

# The Wilson-Cowan model

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**Project:** Study the dynamics of the Wilson-Cowan Model

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# 1 Abstract

This report outlines our efforts to understand the Wilson-Cowan model using a Jupyter notebook. The Wilson-Cowan model is a fundamental framework for studying how large groups of neurons, both excitatory and inhibitory, interact to produce various patterns of neural activity. Unlike other models that focus on individual neurons, this one looks at the collective behavior and how it's shaped by the network of connections within these groups.

Our project consists of building a Jupyter notebook that allows us to simulate the model, adjust its parameters, and observe the outcomes. At the basis of this model we have the following assumptions: populations' cells are close in space, random interconnections but there is at least one path connecting any two cells within the population. Key component of the Wilson-Cowan model is the firing rate of neurons, which represents the overall activity levels of each population. Moreover, the interactions between excitatory and inhibitory populations create feedback mechanisms that can lead to unfolding of neural activity patterns and peculiar dynamical phenomenon such as oscillations, stable fixed points, or transient responses.

## 2 Introduction

The Jupiter Notebook is build on the given GitHub repository [1]. The code aims to investigate the behavior of the Wilson-Cowan model for understanding the collective dynamics of neural populations. The implementation involves simulations, visualizations, and analysis of the model's response under different conditions.

Three useful functions are defined in the beginning of the notebook, they will be used to plot functions. These functions, namely *plot-FI-inverse()*, *plot-FI-EI()*, and *my-test-plot()*, respectively facilitate the visualization of the inverse of the activation function, the activation function for excitatory and inhibitory populations, and the activities of the populations over time.

Afterwards, some helper functions are introduced to be used in the Wilson-Cowan model simulation. These functions include:

- *default-pars*: Defines default parameters for the model.
- *F - dF*: Represent the population activation function and its derivative.
- *F-inv*: Computes the inverse of the population activation function.
- *EIderivs*: Calculates the time derivatives for excitatory and inhibitory variables.
- *simulate-wc*: Simulates the Wilson-Cowan model given specific parameters.

These functions collectively form the foundation for the model's dynamics and numerical simulations. The code proceeds to simulate the Wilson-Cowan model with default parameters and plot the activities of excitatory and inhibitory populations under different initial conditions. The *simulate-wc()* function is employed, taking advantage of the previously defined helper functions.

### 3 Tasks

#### 3.1 Task 1: Find the steady states

We plot the nullcline curves of the Wilson-Cowan model, and find an estimate for the points of intersection using a numerical root-finding algorithm.

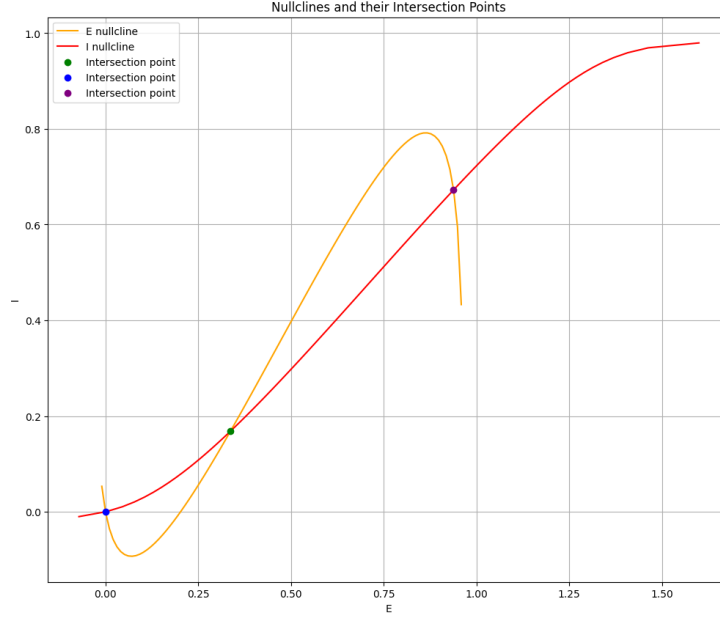


Figure 1: Nullcline for Excitatory and Inhibitory neural populations

A nullcline is a curve in a phase space where one of the variables of a dynamical system does not change, meaning its derivative is zero. For Wilson-Cowan model, nullclines provide valuable insights into the equilibrium states and the behaviour of the system. Hence, we are able to identify and classify states of the dynamical system. Points where the Excitatory Nullcline and the Inhibitory Nullcline intersect are critical points of the system. At these points, both the excitatory and inhibitory populations are at values where their respective derivatives are zero, suggesting a steady state.

#### 3.2 Task 2: Classification of Critical Points

After the research for critical points, now we had to classify them. We performed a stability analysis on the Wilson-Cowan model, a representation of the dynamics between excitatory (E) and inhibitory (I) neural populations. The *get-eig-Jacobian(pars)* function is employed to calculate the eigenvalues of the Jacobian matrix at specified fixed points of intersection on the nullclines where the system's derivatives vanish. These eigenvalues are crucial for determining

the nature of each fixed point: a fixed point is classified as **stable** (green points) if all eigenvalues have negative real parts, suggesting that the system will return to this point after small perturbations, and **unstable** (purple point) if any eigenvalue has a positive real part, indicating that the system will diverge from this point.

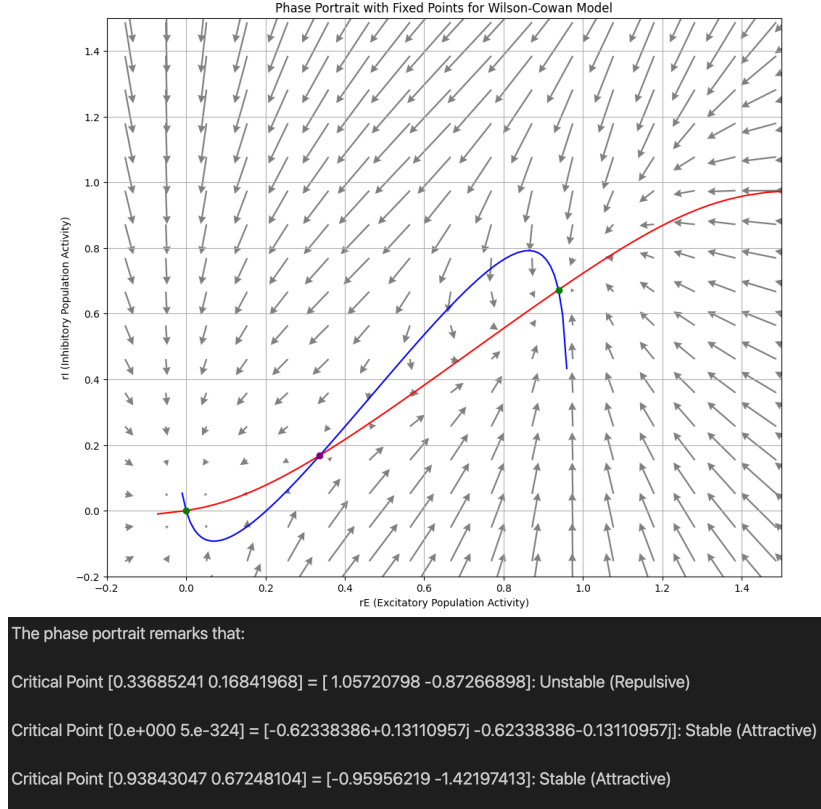


Figure 2: Phase portrait for fixed points, Wilson-Cowan model

The critical points, emphasized by the graphical representation as phase portrait, offer valuable insights into the potential long-term patterns of activity between the excitatory and inhibitory populations.

### 3.3 Task 3: Change the non-linearities

The nonlinearity of the population activation function helps shaping the dynamics of excitatory and inhibitory neural populations. The original model uses a sigmoidal function to describe the population activation, but by modifying this nonlinearity we can have produce significant effects on the behaviour of the system. We proceeded by modifying several times, with different parameter combinations, the following values:

- **Gain value:** Modification to the gain in the sigmoidal activation function of the Wilson-Cowan model affects the shape of the nullclines and, consequently, the number and nature of their intersection points, which represent the model's fixed points or steady states. The gain parameter controls the **steepness** of the sigmoidal activation function and, of course, has a relevant impact on nullclines' shapes.
- **Threshold value:** By adjusting the threshold, we are essentially tuning the sensitivity of the neuronal populations, and producing a shifting of the curves.

### 3.3.1 Higher Gain

Increasing the gain while keeping the threshold constant allows us to observe the effects of heightened sensitivity to inputs near the threshold. High gain implies that a small change in input can lead to a large change in the population's firing rate, pushing the system more rapidly towards saturation. This sensitivity can lead to scenarios where the system stabilizes more quickly to particular states, reducing the complexity of the system's dynamics as reflected in fewer fixed points. We have made several tests in the code, but here we just report representative figures that summarize the concepts.

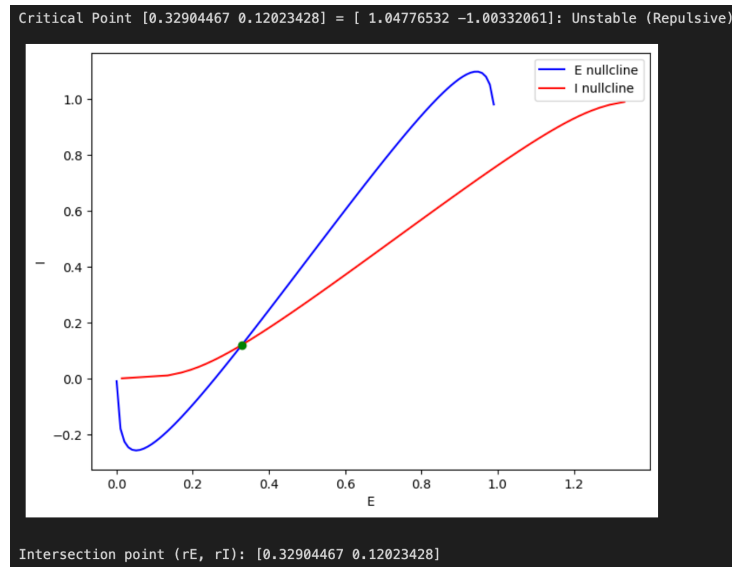


Figure 3: Nullclines - Increasing Gain parameter value

### 3.3.2 Lower Gain

Lowering the gain while keeping the threshold constant makes the response functions become so insensitive that they barely respond to inputs below a certain level; as a result, the system's dynamics change significantly, potentially eliminating any non-zero fixed points. The neural populations might only be able to achieve equilibrium at very low activity levels (close to zero) because the reduced gain prevents them from reaching higher activity levels without certain inputs.

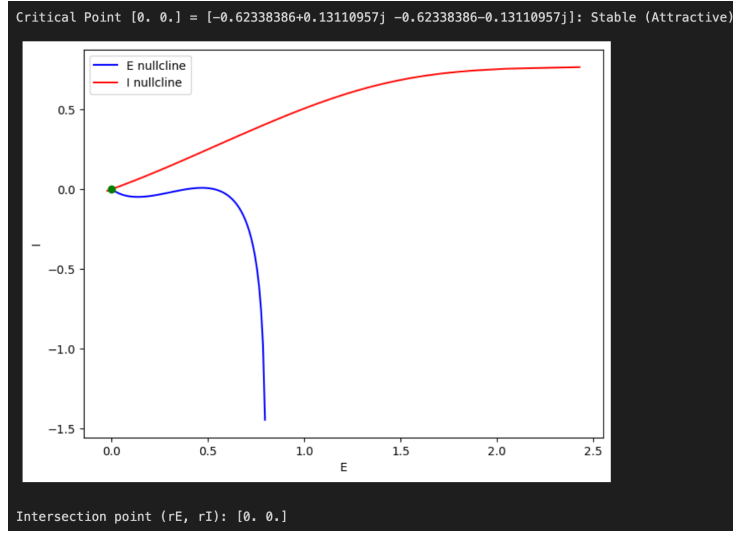


Figure 4: Nullclines - Decreasing Gain parameter value

### 3.3.3 Higher Threshold

By increasing the thresholds, the system's behavior has been altered, leading to a lack of stable equilibrium within the observed activity range. .

The excitatory (E) nullcline (in blue) has a visible rightward shift, indicating that a higher level of the E population activity is now required to achieve the point where the derivative (change over time) is zero. Without an intersection in the displayed range, the system may exhibit a persistent dynamic activity rather than settling into a fixed point.

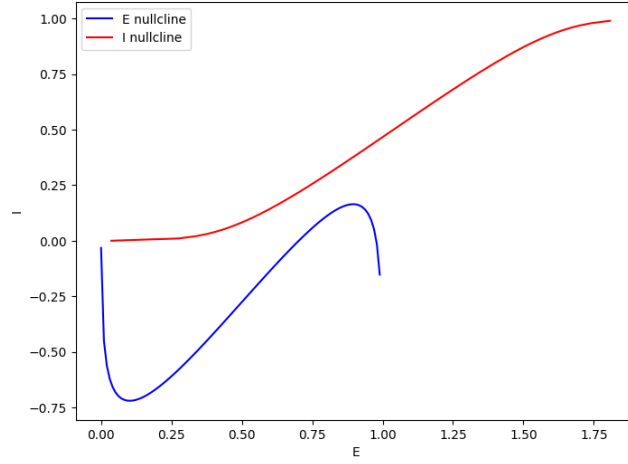


Figure 5: Nullclines - Increasing Threshold parameter value

### 3.3.4 Lower Threshold

Decreasing the threshold makes the system more responsive to lower inputs, which is reflected in the nullclines shifting towards the origin. This can lead to an increase in the likelihood of the system reaching an active state with less external stimulation.

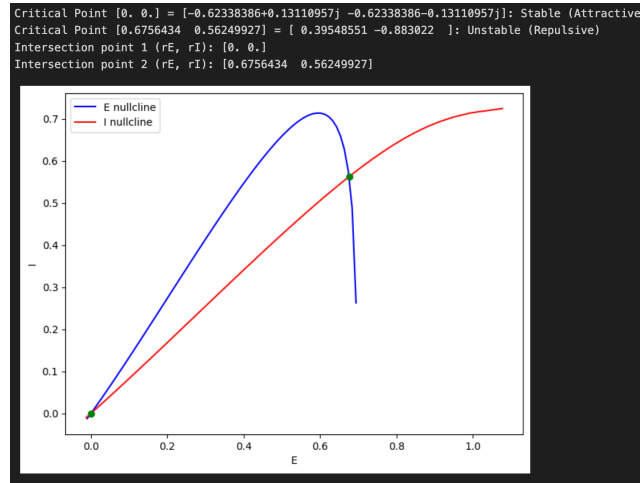


Figure 6: Nullclines - Decreasing Threshold parameter value



### 3.4 Task 4: Change the connection strengths parameters

The connection strengths are defined by four parameters:  $w_{EE}$ ,  $w_{EI}$ ,  $w_{IE}$ , and  $w_{II}$ . These parameters determine the strength of connections between excitatory (E) and inhibitory (I) neural populations in the model.

- **$w_{EE}$** : This parameter represents the strength of connections between excitatory neurons. Higher values of  $w_{EE}$  increase the influence of excitatory neurons on each other.
- **$w_{EI}$** :  $w_{EI}$  represents the strength of inhibitory connections onto excitatory neurons. Larger values of  $w_{EI}$  imply stronger inhibitory influence on excitatory neurons. Higher values of  $w_{EI}$  are likely to result in greater suppression of excitatory activity, leading to more controlled and regulated behavior.
- **$w_{IE}$** :  $w_{IE}$  indicates the strength of connections from excitatory to inhibitory neurons. Increased  $w_{IE}$  values amplify the excitatory influence on inhibitory neurons.
- **$w_{II}$** : This parameter,  $w_{II}$ , denotes the strength of inhibitory connections between inhibitory neurons. Larger values of  $w_{II}$  increase the inhibitory influence on inhibitory neurons. Increasing  $w_{II}$  may lead to stronger inhibitory feedback within the inhibitory population, potentially resulting in more stable and controlled dynamics.

#### 3.4.1 Enhanced Inhibitory to Excitatory influence

Increasing the inhibitory effect on the excitatory population can demonstrate the impact of strong inhibition on network dynamics.

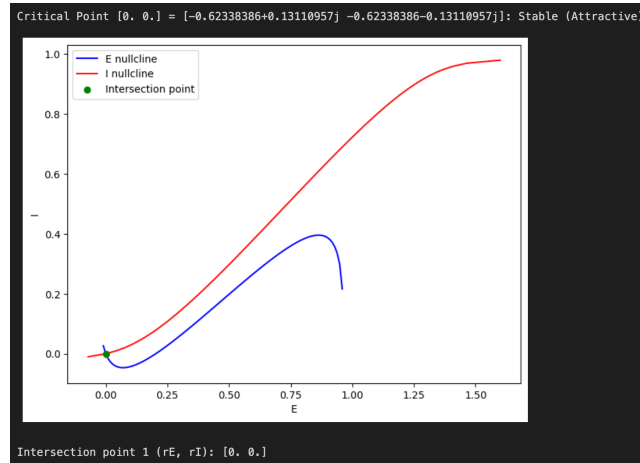


Figure 7: Nullclines - Enhanced Inhibitory effect on Excitatory population

- $w_{EE} = 9.0$  (Kept the same for direct comparison)
- $w_{EI} = 8.0$  (Increased from 4.0 to enhance the inhibitory effect on E)
- $w_{IE} = 13.0$  (Kept the same)
- $w_{II} = 11.0$  (Kept the same)

This set leads to a scenario where the increased inhibitory effect on the excitatory population suppresses excitatory activity more strongly, potentially stabilizing the system at lower activity levels and leading to the disappearance of certain unstable fixed points. In fact, the E nullcline is shifted, reflecting that it takes more excitatory drive to overcome inhibition and increase the firing rate of excitatory neurons.

### 3.4.2 Balanced mutual inhibition

In this case we adjusted the model to reflect a scenario where both populations strongly inhibit each other.

- $w_{EE} = 9.0$  (Kept the same)
- $w_{EI} = 13.0$  (Increased to match the strength of E to I)
- $w_{IE} = 13.0$  (Kept the same)
- $w_{II} = 9.0$  (Decreased to match the strength of E to E)

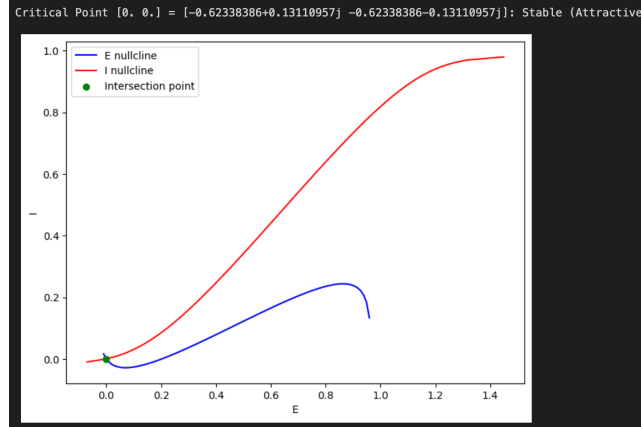


Figure 8: Nullclines - Balanced mutual inhibition

The excitatory population now has a stronger inhibitory effect on itself, which leads to a decrease in overall excitatory activity since the excitatory neurons are more heavily suppressed by the inhibitory neurons. On the other hand, the inhibitory neurons have less influence on each other, potentially leading to less

overall inhibitory activity. The combination of increased  $wEI$  and decreased  $wII$  results in a balancing act between excitation and inhibition in the network. The network becomes less likely to exhibit spontaneous activity since the excitatory neurons are kept "in check" by strong inhibition, and the inhibitory neurons are less likely to suppress each other.

### 3.4.3 General parameter variation

In this last scenario we wanted to understand the response of the model when parameters values completely changes.

- $wEE = 16.0$ ; A high value for  $wEE$  indicates that the excitatory population has a strong positive feedback, which tends to increase its own activity level.
- $wEI = 3.0$ ; A lower value for  $wEI$  compared to  $wEE$  suggests that while inhibition can affect the excitatory population, it is not strong enough to fully counteract the high excitatory feedback. This allows the excitatory population to reach higher activity levels before being modulated by inhibition.
- $wIE = 7.0$ ; This value indicates that as the excitatory activity increases, it will moderately and slightly increase the inhibitory population's activity.
- $wII = 6.0$ ; The  $wII$  value, which is close to  $wEI$ , suggests that the inhibitory population has a feedback mechanism that is not so strong.

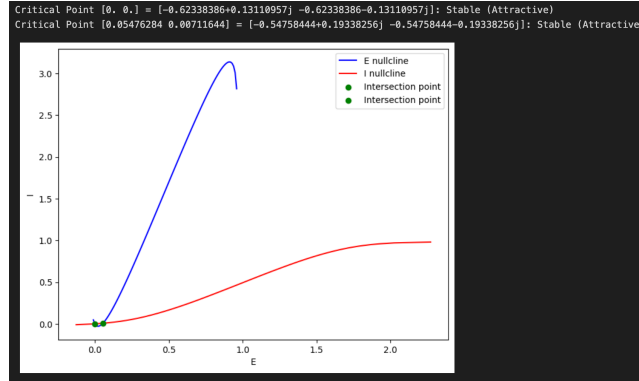


Figure 9: Nullclines - General strength connections variation

### 3.5 Task 5: Add an external stimulus

#### 3.5.1 Mild External Drive

With both populations receiving mild external stimulation, the balance of excitation and inhibition shifts, resulting in a new equilibrium state that reflects the combined effect of internal network dynamics and external inputs. This represents a scenario where the network adapts to a constant external drive, which could be a simplified model of sensory input or other constant external factors affecting neural activity. The critical points suggests that both the excitatory and inhibitory populations have reached a new equilibrium with higher activity levels than in the absence of stimulation.

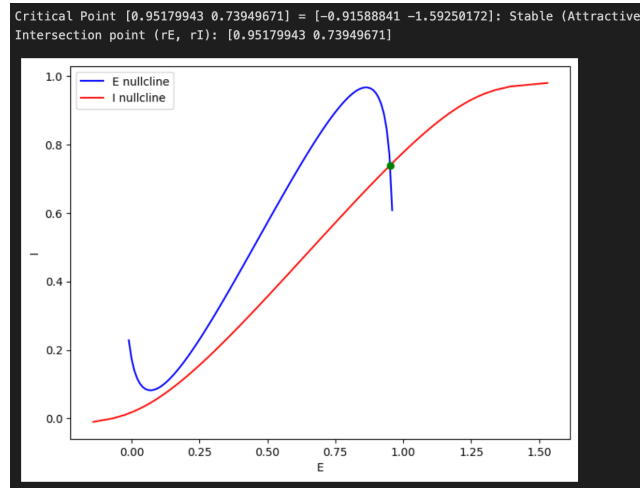


Figure 10: Nullclines - Mild external drive

#### 3.5.2 Strong External Drive

A stronger external drive means that both populations are receiving more input, which shifts their nullclines. This can lead to changes in the location and number of fixed points (equilibria). In fact, unlike previous configurations, with strong external drive does not appear an intersection point (at least in the considered range). This suggests that, in the given conditions, the system does not settle into a fixed point within this range of E and I population activities. This could indicate that the system's behavior is more dynamic overall.

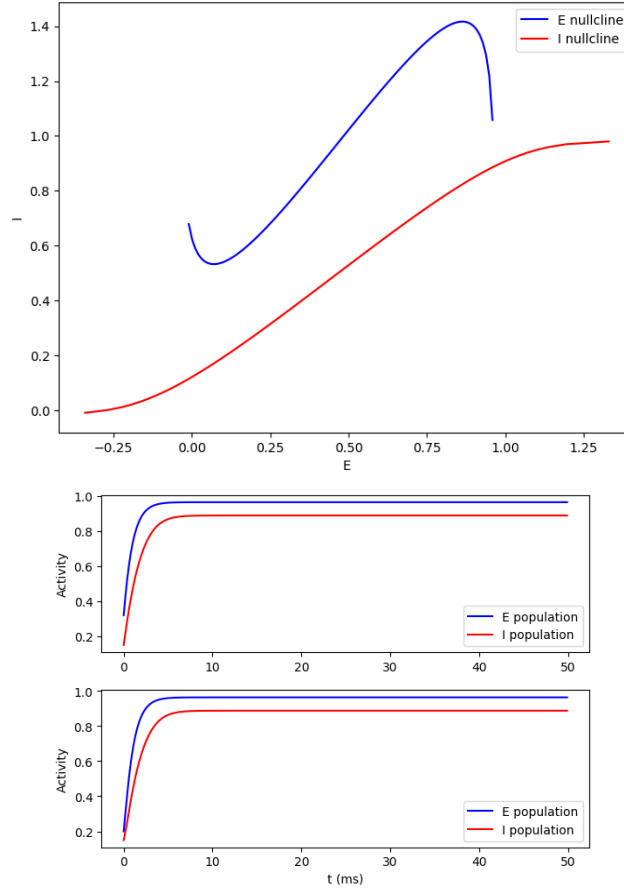


Figure 11: Nullclines - Strong external drive

### 3.5.3 Unbalanced External drive

An unbalanced external drive might represent situations where one population is more strongly driven than the other, such as during certain cognitive tasks. In this scenario our model could go towards a saturation condition.

Saturation of the neural response refers to a condition in neural systems where increases in stimulus intensity no longer lead to corresponding increases in neural activity. This happens when the neurons or neural populations have reached their maximum firing rates and cannot fire any faster, no matter how strong the stimulus becomes.

In the context of the Wilson-Cowan model, which uses sigmoidal activation functions to represent the response of neural populations to inputs, saturation occurs at the upper asymptote of the sigmoid function. Here's how saturation manifests in such models:

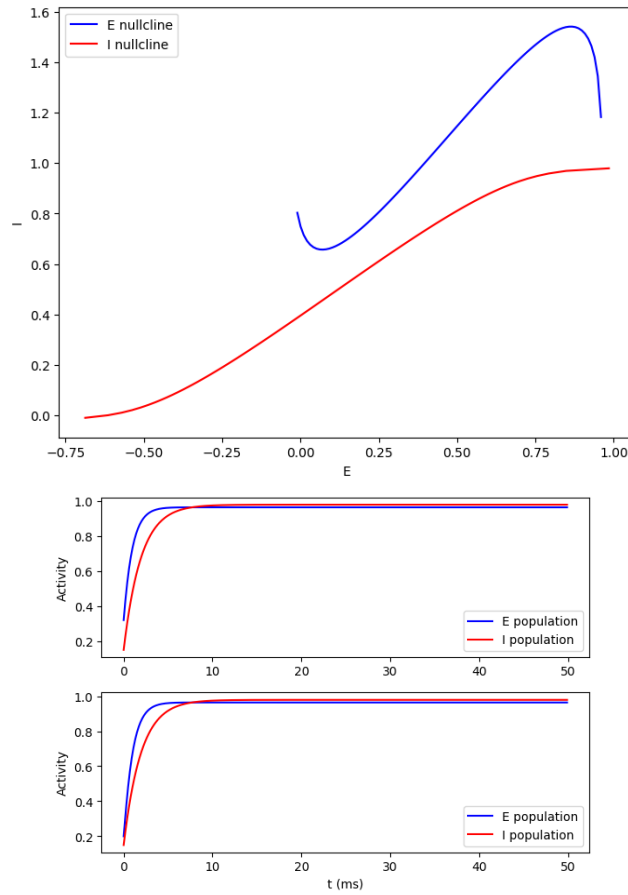


Figure 12: Nullclines - Unbalanced external drive

**Upper Asymptote of Sigmoid Function:** The sigmoid function approaches a value (typically 1 for normalized functions) asymptotically. As the input increases, the function's value gets closer to this upper limit but never actually reaches it.

**Physiological Limits:** Neurons have a refractory period after firing an action potential during which they cannot fire again. This physical limitation caps the maximum firing rate of a neuron or neural population.

Saturation is an important aspect of neural systems, reflecting both their computational properties and their limitations.

## References

- [1] [https://github.com/alessiomarta/mean\\_field\\_wilson\\_cowan\\_lecture/blob/master/wilson\\_cowan\\_jupyter.ipynb](https://github.com/alessiomarta/mean_field_wilson_cowan_lecture/blob/master/wilson_cowan_jupyter.ipynb).