [12D](#) the activity of the winner population decrease less than the other.

In [Figure 13](#), the network remains at a total size of 300 neurons, but the sizes of the different populations within it have changed. The proportions are now in a 2:2:1 ratio, with the inhibitory population being smaller than the two excitatory populations. In panels [13A](#) and [13B](#), lowering the average strength of connections for all neurons results in the excitatory neurons' input currents staying consistent, while the input current to the smaller inhibitory population increases significantly. By the end of the iteration, the inhibitory population's activity surpasses that of both excitatory populations.



**Figure 13** Variation in the mean ( $\mu$ ) of Gaussian weight distribution of **full connectivity scheme** across three populations. Excitatory neuron group 2 (green) receives an input of  $I=9$ , while excitatory neuron group 1 (blue) receives an input of  $I=5$ . Excitatory populations has size of  $N=120$ , while inhibitory population is of size  $N=60$ .

The reason for this observation may be the fact despite the decrease in mean synaptic value, the input current to the excitatory neurons remains steady. This stability can result from the excitatory neurons compensating for the decreased strength by leveraging their larger numbers or possibly through homeostatic mechanisms that adjust their responsiveness to maintain activity levels.

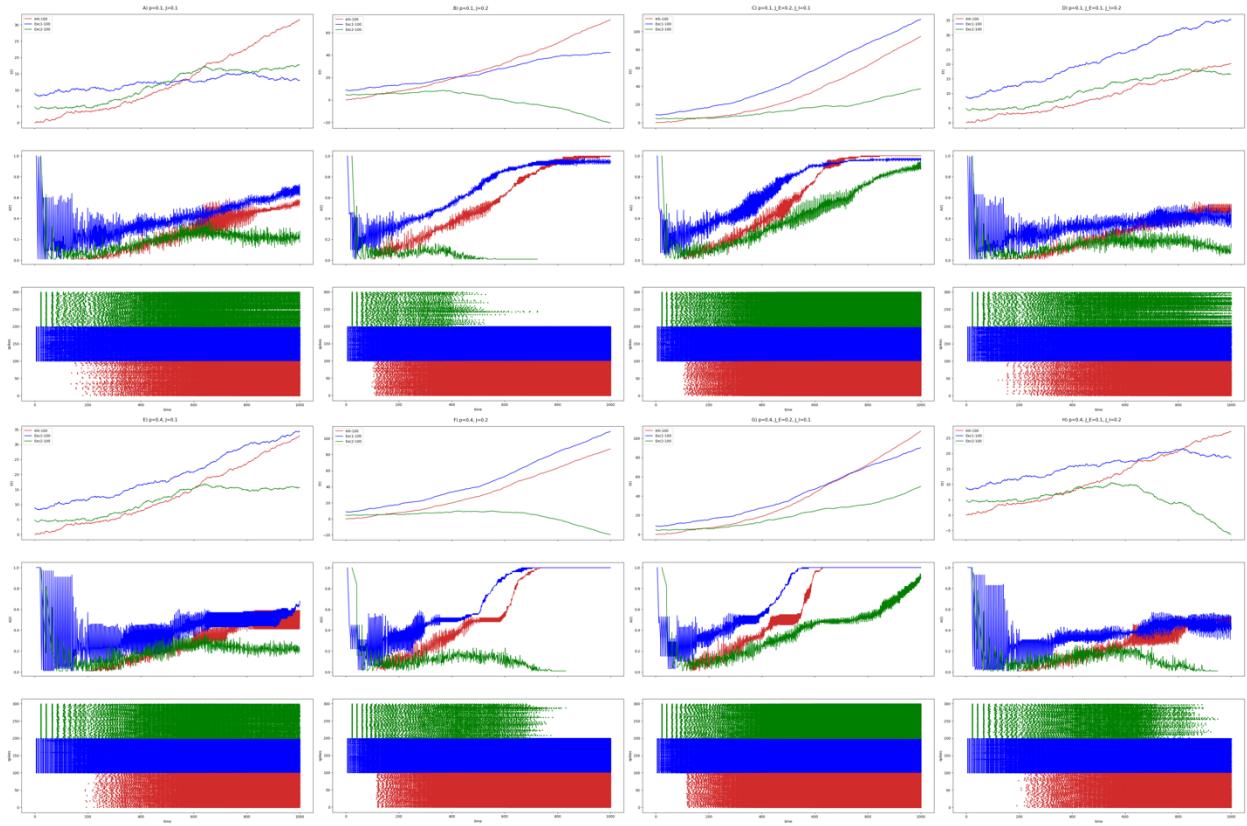
The smaller inhibitory population, on the other hand, experiences a significant increase in its input current. With fewer neurons to distribute the incoming excitatory signals, each inhibitory neuron might receive disproportionately more input relative to its excitatory counterparts, especially if synaptic scaling or other forms of plasticity are at play, making the inhibition more potent.

By the end of the iteration, the activity of the inhibitory population exceeds that of the excitatory populations. Inhibitory neurons, responding to excitatory signals, can increase their activity to counterbalance the excitatory drive, effectively increasing their suppression of the excitatory populations. Also, the smaller size of the inhibitory population might allow for faster and more coordinated responses. Moreover, inhibitory neurons often have mechanisms that allow them to be more responsive to increases in synaptic input, making their activity more robust in the face of network-wide changes.

#### Simulation Analysis (Random coupling: Fixed coupling probability)

With a constant connection probability of 0.1 and 0.4 in a neural network (as shown in the first two columns of [Figure 14](#)), increasing the strength of the neural connections (denoted as  $J$ ) quickly boosts the activity levels of both the inhibitory and the dominant (winner) groups of neurons.

As a consequence, the activity of the underperforming (loser) group drops to zero. Additionally, the last two columns of Figure 14 reveal that when the synaptic strength ( $J$ ) is increased for the inhibitory group specifically, the activity across all neuron groups decreases. On the other hand, enhancing the synaptic strength for the excitatory group results in increased activity for every group, including the initially underperforming (loser) group, which eventually reaches peak

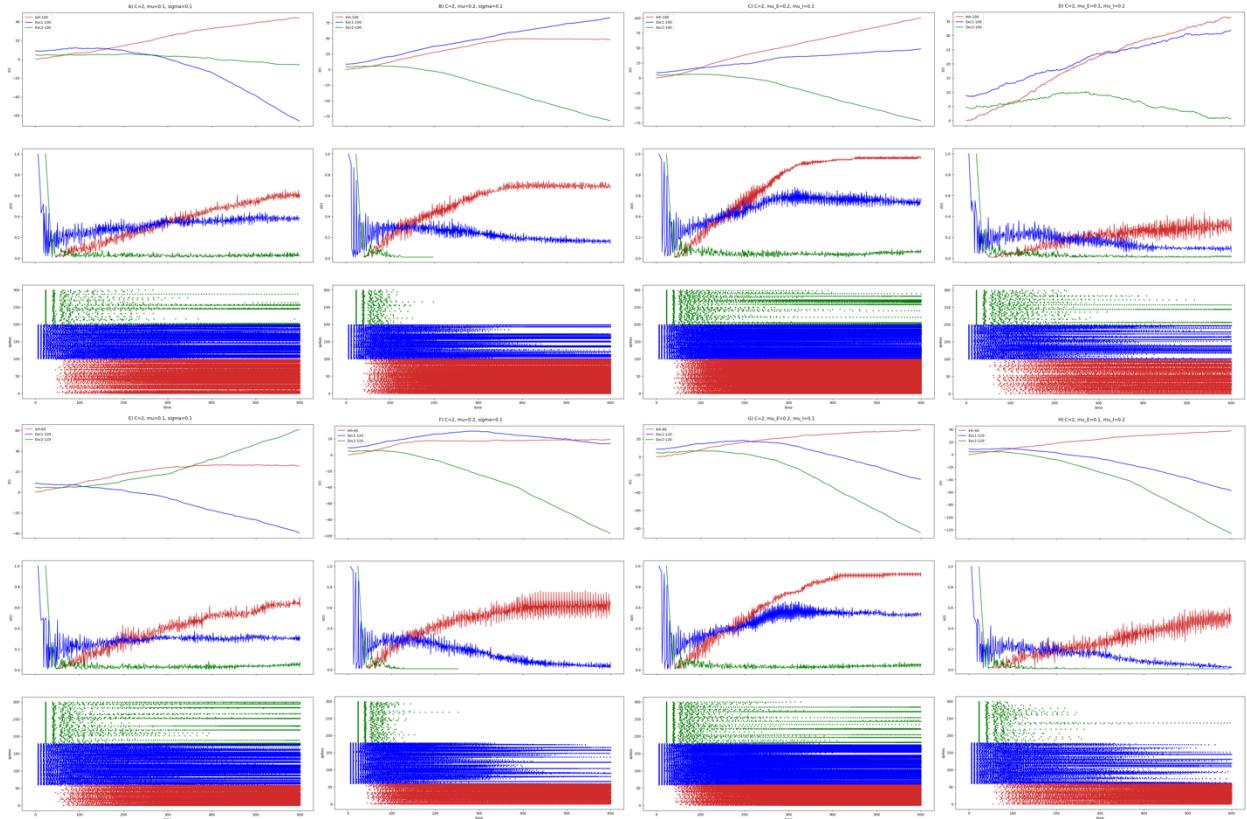


**Figure 14** Variation in the coupling probability( $p$ ) and  $J$  of **fixed coupling probability** connectivity scheme across three populations. Excitatory neuron group 2 (green) receives an input of  $I=5$ , while excitatory neuron group 1 (blue) receives an input of  $I=9$ . All three populations are of size 100.

activity after several iterations, although the dominant (winner) group achieves this peak activity more quickly.

At fixed probabilities  $p = 0.1$  and  $0.4$ , increasing  $J$  directly influences the intensity of the connections within the network. For both the inhibitory and winner populations, a higher  $J$  leads to more potent synaptic inputs whenever neurons fire, thus increasing their activity more rapidly. The increased activity in these populations, especially from the inhibitory neurons, suppresses the loser population's activity, eventually reducing it to zero. This suppression occurs because inhibitory neurons, having stronger connections, can more effectively dampen the activity of the loser population by outweighing its received excitatory inputs.

An increase in  $J$  within the inhibitory population specifically amplifies the effectiveness of their inhibitory synapses on other neurons. This heightened inhibitory influence can overtake the excitatory inputs, leading to a general decrease in network activity. The inhibitory neurons effectively suppress not just one target population but all neuronal activity to some extent, illustrating how a stronger inhibitory control can dampen overall network excitability. Conversely, when  $J$  is increased for the excitatory population, it results in escalated excitatory synaptic inputs across the network.



**Figure 15** Variation in the presynaptic partners ( $C$ ) of Fixed number of presynaptic partners and gaussian mean value in three homogenous population. Excitatory neuron group 2 (green) receives an input of  $I=5$ , while excitatory neuron group 1 (blue) receives an input of  $I=9$ . In A, B, C, and D all three populations are of size 100 while in E, F, G, and, H excitatory populations has size of  $N=120$ , while inhibitory population is of size  $N=60$ .

This overall increase in excitation elevates activity levels for all groups, including the loser population. However, the winner population reaches full activity more swiftly because the heightened excitatory drive favors those neurons already more active due to their initial conditions or network topology. As excitation builds up, even neurons in the loser population, which initially have reduced activity, can eventually achieve full activation, given sufficient iterative cycles and synaptic input.

#### Simulation Analysis (Random coupling: Fixed number of presynaptic partners)

In [Figure 15A](#) and [B](#), increasing the mean value causes an increase in activity and current of inhibitory population while it decreases the activity and current of excitatory population. For the inhibitory population, boosting the mean value of their connections increases both their activity levels and the current they experience. This happens because inhibitory neurons, which serve to dampen or reduce the activity of other neurons, become more effective with stronger connections. As their connections strengthen, they are better able to suppress the activity of other neurons,

including excitatory neurons, leading to an increase in their own activity levels and the inhibitory current they generate.

On the other hand, the activity and current of the excitatory population decrease as the mean value of their connections increases. Although it might seem counterintuitive, this effect can be attributed to the enhanced efficiency of the inhibitory neurons. As inhibitory connections strengthen, they more effectively counteract the excitatory signals, thus reducing the overall activity and current within the excitatory population.

Excitatory neurons generally aim to increase activity within the network, but when the inhibitory neurons that are connected to them become more potent, the overall excitatory effect is diminished. This dynamic interplay between excitatory and inhibitory forces is crucial for the balance and proper function of neural networks, enabling complex processing and preventing excessive activity that could lead to undesirable outcomes like overstimulation or neural noise.

In a neural network where each neuron is linked to a fixed number of other neurons, boosting the average strength of just the excitatory neuron connections leads to more activity and electrical flow across all types of neurons. On the other side, when only the average strength of inhibitory neuron connections is increased, the activity and electrical flow throughout the network decreases for all neuron types ([Figure 15C](#) and [D](#)).

When the mean value of *excitatory neuron connections* is increased, all neuron populations see an increase in activity and current. This happens because excitatory neurons promote activity in other neurons. Making their connections stronger means they are more effective at exciting other neurons across the network, thus amplifying the overall network activity. This includes not just other excitatory neurons but inhibitory ones as well. Since excitatory inputs to inhibitory neurons are also enhanced, this, in turn, increases the activity of inhibitory neurons, which then more effectively regulate the network activity but don't overpower the increased excitatory drive.

Conversely, increasing the mean value of connections for *inhibitory neurons alone* results in decreased activity and current across all populations. Inhibitory neurons act to suppress or dampen the activity of other neurons. Strengthening their connections makes them more effective at this role, thus reducing the activity levels not just of excitatory neurons, but of the network as a whole. Even though inhibitory neurons are also more active in their role of suppression, the net effect on the network is a reduction in overall activity and current flows, because they counteract the excitatory influences more efficiently.

Therefore, the nature of the neuron (excitatory vs. inhibitory) whose connection strength is increased determines the overall effect on network activity. Strengthening excitatory connections boosts overall network activity, whereas enhancing inhibitory connections damps down activity across the board.

Both above trends are present for [E, F, G and H](#) that the sizes of populations are variant. This is because each neuron, regardless of its type, maintains a constant number of connections. This setup means that every neuron, whether it's part of the larger excitatory group or the smaller inhibitory group, influences the network to the same extent through its fixed number of connections.

So, despite the varying proportions of neuron types, the impact on overall network activity is balanced by the uniformity in connectivity. Each neuron contributes equally to the network's function, ensuring that changes in population size don't skew the activity levels. This design

maintains a stable activity pattern across the network, irrespective of the sizes of the neuron populations. In this specific connectivity scheme, the effect of population size on activity and current levels is notably neutralized, in contrast to other two schemes where activity and current levels vary depending on the size of each population.