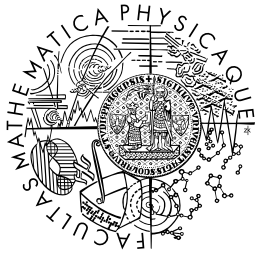

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BACHELOR THESIS

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Effect of inhibitory neuron subtypes on neural population dynamics

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Study programme: Mathematics (B1101)
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Title: Effect of inhibitory neuron subtypes on neural population dynamics

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Abstract: Canonical models of neural circuits rarely differentiate between inhibitory population subtypes. Due to the recent development of optogenetic techniques, the effect of distinct inhibitory classes may now be analysed. This thesis adapts a neural population model of the mouse visual cortex and analyses the stability of equilibria with overall connectivity of the network as a bifurcation parameter. A stability result for small values of the parameter is obtained.

Keywords: inhibitory neuron subtypes population activity neural circuit modeling visual cortex dynamical system

Contents

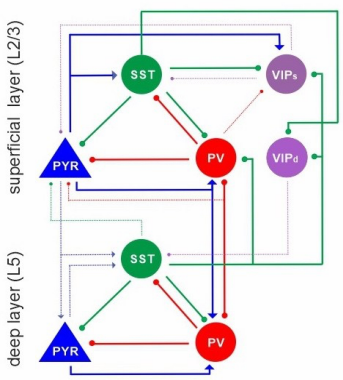
Introduction

The aim of this thesis is to analyse the properties of equilibria of a dynamical system describing a subcircuit of a neural population model adapted from the article *Hahn et al., 2022*. Specifically, presented are proofs of the existence and uniqueness of an equilibrium point and a limited analysis of its stability dependent on a bifurcation parameter g.

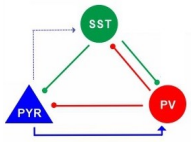
We considered a system of three ordinary differential equations (ODE), with each variable representing the mean activity of a distinct neural population, and a reduced system of two ODE's corresponding to one population being silenced.

Motivation and Background

The cited article numerically investigated the computational properties of a microcircuit in the mouse visual cortex, comprising of two layers with four distinct neuron cell types – their connectivity diagram shown in *Figure 1(a)*. The vertices of the graph represent distinct neural populations in two layers, namely the excitatory population of pyramidal neurons and four types of inhibitory populations, discernable by an expression of a characteristic protein. The presently analysed subcircuit, depicted in *Figure 1(b)*, consists only of one excitatory class (PYR) and two inhibitory populations (PV, SST).[1]



(a) The studied mouse visual cortex microcircuit. *Hahn et al., 2022* (CC BY).



(b) Presently analysed subcircuit. Modified, from *Hahn et al., 2022* (CC BY).

Figure 1: Connectivity diagrams of the neural population model

The connectivity is represented naturally via a 8×8 or 3×3 matrix respectively, with each connection weighted proportional to its strength, and signed according to the signalling population being excitatory or inhibitory.

[1] PYR stands for *pyramidal neurons*, PV stands for *parvalbumin*, SST for *somatostatin*

1. Neurons, models

For appreciation of the subsequent mathematical treatment of the system, this chapter provides a brief overview of some of the themes in computational neuroscience.

1.1 Basics of neural physiology and circuits

In simplest terms, a neuron (*Figure 1.1*) is a cell which utilises the difference in electric potential across its membrane to transmit information to other connected cells . It does not do so continuously – rather a short impulse is triggered in the cell's body when the difference in potential reaches a certain threshold. This impulse, called the action potential, then travels down the axon, to the axon terminal, where it causes a discharge of neurotransmitters into the synapse between its membrane and the post-synaptic cell's. These neurotransmitters then act on the post-synaptic cell's receptors interweaved in its membrane, to bring its membrane potential closer or farther from the threshold, depending on the pre-synaptic neuron being **excitatory** or **inhibitory** respectively. *(Bear et al., 2020)*

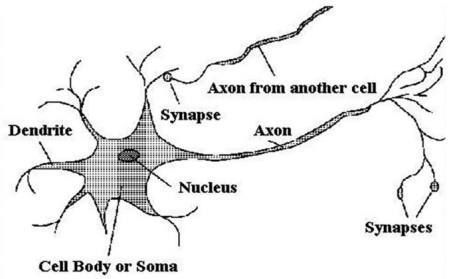


Figure 1.1: Scheme of neuron. Adapted, with permission from *Livshin, 2022*.

Such connections between neurons may be conceptualized as a weighted edge in a directed graph, with the sign of the weight determined by the pre-synaptic neuron's membership to either class. The strength of such a connection between neurons serves as a basis for the values in the connectivity matrix C defined in later text.

The activity of excitatory neurons – in present case pyramidal neurons – is believed to carry the signal being transmitted in neural circuits. This view is supported by the contrast in connectivity patterns with inhibitory neurons, which are usually connected only to local cells, whereas pyramidal neurons typically project to more distant areas. While there are only a few meaningfully distinguishable subtypes of excitary neurons, there is pronounced variability in the family of inhibitory interneurons *(Buzsáki, 2006)*. They are differentiated usually by their morphology, connectivity pattern or a characteristic expression of a protein, however a widely accepted classification is still lacking *(DeFelipe et al., 2013)*.

Most of the spotlight has historically been focused on the excitatory class – as illustrated by their alternate label *principal cells*, however this paradigm has

shifted to acknowledge the significance of the inhibitory class, namely that the diversity of cortical functions is provided by inhibition *(Buzsáki, 2006)*.

Despite this, canonical past and present neural microcircuit models only consider one inhibitory population *(Dayan and Abbott, 2005; Potjans and Diesmann, 2014; Mejias and Longtin, 2014)*, with a contemporary trend for incorporating higher differentiation or heterogeneity *(Hahn et al., 2022; Zhang et al., 2020)*.

This is due to past experimental limitations and subsequent lack of data, however recent applications of the selective optogenetic[1] silencing of individual interneuron classes *(Tremblay et al., 2016)* allowed meaningful development of models incorporating diversity among inhibitory interneurons.

1.2 Overview of common modeling approaches

Single neuron models. One of the prevalent simplifications in modeling is to consider the membrane potential of one neuron as a single variable, hence ignoring any morphology. Among these so-called *single-compartment models* are the praised Hodgin-Huxley model, or the simplified integrate-and-fire family of models.[2]

Presumably all the information a given neuron transmits is encoded in the "all-or-none" action potentials – respectively their timing. Because there is considerable variability in the sequence of spikes generated by a single stimulus, an alternative approach is to focus on their *mean firing rate* over some observation window. There is some ambiguity in the conceptualization of mean firing rate, however that is beyond the scope of this overview. *(Dayan and Abbott, 2005)*

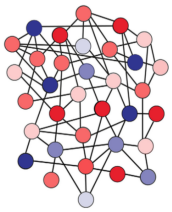


Figure 1.2: A scheme of a network, excitatory neurons colored red, inhibitory colored blue. *Mejias and Longtin, 2014* (CC BY).

Network models. A straightforward way to model a network of neurons (as in *Figure 1.2*), is to represent each connection in a connectivity matrix and produce a system of differential equations as large as the number of neurons. In such case each equation describes the change of a given neuron's membrane potential or mean firing rate, depending on the single neuron model chosen.

Computational limitations and analytical unwieldiness motivates factoring similar neurons into averaging units, which is not without its drawbacks, however it may also be more suitable for the analysis of higher cortical functions *(Wilson and Cowan, 1972)*. Another typical simplification is choosing the connecting

[1] A combination of optics and genetics, this technique usually uses adenoviruses that encode photosensitive protein to control specific neurons. By shining light on the target nerve region, the photosensitive protein encoded by the adenovirus is controlled. *(Chen et al., 2022)*

[2] Both decribed mathematically in the language of ordinary differential equations

strengths only from a few values, often distinguishing only between the excitatory or inhibitory populations as wholes – as was discussed above.

Both *spiking models* and *firing-rate models* (as they are named accordingly) have their virtues and pitfalls, and together are central tools in the exploration of networks of neurons. **(Dayan and Abbott, 2005)**

1.3 A population model of the mouse visual cortex

The authors in **Hahn et al., 2022** follow the firing rate model approach, where all neurons of the distinct populations are averaged into a single unit.[3] The connectivity matrix C is constructed from the measurement of synaptic strengths between individual neuron pairs, however the effective interaction between populations is dependent on the number of cells, which is not known. Hence an overall connectivity scaling parameter g was introduced, which may also be interpreted as the network size.

The description of the population activity in **Hahn et al., 2022** is based on the Wilson-Cowan firing-rate model **(Wilson and Cowan, 1972)**, and is cited as follows:

$$\tau \boldsymbol{r}' = -\boldsymbol{r} + \Phi(I_{circuit} + I_{ext}) + \eta,$$

where $\boldsymbol{r}$ is a vector of firing rates, τ is a vector of neuron specific time constant and Φ is an input-output transfer function.[4] The term $I_{circuit} = gC\boldsymbol{r}$ denotes inputs across the circuit population, I_{ext} are inputs from an external source, and η is a Gaussian noise term.

The transfer function Φ has $\varphi(r_i)$ as its i-th element, where the function φ is defined as

$$\varphi(x) = \frac{ax}{1 - e^{-x/b}},$$

for some positive constants a, b. Precise mathematical treatment of this function is presented in the Appendix (i).

Subcircuit model. As was previously indicated, we have made some modifications to the cited model for the purpose of this thesis. Most notably, we reduced the number of equations to three, dropped the I_{ext} and η terms, set τ equal in all its elements, and put $a = b = 1$. Less significant is a slight alteration of the elements in the matrix C to approximating fractions for computational leisure.

We will be analysing the following dynamical system:

$$\tau \boldsymbol{r}' = -\boldsymbol{r} + \Phi(gC\boldsymbol{r}),$$

where the elements of $\boldsymbol{r}$ correspond to the firing rates of the PYR, PV and SST populations respectively.

The effect of silencing the SST population can be achieved by restricting our view only to the top 2×2 submatrix of C and the corresponding two equations, as will be formulated more precisely in subsequent text. We will refer to these systems as SC and rSC respectively.

[3] The cited article has actually been even more lenient, factoring multiple different types of inhibitory interneurons into a single population

[4] By the term $\tau\boldsymbol{r}$ the authors mean a vector, whose elements are the products $\tau_i r_i$, where i spans across population indices.

2. Analysis of model

2.1 Model description

Consider the system of differential equations:

$$\tau \boldsymbol{r}' = -\boldsymbol{r} + \Phi(gC\boldsymbol{r}), \tag{SC}$$

where $\boldsymbol{r} = (r_1, r_2, r_3)^\top$, with $r_1, r_2, r_3 \geq 0$,

$$C = \left(c_{ij}\right)_{i=1,j=1}^{3,3} = \begin{pmatrix} 2/3 & -5/3 & -1/3 \\ 30 & -3 & -1/2 \\ 18 & -3/2 & 0 \end{pmatrix},$$

$\tau \geq 0$ is a constant and $g \geq 0$ is a parameter. The function φ is as defined above (with $a = b = 1$), and Φ is defined on $\mathbb{R}^3$ by the formula

$$\Phi(x, y, z) = (\varphi(x), \varphi(y), \varphi(z)$$

At this point it is useful to observe that by a change of coordinates, we are able to simplify the system somewhat. The matrix C is regular, and we denote the inverse matrix by $D = (d_{ij})_{i=1,j=1}^{3,3}$. By setting $\boldsymbol{x} = gC\boldsymbol{r}$ for $\boldsymbol{x} = (x_1, x_2, x_3)^\top \in \mathbb{R}^3$ and applying the substitution technique $t = t/g$, the system becomes

$$\tau D\boldsymbol{x}' = -1/gD\boldsymbol{x} + \Phi(\boldsymbol{x}),$$

which in turn can be written as

$$\tau g\boldsymbol{x}' = -\boldsymbol{x} + gC\Phi(\boldsymbol{x}). \tag{2.1}$$

It follows from a simple calculation that $r_1, r_2, r_3 \geq 0$ if and only if

$$d_{i1}x_1 + d_{i2}x_2 + d_{i3}x_3 \geq 0 \quad i = 1, 2, 3. \tag{2.2}$$

Reduced subcircuit – SST population silenced

We are also interested in the reduced version of the system, namely one with $\boldsymbol{r} = (r_1, r_2)^\top$ and the corresponding sub-matrix $\tilde{C} = (c_{ij})_{i=1,j=1}^{2,2}$. This is as if all connections to and from the SST population were severed, as indicated in *Figure 2.1*. Directly analogous to the three-dimensional case, we may set $\boldsymbol{x} = g\tilde{C}\boldsymbol{r}$ and performing the same substitution $t = t/g$ we arrive at the systems

$$\tau \boldsymbol{r}' = -\boldsymbol{r} + \tilde{\Phi}(g\tilde{C}\boldsymbol{r})$$
$$g\tau \boldsymbol{x}' = -\boldsymbol{x} + g\tilde{C}\tilde{\Phi}(\boldsymbol{x}),$$

where we denote $\tilde{\Phi}(x, y) = (\varphi(x), \varphi(y))^\top$ with conditions $r_1, r_2 \geq 0$,

$$-9x_1 + 5x_2 \geq 0 \qquad \text{and} \qquad -45x_1 + x_2 \geq 0. \tag{2.3}$$

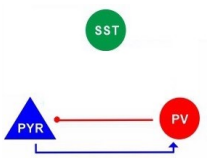


Figure 2.1: Reduced circuit. Modified, from *Hahn et al., 2022* (CC BY)

Remark. In following text, we will drop the notation with tilde (˜) in favor of denoting both connectivity matrices as C and transfer functions as Φ, if it is either clear from context or the reasoning is directly analogous in both versions.

In view of the above remark, we may rewrite the reduced system as

$$\tau \boldsymbol{r}' = -\boldsymbol{r} + \Phi(gC\boldsymbol{r}), \tag{rSC}$$

$$g\tau \boldsymbol{x}' = -\boldsymbol{x} + gC\Phi(\boldsymbol{x}), \tag{2.4}$$

with the conditions on $\boldsymbol{r}$ and $\boldsymbol{x}$ as stated in (2.3).

2.2 Global existence and uniqueness of solutions

In this section, we present and utilise well-known results from the theory of ODE's to prove global existence and uniqueness of the solutions of (SC). Consider the general form of a system of ordinary differential equations

$$\boldsymbol{x}' = f(t, \boldsymbol{x}) \tag{DE}$$

where $\Omega \subset \mathbb{R}^n$ is open and nonempty, $\mathbb{J} \subset \mathbb{R}$ is a nonempty interval and $f : \mathbb{J} \times \Omega \to \mathbb{R}^n$ is given.

Theorem 1 (Vrabie, 2004)**.** *Let $f : \mathbb{J} \times \mathbb{R}^n \to \mathbb{R}^n$ be continuous on $\mathbb{J} \times \mathbb{R}^n$, and let us assume that there exist two continuous functions $h, k : \mathbb{J} \to \mathbb{R}_+$ such that*

$$\|f(t, \boldsymbol{y})\| \leq k(t)\|\boldsymbol{y}\| + h(t),$$

for each $(t, \boldsymbol{y}) \in \mathbb{J} \times \mathbb{R}^n$. Then, for each $(a, \boldsymbol{\xi}) \in \mathbb{J} \times \mathbb{R}^n$ (DE) has at least one global solution.

We apply this theorem to prove the existence of a global solution of (SC) and (rSC). First, we perform some estimates:

$$\frac{1}{1-e^{-x}} = \frac{e^{-x}}{1-e^{-x}} + 1 = \frac{1}{e^x - 1} + 1 \leq \tfrac{1}{x} + 1, \quad \text{for } x > 0,$$

which gives

$$\frac{|x|}{|1-e^{-x}|} \leq |x||\tfrac{1}{x} + 1| \leq 1 + |x|, \quad x \neq 0,$$

$$|\varphi(x)| \leq 1 + |x|, \quad x \in \mathbb{R},$$

since the left and right hand side is defined for any x.

From this we estimate further:

$$\begin{aligned} \|\Phi(x,y,z)\|^2 &\leq (1+|x|)^2 + (1+|y|)^2 + (1+|z|)^2 \leq \\ &\leq (3+2x^2) + (3+2y^2) + (3+2z^2) = \\ &= 9 + 2(x^2+y^2+z^2) = 9 + 2\|(x,y,z)\|^2 \end{aligned}$$

utilising the Young inequality (e.g. $2|x| \leq x^2/2 + 2^2/2 \leq x^2 + 2$).

This leads to:

$$\|\Phi(x,y,z)\| \leq 3 + \sqrt{2}\|(x,y,z)\|,$$

which finally gives the estimate

$$\begin{aligned} \|-\gamma\boldsymbol{x} + C\Phi(\boldsymbol{x})\| &\leq \gamma\|\boldsymbol{x}\| + ||C|| \cdot \|\Phi(\boldsymbol{x})\| \leq \gamma\|\boldsymbol{x}\| + ||C||(3 + \sqrt{2}\|\boldsymbol{x}\|) \leq \\ &\leq (\gamma + \sqrt{2}||C||)\|\boldsymbol{x}\| + 3||C||. \end{aligned}$$

Because the system (2.1) is equivalent to the system (SC), by Theorem 1 we obtain global existence for any solution of the system (SC).

It still needs to be shown that any solution $\boldsymbol{r}(t)$ with the initial condition $(s_1, s_2, s_3) \in \mathbb{R}^3, s_1, s_2, s_3 \geq 0$ stays in this quadrant. Let $r_i = 0$, it then follows for the derivative

$$\tau r_i' = -r_i + \varphi(g(c_{i1}, c_{i2}, c_{i3}) \cdot \boldsymbol{r}) = 0 + \varphi(g(c_{i1}, c_{i2}, c_{i3}) \cdot \boldsymbol{r}) > 0, \quad i = 1, 2, 3$$

in other words, the solution $\boldsymbol{r}$ cannot cross into any other quadrant at any point.

Using the previous estimates, it is easy to show the right hand side of (2.1) is Lipschitz:

$$\begin{aligned} \| -\gamma(\boldsymbol{x} - \boldsymbol{y}) + C(\Phi(\boldsymbol{x}) - \Phi(\boldsymbol{y}))\| &\leq \gamma\|\boldsymbol{x} - \boldsymbol{y}\| + \sqrt{2}||C|| \cdot (\|\boldsymbol{x}\| - \|\boldsymbol{y}\|) \leq \\ &\leq (\gamma + \sqrt{2}||C||)\|\boldsymbol{x} - \boldsymbol{y}\|, \end{aligned}$$

where $\boldsymbol{x}, \boldsymbol{y} \in \mathbb{R}^3$.

This establishes the uniqueness by virtue of the following theorem.

Theorem 2 (Vrabie, 2004)**.** *If $f : \mathbb{J} \times \Omega \to \mathbb{R}^n$ is locally Lipschitz on Ω, then* (DE) *has the uniqueness property.*

The proof of global existence and uniqueness of solutions of the simpler system (rSC) is analogous.

Analysis of the equilibria

Now we provide an argument for the existence and uniqueness of the equilibrium point in the reduced model (rSC) for all positive values of parameter g. It relies largely on the analysis of the asymptotical behavior of functions defined as the right hand sides of (2.4). In subsequent reasoning, a weaker result considering g in a positive neighbourhood of 0 will be presented for the system (SC).

Remark. The differential equation (DE) is called *autonomous* if it is in the form $\boldsymbol{x}' = f(\boldsymbol{x})$. In such case, recall that equilibrium points are such $\boldsymbol{\xi} \in \Omega$, for which $f(\boldsymbol{\xi}) = \boldsymbol{0}$.

At this point it is appropriate to establish that if the equation

$$-\boldsymbol{\rho} + \Phi(gC\,\boldsymbol{\rho}) = \boldsymbol{0}$$

is satisfied for some $\boldsymbol{\rho} \in \mathbb{R}^3 \setminus \{\boldsymbol{0}\}$, then its elements ρ_i are necessarily positive, owing to the positivity of φ, $(i = 1, 2, 3)$.

2.3 Existence and uniqueness of equilibria of the reduced circuit (rSC)

By setting the left hand side to zero, rearranging and applying the inverse matrix $D = C^{-1}$ we obtain the system of equations:

$$\begin{aligned} (d_{11}x_1 + d_{12}x_2) &= g\varphi(x_1) \\ (d_{21}x_1 + d_{22}x_2) &= g\varphi(x_2), \end{aligned} \tag{2.5}$$

where $D = \left(d_{ij}\right)_{i=1,j=1}^{2,2}$ works out to be

$$D = \frac{1}{48}\begin{pmatrix} -3 & 5/3 \\ -30 & 2/3 \end{pmatrix}.$$

Substituting numerical values into (2.5) and some rearranging we obtain:

$$\begin{aligned} 5/3x_2 &= 48g\varphi(x_1) + 3x_1 \\ -30x_1 &= 48g\varphi(x_2) - 2/3x_2. \end{aligned} \tag{2.6}$$

The aim is to prove the following claim:

Claim 3. *The system of equations* (2.6) *has a solution* $(x_1, x_2)^\top$ *satisfying conditions* (2.3).

First, we take advantage of the fact that the right hand sides can be viewed as a function of one variable. After some fraction manipulation we can define functions $X(y)$ and $Y(x)$

$$\begin{aligned} Y(x) &= \tfrac{1}{5}(144g\varphi(x) + 9x), \quad x \in \mathbb{R}, \\ X(y) &= \tfrac{1}{45}(-72g\varphi(y) + y), \quad y \in \mathbb{R}. \end{aligned} \tag{2.7}$$

It is easy to see that Y is strictly increasing by differentiating

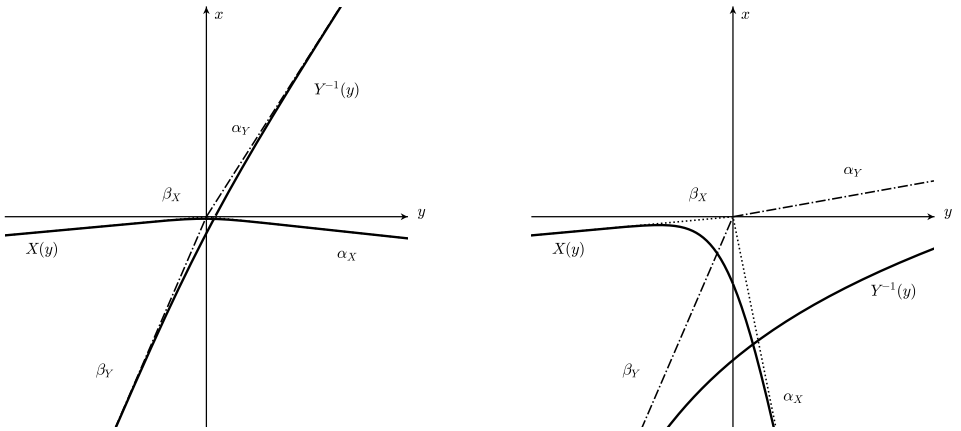
$$Y'(x) = \tfrac{1}{5}(144g\varphi'(x) + 9) > 0, \quad x \in \mathbb{R},$$

since we know that $\varphi' > 0$. This means we can consider the inverse function Y^{-1}.

Next we analyse the asymptotic behavior:

$$\begin{aligned} \lim_{x\to+\infty} \frac{Y(x)}{x} &= \lim_{x\to+\infty} \frac{144g}{5(1-e^{-x})} + \frac{9}{5} = \frac{144g+9}{5} =: \frac{1}{\alpha_Y}, \\ \lim_{x\to-\infty} \frac{Y(x)}{x} &= \lim_{x\to+\infty} \frac{144g}{5(1-e^{x})} + \frac{9}{5} = \frac{9}{5} =: \frac{1}{\beta_Y}, \\ \lim_{y\to+\infty} \frac{X(y)}{y} &= \frac{-72g+1}{45} =: \alpha_X, \\ \lim_{y\to-\infty} \frac{X(y)}{y} &= \frac{1}{45} =: \beta_X. \end{aligned}$$

Slope coefficients α_Y, β_Y are set as the reciprocal of the respective limits because we are interested in the slightly unusual expression of the asymptotes with respect to the second variable (e.g. $x = \alpha_Y y$). This is more suitable since we are expressing X, Y^{-1} as functions of y, see ***Figure 2.2***.



(a) g small (b) g large

Figure 2.2: Graphs of functions (2.7), with asymptote slopes labeled

Notice the asymptotes all intersect in $(0,0)^\top$, $\beta_Y < \beta_X$, and $\alpha_Y > \alpha_X$ for any $g > 0$. From the definiton of limits, this means there are $s < 0 < r$, such that

$$X(s) > Y^{-1}(s), \quad \text{and} \quad X(r) < Y^{-1}(r).$$

Since both functions are continuous, by the *Intermediate Value Theorem* there exists $y_0 \in \mathbb{R}$ such that $X(y_0) = Y^{-1}(y_0)$, which in turn means the point $(Y^{-1}(y_0), y_0)^\top$ is a solution to the system (2.6).

It remains to see that this point is in the region defined by the inequalities (2.3). This holds, as was demonstrated for the original system at the beginning of this section. □

2.3.1 Uniqueness of equilibrium

The proof of the uniqueness of the equilibrium is split into two cases dependent on the value of g.

1. Case of small g: We will show that for small enough g, it holds that $(Y^{-1} - X)' > 0$. Applying the formula for the derivative of the inverse function, we can compute:

   $$(Y^{-1})'(y) = \frac{5}{144g\varphi'(Y^{-1}(y)) + 9} \geq \frac{5}{144g + 9},$$

   using the fact that $1 \geq \varphi' \geq 0$. Similarily it can be shown that $X' \leq 1/45$, and since the following holds for sufficiently small g:

   $$\frac{5}{144g + 9} > \frac{1}{45},$$

   we obtain $(Y^{-1})' > 1/45 \geq X'$ for $g < 3/2$.

2. Case of unbounded g: We will compute the derivative at the intersection of X and the asymptote of Y^{-1} as $y \to -\infty$. Let y_0 denote the horizontal coordinate of the (unique) point of intersection, and it is our aim to find a lower bound on g so the conditions

   $$X(y_0) = \tfrac{5}{9}y_0, \quad \text{and} \quad X'(y_0) < 0, \tag{2.8}$$

are satisfied. It will then follow from the monotonicity of X' that X is injective in the right half-plane bounded by the asymptote, and exploiting the fact that Y is under its asymptotes, we obtain the uniqueness of the equilibrium with some condition on g.

Recall that $\varphi(y_0)$ has y_0 in the numerator, hence by expanding $X(y_0)$ in the first equation in (2.8), we are able to cancel it out and obtain

$$\frac{-72g}{45(1-e^{-y_0})} + \frac{1}{45} = \frac{5}{9}.$$

Employing some straightforward arithmetic and rearranging, we compute:

$$\begin{aligned} -72g &= 24(1-e^{-y_0}) \\ -3g &= 1-e^{-y_0} \\ -y_0 &= \ln(3g+1). \end{aligned}$$

Note that these expressions also establish the uniqueness of the intersection, which is essential.

This we substitute into the formula for φ' :

$$\begin{aligned} g\varphi'(y_0) &= g\frac{1-(3g+1)+\ln(3g+1)(3g+1)}{(-3g)^2} \\ &= \frac{-3g+\ln(3g+1)(3g+1)}{9g} = \tfrac{1}{3}(-1+\ln(3g+1)\frac{3g+1}{3g}) \\ &= \tfrac{1}{3}(h(3g)-1). \end{aligned}$$

The function $h(x) \coloneqq \ln(x+1)\frac{x+1}{x}$ is monotone, concave and continuously extendable to $x=0$ by 1 – facts demonstrated in the Appendix (ii). As a result, we can implement an estimate of its secant line between points $x=0$ and $x=e-1$

$$h(x) = \ln(x+1)\frac{x+1}{x} \geq \ (\ln(e)\frac{e}{e-1}-1)\frac{x}{e-1}+1 = \frac{x}{(e-1)^2}+1.$$

Now, we may perform the estimate for $X'(y_0)$:

$$\begin{aligned} &\tfrac{1}{45}(-72g\varphi'(y_0)+1) < 0 \\ &-24(h(3g)-1)+1 < 0 \quad \Leftrightarrow \quad h(3g) > 1+\frac{1}{24} \end{aligned}$$

hence it is sufficient that

$$\frac{3g}{(e-1)^2} > \frac{1}{24} \quad \Leftrightarrow \quad 3g > \frac{(e-1)^2}{24}$$

since the right hand value still clearly lies in the interval $(0, e-1)$.

Since the sufficient conditions for uniqueness presented in 1. and 2. jointly cover all positive values of g, we have proved the uniqueness claim:

Claim 4. *The system of differential equations* (2.4) *has a unique equilibrium point for every positive g satisfying conditions* (2.3).

□

2.4 Existence and uniqueness of equilibria in the full subcircuit

Since the reasoning in the case of the reduced subcircuit exploits the expression of the equation as functions of one variable, it does not scale naturally with the added dimension. The strategy presented here is to make use of the *Implicit Function Theorem* to prove existence and uniqueness of the equlibrium at least locally for small g. This is not meaningless, since small values of g are suggested to be more biologically significant in mouse V1 cortex *(Hahn et al., 2022)* .

The following well-known theorem is taken from *Rudin, 1976*, modified with present notation.

Theorem 5 (Implicit Function Theorem)**.** *Let $F = F(\boldsymbol{x}, \boldsymbol{y})$ be a $\mathcal{C}^k$-mapping of an open set $E \subset \mathbb{R}^{n+m}$ into $\mathbb{R}^n$, such that $F(\boldsymbol{a}, \boldsymbol{b}) = \mathbf{0}$ for some point $(\boldsymbol{a}, \boldsymbol{b}) \in E$.*

Put $A_x = \mathcal{D}_x F(\boldsymbol{a}, \boldsymbol{b})$ and assume that $\det A_x \neq 0$. Similarly denote by $A_y = \mathcal{D}_y F(\boldsymbol{a}, \boldsymbol{b})$.

Then there exist open sets $U \subset \mathbb{R}^{n+m}$ and $W \subset \mathbb{R}^m$, with $(\boldsymbol{a}, \boldsymbol{b}) \in U$, and $\boldsymbol{b} \in W$, having the following property:

To every $\boldsymbol{y} \in W$ corresponds a unique $\boldsymbol{x}$, such that

$$(\boldsymbol{x}, \boldsymbol{y}) \in U, \qquad \text{and} \qquad F(\boldsymbol{x}, \boldsymbol{y}) = \mathbf{0}.$$

If this $\boldsymbol{x}$ is defined to be $\psi(\boldsymbol{y})$, then ψ is a $\mathcal{C}^k$-mapping of W into $\mathbb{R}^n$, $\psi(\boldsymbol{b}) = \boldsymbol{a}$,

$$F(\psi(\boldsymbol{y}), \boldsymbol{y}) = \mathbf{0} \quad (\boldsymbol{y} \in W), \quad \text{and} \qquad \mathcal{D}\psi(\boldsymbol{b}) = -\Big(A_x\Big)^{-1} A_y.$$

In accordance with the above theorem, denote by $F(\boldsymbol{x}, g)$ the right hand side of the system (2.1), namely

$$F(\boldsymbol{x}, g) = -\boldsymbol{x} + gC\Phi(\boldsymbol{x}).$$

This corresponds to a specific value of the parameter $g \geq 0$, (which plays the role of $\boldsymbol{y}$ in the theorem).

It is easy to see that $F(\mathbf{0}, 0) = \mathbf{0}$. We compute the derivative of F with respect to the first three variables, and evaluate the Jacobian

$$\mathcal{D}_x F(\boldsymbol{x}, g) = -\mathbb{1} + gC\Big(\mathcal{D}\Phi(\boldsymbol{x})\Big) = -\mathbb{1} + gC \begin{pmatrix} \varphi'(x) & 0 & 0 \\ 0 & \varphi'(y) & 0 \\ 0 & 0 & \varphi'(z) \end{pmatrix}, \tag{2.9}$$

which implies

$$\det \mathcal{D}_x F(\mathbf{0}, 0) = -\det \mathbb{1} \neq 0.$$

This guarantees the existence of a unique point $\boldsymbol{\xi} = \boldsymbol{\xi}(g)$ for any g in a neighbourhood of $g = 0$, which is more than enough, as we are interested only in positive values of the parameter.

Next, recall $\varphi(0) = 1$, and we calculate

$$\boldsymbol{x}_0'(0) = -\Big(-\mathbb{1}\Big)^{-1} \mathcal{D}_g F(\mathbf{0}, 0) = C\Phi(\mathbf{0}) = \begin{pmatrix} \sum c_{1j} \\ \sum c_{2j} \\ \sum c_{3j} \end{pmatrix} = \begin{pmatrix} -4/3 \\ 53/2 \\ 33/2 \end{pmatrix} =: \mathbf{y},$$

where $\sum$ is shorthand for $\sum_{j=1}^{3}$.

Since it has already been reasoned that potential equilibrium points must lie in the desired region, we proved the following:

Claim 6. *There exists an $\varepsilon > 0$ such that the system of differential equations* (SC) *has a unique equilibrium point $\boldsymbol{\xi}_g \in \mathbb{R}^3$ for any value of the parameter $g \in (0, \varepsilon)$.*

The position of this equilibrium point may be estimated as

$$\boldsymbol{\xi}_g = g\boldsymbol{x}_0'(0) + \boldsymbol{o}(g) = g\mathbf{y} + \boldsymbol{o}(g)$$

□

2.5 Analysis of stability

We may now turn our attention to analysing the stability of the dynamical systems at equilibria using linearization techniques. First, recall the relevant types of stability of the system (DE), now considered with the essential added constraint on f being Lipschitz with respect to the variable $\boldsymbol{x}$.

Definition 1 (Vrabie, 2004)**.** The null solution of (DE) is *stable* if:

(i) for every $a \geq 0$ there exists $\mu(a) > 0$ such that, for every $\boldsymbol{\xi} \in \Omega$ with $\| \boldsymbol{\xi} \| \leq \mu(a)$, the unique saturated solution $\boldsymbol{x}(\cdot, a, \boldsymbol{\xi})$ of the system (DE), satisfying $\boldsymbol{x}(a, a, \boldsymbol{\xi}) = \boldsymbol{\xi}$, is defined on $[a, +\infty)$, and

(ii) for every $a \geq 0$ and every $\varepsilon > 0$ there exists $\delta(\varepsilon, a) \in (0, \mu(a)]$ such that, for each $\boldsymbol{\xi} \in \Omega$ with $\| \boldsymbol{\xi} \| \leq \delta(\epsilon, a)$, the unique saturated solution $\boldsymbol{x}(\cdot, a, \boldsymbol{\xi})$ of the system (DE), satisfying $\boldsymbol{x}(a, a, \boldsymbol{\xi}) = \boldsymbol{x}(\cdot, a, \boldsymbol{\xi})$ also satisfies $\|\boldsymbol{x}(t, a, \boldsymbol{\xi})\| \leq \varepsilon$ for every $t \in [a, +\infty)$.

Definition 2 (Vrabie, 2004)**.** The null solution of (DE) is *asymptotically stable* if it is stable and, for every $a > 0$ and $\mu(a) > 0$ in definition 1 can be chosen such that, for each $\boldsymbol{\xi} \in \Omega$ with $\| \boldsymbol{\xi} \| \leq \mu(a)$, the unique saturated solution $\boldsymbol{x}(\cdot, a, \boldsymbol{\xi})$ of the system (DE), which satisfies $\boldsymbol{x}(a, a, \boldsymbol{\xi}) = \boldsymbol{\xi}$, also satisfies $\lim_{t \to +\infty} \|\boldsymbol{x}(t, a, \boldsymbol{\xi})\| = 0$.

We remark that a saturated solution of an (DE) is such that cannot be extended to a larger domain.

2.5.1 Stability of the subcircuits

Remark. Even though the systems we are considering (2.1) and (2.4) both have the factor $g\tau > 0$ on the left hand side, this only scales the spectrum of their respective linearization matrix, hence we can consider the further simplified equations

$$\boldsymbol{x}' = -\boldsymbol{x} + gC\Phi(\boldsymbol{x}). \tag{sSC}$$

Furthermore, the following reasoning is parallel in both the full and reduced circuit analysis, which we will exploit later.

Put $F(\boldsymbol{x})$ the right hand side of (sSC). Owing to (2.9), we have already once computed the Jacobian matrix $\mathcal{D}F$.

Referencing the estimate from Taylor expansion of φ' established in the appendix (2.11), we derive the matrix version for $\mathcal{D}\Phi$:

$$\begin{aligned}\mathcal{D}\Phi(\boldsymbol{x}) &= \begin{pmatrix}\varphi'(x) & 0 & 0\\ 0 & \varphi'(y) & 0\\ 0 & 0 & \varphi'(z)\end{pmatrix} = \begin{pmatrix}\frac{1}{2}+\frac{x}{6}+o(x) & 0 & 0\\ 0 & \frac{1}{2}+\frac{y}{6}+o(y) & 0\\ 0 & 0 & \frac{1}{2}+\frac{z}{6}+o(z)\end{pmatrix}\\ &= \tfrac{1}{2}\,\mathbb{1} + \tfrac{1}{6}\operatorname{diag}(x,y,z) + \operatorname{diag}(o(x),o(y),o(z))\\ &= \tfrac{1}{2}\,\mathbb{1} + \tfrac{1}{6}\operatorname{diag}(\boldsymbol{x}) + \mathcal{O}(\boldsymbol{x}),\end{aligned}$$

where $\operatorname{diag}(x,\dots)$ denotes the diagonal matrix with elements in their respective order, and $\mathcal{O}(\boldsymbol{x})$ is the generic error matrix.

Combining this with the estimate on the location of the equilibirum point formulated in Claim 6, we obtain for the full subcircuit:

$$\begin{aligned}\mathcal{D}F(\boldsymbol{\xi}_g) &= -\mathbb{1} + gC\Big(\mathcal{D}\Phi(\boldsymbol{\xi}_g)\Big) = -\mathbb{1} + gC\Big(\tfrac{1}{2}\,\mathbb{1} + \tfrac{1}{6}\operatorname{diag}(\boldsymbol{\xi}_g) + \mathcal{O}(\boldsymbol{\xi}_g)\Big)\\ &= -\mathbb{1} + gC(\tfrac{1}{2}\,\mathbb{1} + \tfrac{1}{6}\operatorname{diag}(g\mathbf{y}+\boldsymbol{o}(g)) + \mathcal{O}(g\mathbf{y}+\boldsymbol{o}(g))\Big)\\ &= -\mathbb{1} + gC(\tfrac{1}{2}\,\mathbb{1} + \tfrac{1}{6}\operatorname{diag}(g\mathbf{y})\Big) + g\mathcal{O}(g\mathbf{y})\\ &= -\mathbb{1} + \tfrac{1}{2}gC + \tfrac{1}{6}g^2C\operatorname{diag}(\mathbf{y}) + \mathcal{O}(g^2),\end{aligned}$$

where $\mathcal{O}(g) = \mathcal{O}(g,\dots)$. Evaluating the matrix multiplication of the third term is straightforward and yields

$$\tfrac{1}{6}C\operatorname{diag}(\mathbf{y}) = \begin{pmatrix}-4/21 & -265/36 & -11/12\\ -20/3 & -53/4 & -11/8\\ -4 & -53/8 & 0\end{pmatrix} =: B,$$

hence overall we have arrived at the formula

$$\mathcal{D}F(\boldsymbol{\xi}_g) = -\mathbb{1} + \tfrac{1}{2}gC + g^2B + \mathcal{O}(g^2). \tag{2.10}$$

Due to the definition of $\mathbf{y}$, the corresponding matrix B will not simply be the top right submatrix in the case of the reduced circuit, nevertheless, the derivation of the analogous formula is clearly possible. It should be also noted that the expression (2.10) and evaluation of the matrix B is not necessary for the following argument.

The following theorem is applied to obtain the stability result, leveraging the subsequent simple lemma.

Definition 3 (Vrabie, 2004)**.** The matrix A is *hurwitzian* if all the roots of the characteristic equation $\det(A-\lambda\,\mathbb{1}) = 0$ have strictly negative real parts.

Theorem 7 (Vrabie, 2004)**.** *Let Ω be a neighbourhood of $\mathbf{0}\in\mathbb{R}^n$. If $f:\Omega\to\mathbb{R}^n$ is a function of class $\mathcal{C}^1$ with $f(\mathbf{0})=\mathbf{0}$ and whose Jacobian matrix $A=\mathcal{D}f(0)$ is hurwitzian, then the null solution of the system* (DE) *is asymptotically stable.*

Lemma 8. *Let A be a matrix and $\sigma(A)$ its spectrum. It then holds that*

$$\sigma(A-\mathbb{1}) = \sigma(A) - 1$$

Proof. Derivation is straightforward from the definition of spectrum:

$$A-\lambda\,\mathbb{1} = A \pm \mathbb{1} - \lambda\,\mathbb{1} = (A-\mathbb{1}) - (\lambda-1)\,\mathbb{1},$$

hence $\lambda\in\sigma(A)$, if and only if $(\lambda-1)\in\sigma(A-\mathbb{1})$. □

We are now in the position to prove the stability, as is formulated in the claim:

Claim 9. *There exists $\varepsilon > 0$ such that the systems* (sSC) *have unique asymptotically stable equilibria for $g \in (0, \varepsilon)$.*

Proof. The existence and uniqueness of the equilibria for all $g \in (0, \varepsilon')$, with $\varepsilon' > 0$, has already been established. From (2.10) and the lemma above we obtain the formula

$$\sigma\Big(\mathcal{D}F(\boldsymbol{\xi}_g)\Big) = \sigma(\tfrac{1}{2}gC + \mathcal{O}(g)) - 1,$$

and recalling the definition of $\mathcal{O}$, jointly with

$$g||C|| \geq \max\{|\lambda|; \lambda \in \sigma(gC)\},$$

yields the existence of $M > 0$, such that

$$\mathrm{Re}\lambda < -1 + Mg.$$

Hence there exists some $\varepsilon \in (0, \varepsilon')$, such that the spectrum of the Jacobian matrix $\mathcal{D}F(\boldsymbol{\xi}_g)$ has strictly negative real parts for $g \in (0, \varepsilon)$. □

As was argued, the above line of reasoning will work identically for the reduced system, hence we can likewise expect stability for small g.

Conclusion

We proved asymptotic stability of equilibria in the systems (SC) and (rSC) for small values of g. A less limited stability result would likely demand an estimate on the location of equilibria as well as a more detailed analysis of the function φ.

The mathematical treatment presented is consistent with the literature *(Ostojic, 2014; Hahn et al., 2022)*, where stability for small synaptic couplings is observed. However, the result is likely too weak to be of biological significance, as there is no estimate provided for the size of the neighbourhood where stability is guaranteed.

Appendix

This chapter establishes several simple, but useful facts.

(i) Properties of φ:
We define the continuous function $\varphi : \mathbb{R} \to \mathbb{R}$ as

$$\varphi(x) = \begin{cases} \dfrac{x}{1-e^{-x}}, & x \in \mathbb{R} \setminus \{0\} \\ 1, & x = 0. \end{cases}$$

It is clear that φ is nonnegative for $x \geq 0$, and both numerator and denominator change sign for $x < 0$, hence φ is nonnegative for all $x \in \mathbb{R}$.

The estimate $\varphi(x) \geq x, \;\; x \in \mathbb{R}$ is obtained by the computation:

$$\frac{x}{1-e^{-x}} - x = \frac{xe^{-x}}{1-e^{-x}} = \varphi(x)e^{-x} \geq 0, \quad x \neq 0.$$

and since $\varphi(0) = 1$, the result is valid for all $x \in \mathbb{R}$.

We show that φ is strictly increasing by differentiating

$$\varphi'(x) = \frac{1-e^{-x}-xe^{-x}}{(1-e^{-x})^2} = \frac{1-(1+x)e^{-x}}{(1-e^{-x})^2}, \quad x \neq 0.$$

Utilising the estimate $e^x > 1 + x$, $x \in \mathbb{R} \setminus \{0\}$, we show for the numerator of the fraction

$$1-(1+x)e^{-x} > 1 - e^x e^{-x} = 0.$$

Furthermore, it holds that $\varphi'(0) = \lim_{x\to 0} \varphi'(x) = 1/2$, hence we obtain $\varphi'(x) > 0$ for all $x \in \mathbb{R}$.

Next, we show $\varphi' \leq 1$:

$$(1-e^{-x})^2 = 1 - 2e^{-x} + e^{-2x} = 1 - e^{-x} + (e^{-x} - 1)e^{-x} \geq 1 - e^{-x} - xe^{-x},$$

and finally, differentiating a second time we compute

$$\begin{aligned} \varphi''(x) &= \frac{xe^{-x}}{(1-e^{-x})^2} + \frac{2xe^{-2x}}{(1-e^{-x})^3} - \frac{2e^{-x}}{(1-e^{-x})^2} \\ &= \frac{x(1-e^{-x})e^{-x}}{(1-e^{-x})^3} + \frac{2xe^{-2x}}{(1-e^{-x})^3} - \frac{2(1-e^{-x})e^{-x}}{(1-e^{-x})^3} \\ &= \frac{xe^{-x} + xe^{-2x} - 2(1-e^x)e^{-x}}{(1-e^{-x})^3} \end{aligned}$$

and evaluate the limit by expanding $e^{-x} = 1 - x + \frac{x^2}{2} + o(x^2)$, as follows

$$\lim_{x\to 0} \varphi''(x) = \frac{2x - 3x^2 + \frac{x^3}{2} + 2x^3 - 2x + 3x^2 - 2x^3 - \frac{x^3}{6} + o(x^3)}{x^3 + o(x^3)} = 1/6.$$

Now, we may estimate φ' via Taylor expansion at zero:

$$\varphi'(\xi) = \varphi'(0) + \varphi''(0)\xi + o(\xi) = \tfrac{1}{2} + \tfrac{\xi}{6} + o(\xi). \tag{2.11}$$

(ii) Properties of $h(x)$:

The function is continuously extendable to 0 by its limit:

$$\lim_{x \searrow 0} \ln(x+1)\frac{x+1}{x} = 1$$

Straightforward differentiation shows monotonicity:

$$\frac{1}{x+1} \cdot \frac{x+1}{x} + \ln(x+1)\frac{x-x-1}{x^2} = \frac{x - \ln(x+1)}{x^2} > 0, \text{ for } x > 0.$$

After differentiating again we assert concavity, likewise for $x > 0$:

$$-\frac{1}{x^2} - \frac{1}{(x+1)x^2} + 2\frac{\ln(x+1)}{x^3} \overset{?}{<} 0$$

$$x + \frac{x}{x+1} - 2\ln(x+1) \overset{?}{>} 0. \tag{2.12}$$

This holds, as verified by the computation

$$(x + \frac{x}{x+1})' = 1 + \frac{1}{x+1} > \frac{2}{x+1} = (2\ln(x+1))',$$

combined with the fact that (2.12) is equal to zero for $x = 0$.

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