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Kenneth P.Dietrich School of Arts & Sciences; Department of Mathematics

Bistability analysis of excitatory, inhibitory and somatostatin neuron network

Arnab Dey Sarkar, Bard Ermentrout

March, 2025

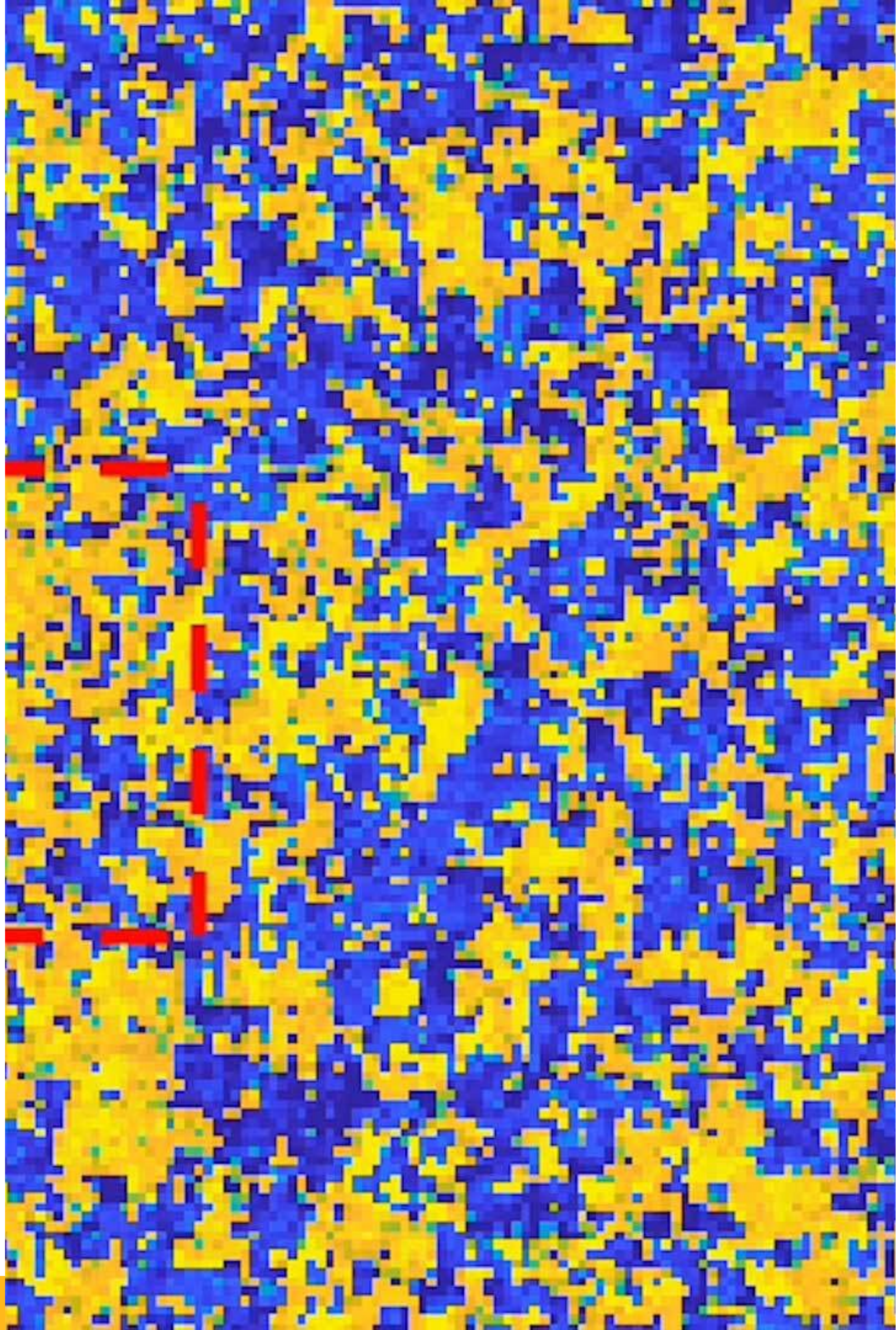


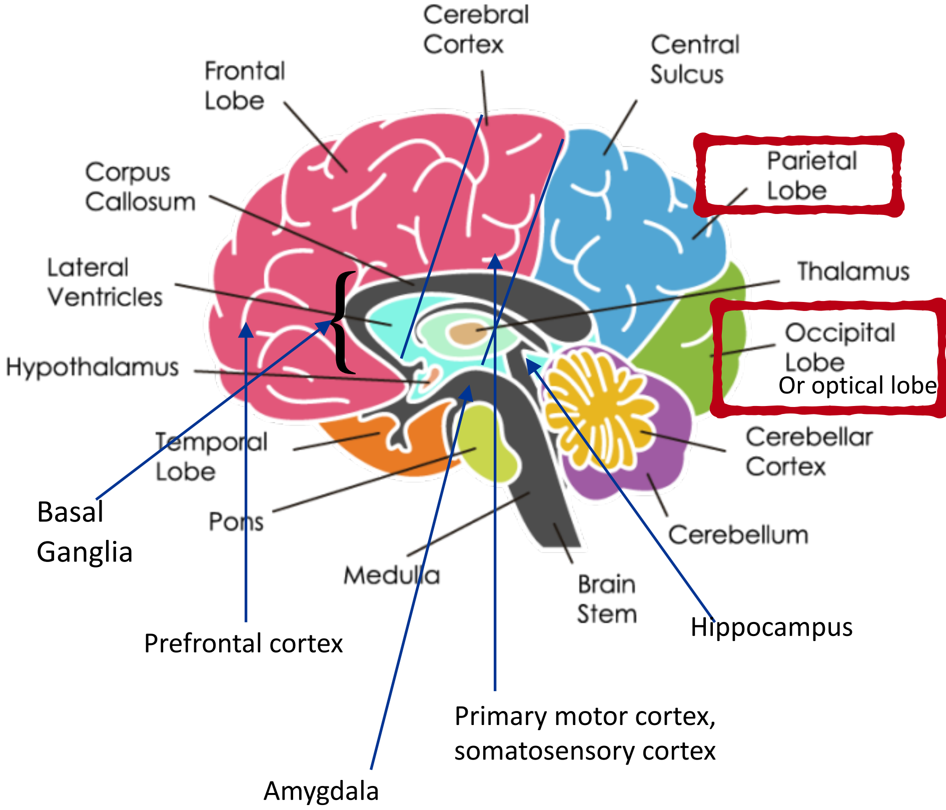
Table of contents

Cells in the Brain

- Neurons: Primary cells responsible for transmitting signals.
- Pyramidal Cells: Excitatory cells;
- Interneurons: Inhibitory cells:
  - FS (Fast Spiking)(we denote it as ‘I’ in our model) Parvalbumin
  - LTS (Low Threshold Spiking)(we denote it as ‘s’ in our model) Somatostatin

Major Brain Regions

- Prefrontal Cortex: Executive functions, decision-making.
- Hypothalamus: Regulates homeostasis, hunger, thirst, and emotions.
- Pituitary Gland: Hormone secretion.
- Amygdala: Emotion processing, particularly fear.
- Hippocampus: Memory formation.
- Cerebellum: Coordination of voluntary movements.
- Motor Cortex: Controls voluntary movement.
- Thalamus: Relay station for sensory and motor signals.
- Optical Lobe: Visual processing.
- Basal Ganglia
- Neocortex: the outermost layer of the brain



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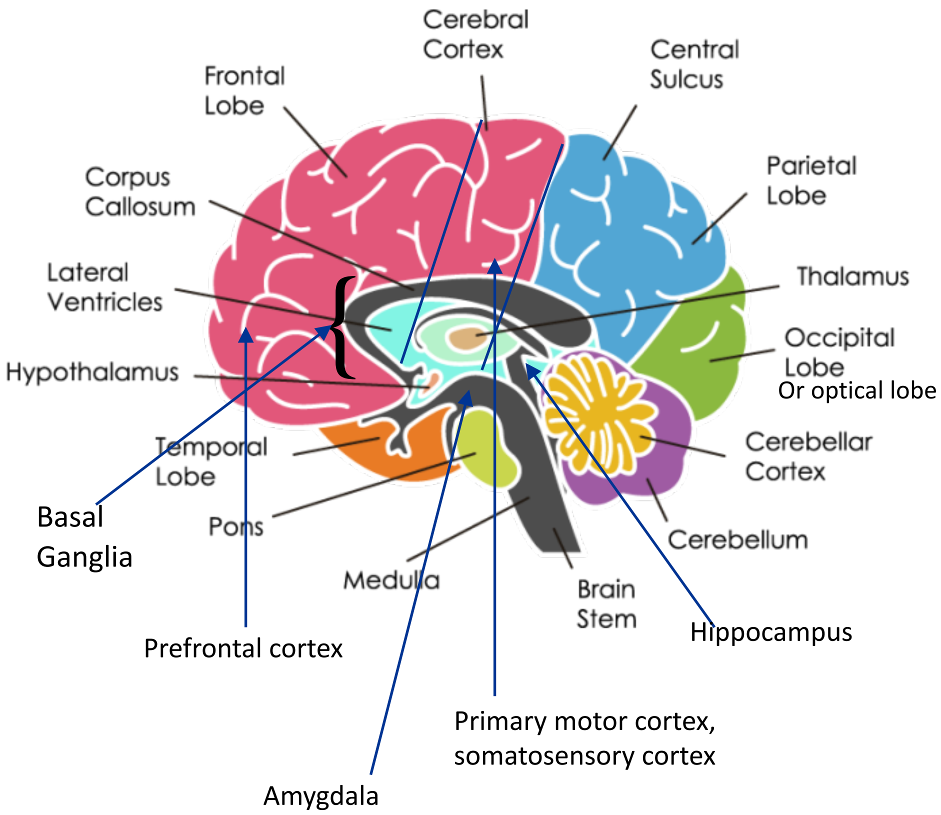

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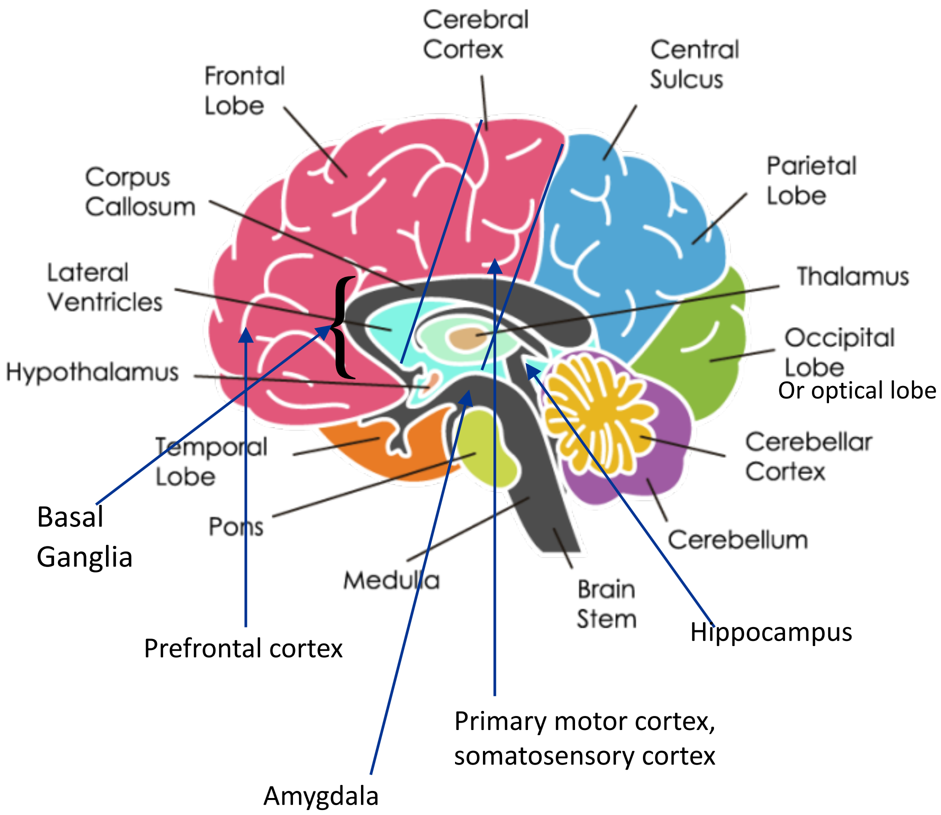

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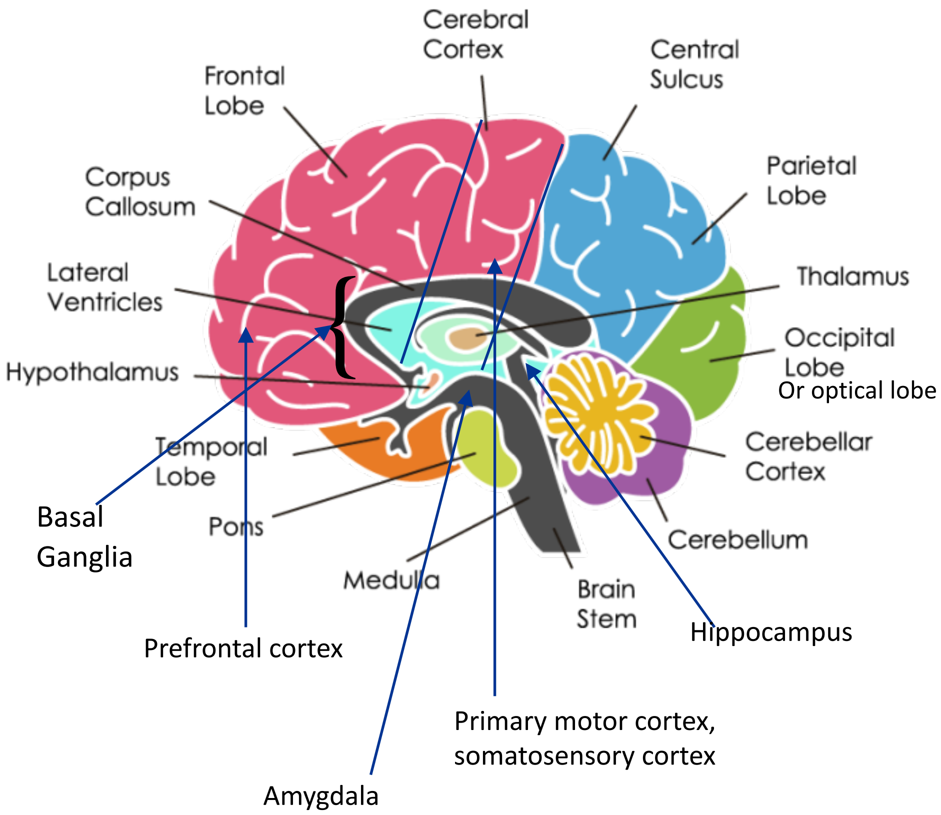

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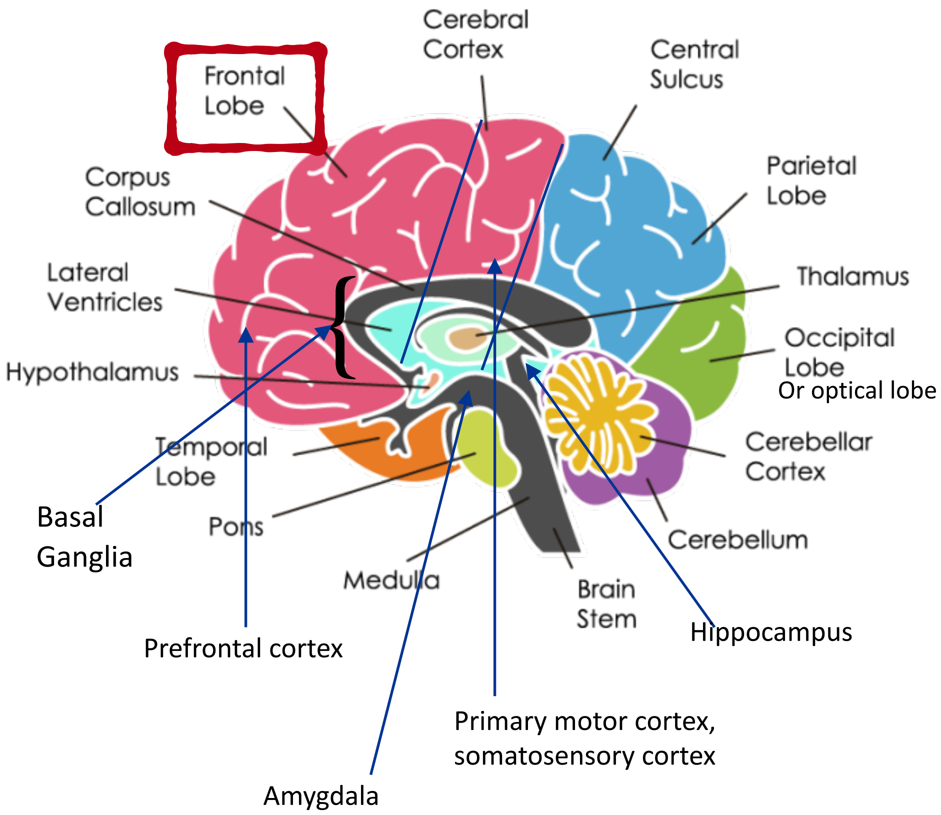

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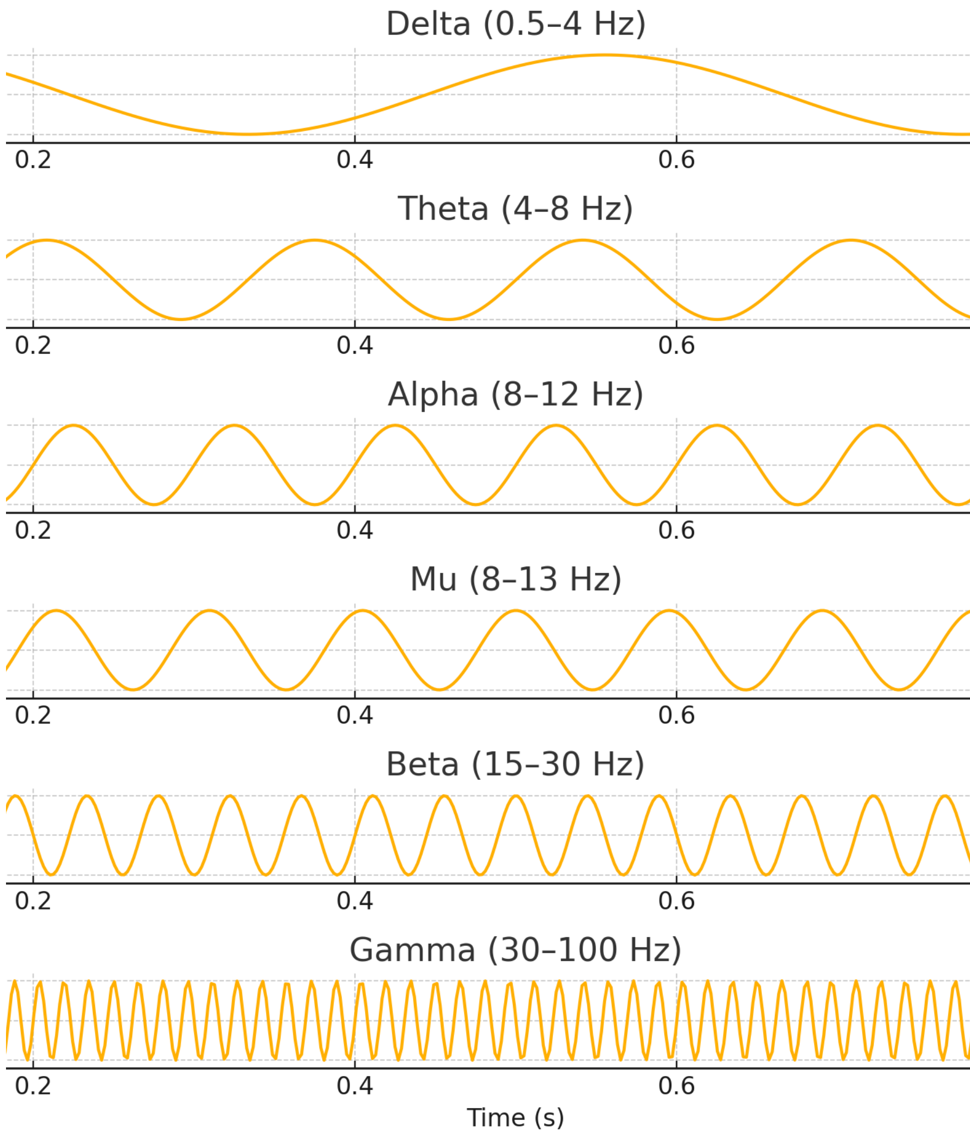
Brain Rhythms

- Delta (0.5–4 Hz): Deep sleep, memory consolidation.
- Theta (4–8 Hz): Navigation, memory encoding and retrieval.
- Alpha (8–12 Hz): Relaxation, calm wakefulness.
- Mu (8–13 Hz): Sensorimotor rhythm.
- Beta (15–30 Hz): Active thinking, attention, motor planning.
- Gamma (30–100 Hz): High-level cognitive processing, perception, attention.



Takeaway

Rhythms are sequentially activated during different brain states.

Some previous research

Guang Chen,[1,2] Yuan Zhang,[1]

SOM and PV neurons work together to control brain rhythms, but they specialize

Ermentrout and Kopell

Simple Explanation of the E-I Model for Gamma Oscillations

Anita K. Roopun, Mark A. Kramer, Lucy M. Carracedo, Marcus

1. The **neocortex** produces brain rhythms across a wide range of frequencies, each linked to different cognitive and motor functions.
2. When two rhythms differ greatly in

Our work: Part 1

A 3-population model of Excitation, Inhibition, and Somatostatin neurons can produce Beta1 and Beta2 rhythms.

Our work: Part 2

If we mix some portions of the population to start with beta1 and some with beta2. We will see interesting oscillations.

Some previous research in this area

Guang Chen,[1,2] Yuan Zhang,[1] Xiang Li,[1] Xiaochen Zhao,[1] Qian Ye,[1] Yingxi Lin,[3] Huizhong W. Tao,[4] Malte J. Rasch,[1], 2017(Distinct Inhibitory Circuits Orchestrate Cortical beta and gamma Band Oscillations)

SOM and PV neurons work together to control brain rhythms, but they specialize in different frequency ranges:

- SOM cells regulate lower-frequency beta waves (important for long-range communication and sensory processing).

- PV cells regulate higher-frequency gamma waves (important for fast information processing and attention).

Takeaway

Rhythms are sequentially

Ermentrout and Kopell

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Some previous research in this area

Ermentrout and Kopell, collection of papers around 2000s

Simple Explanation of the E-I Model for Gamma Oscillations

The **Excitatory-Inhibitory (E-I) Model** is one of the earliest models used to explain **gamma oscillations** (brain rhythms around 30–80 Hz). It describes how **excitatory pyramidal neurons (E)** and **inhibitory interneurons (I)** interact to create rhythmic activity in the brain. They have also introduced the **Theta model**, we will see later.

How the E-I Model Works:

1. **Excitatory neurons fire first**, activating inhibitory neurons.
2. **Inhibitory neurons then fire**, suppressing excitatory neurons.
3. **This cycle repeats**, creating an oscillation where excitation and inhibition alternate.

Takeaway

Rhythms are sequentially

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Some previous research in this area

Anita K. Roopun[1,†], Mark A. Kramer[2,†], Lucy M. Carracedo[1], Marcus Kaiser[1,3], Ceri H. Davies[4], Roger D. Traub[5], Nancy J. Kopell[2,*] and Miles A. Whittington[1,*] 2008(Period concatenation underlies interactions between gamma and beta rhythms in neocortex)

1. The **neocortex** produces brain rhythms across a wide range of frequencies, each linked to different cognitive and motor functions.

2. When two rhythms differ greatly in frequency, they interact through **phase synchronization** or **amplitude modulation (nesting)**.

3. This study shows that **gamma (40 Hz) and beta2 (25 Hz) rhythms** in different cortical layers can combine to generate a **third rhythm, beta1 (15 Hz)**.

Interaction of Brain Rhythms

Our work: Part 1

A 3-population model of Excitation, PV, and Somatostatin neurons can produce two distinct beta rhythms(bi-rhythmicity).

Takeaway

Rhythms are sequentially

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Our work: Part 2

If we mix some portions of the population to start with beta1 and some with beta2. We will see interesting oscillations.

Interaction of Brain Rhythms

Our work: Part 2

If we mix some portions of the population to start with one beta and some with the other beta. We will see interesting oscillations.

Takeaway

Rhythms are sequentially

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Guang Chen,[1,2] Yuan Zhang,[1]

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Part 1

Generation of bi-rhythmicity using 3 population network and 9 dimensional model.

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Introduction: Context

We have seen a lot of studies of E + I neuron networks, spiking models, γ oscillatory neurons etc.

Recent studies indicate the importance of the other populations of inhibitory interneurons.

The E + I network is specifically Pyramidal cells (E) and Parvalbumin(I) cells, notably, somatostatin neurons(S). They have different temporal properties and different connectivities. The goal of this project is to explore the dynamics when there are 3 populations of cells.

Introduction: Quadratic Integrate and fire neuron(QIF)

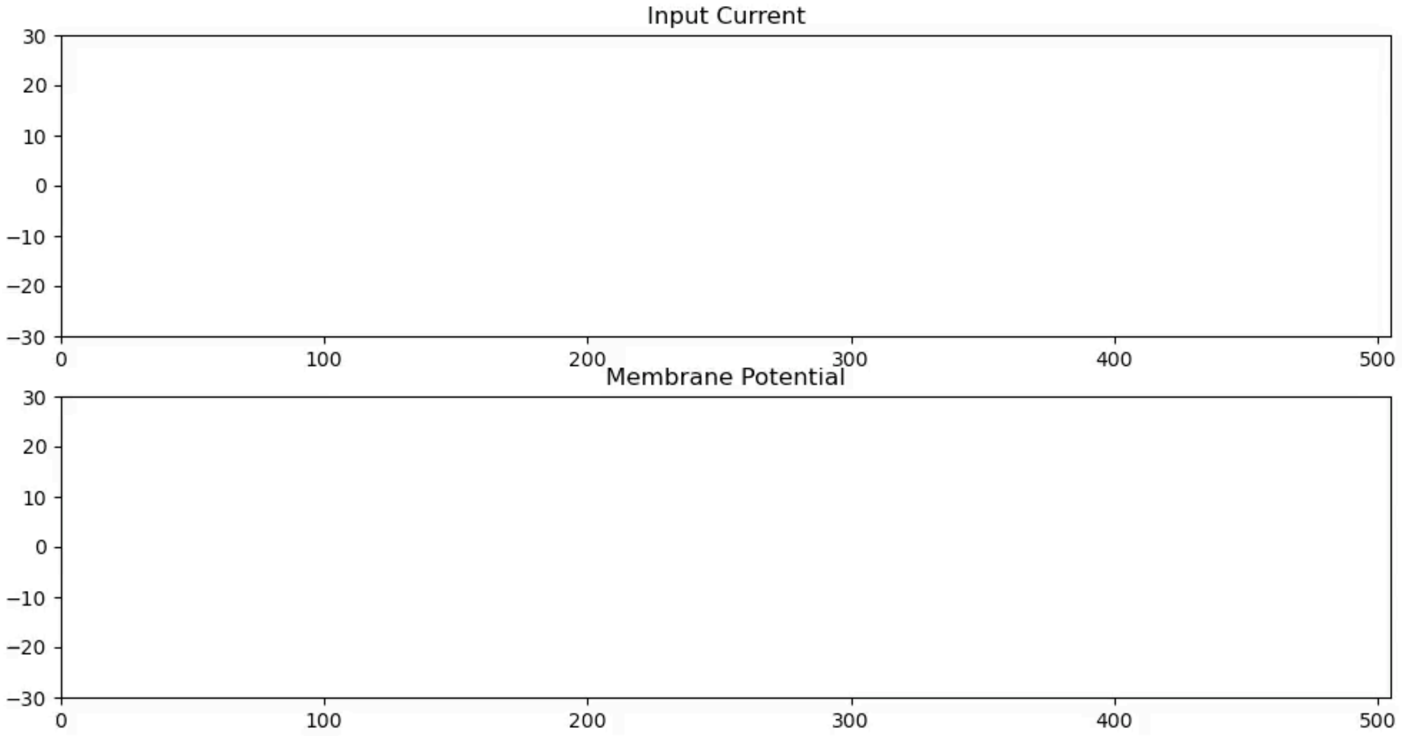
In an Integrate and Fire(IF) model

$$\tau \frac{du}{dt} = F(u)$$

If we have F(u) a quadratic equation then the model will be called Quadratic Integrate and fire neuron; QIF. In the RHS video I have used

$$F(u) = u^2 + I$$

The input current is 30 and 50 at time 50 and 300.



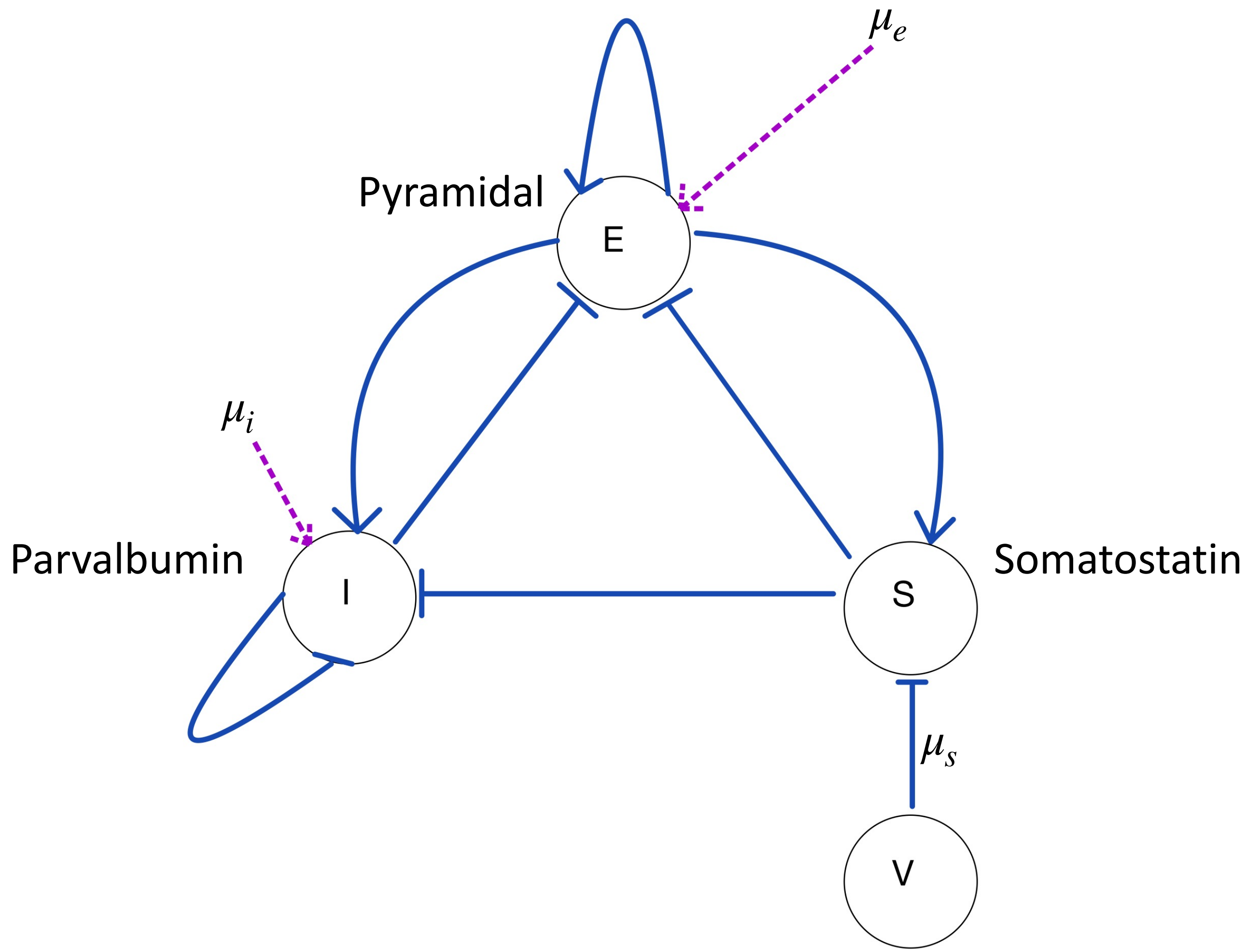
Theta model

Then if we put $u = \tan(\frac{\theta}{2})$ then our equation $F(u) = u^2 + I$ it will become

$$\tau \frac{d\theta}{dt} = (1 - \cos\theta) + I(1 + \cos\theta)$$

This is a Theta model.

Corresponding Network



Each population has N neurons connected
In all to all manner except the VIP which is the
Input to S population via μ_s.

$$\mu_s = \mu_{s_0} - \mu_{s_{\text{VIP}}}$$

$$\mu_{s_{\text{VIP}}} > 0$$

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Infinite dimensional to 3D model

For simplicity we consider one population for N globally coupled quadratic integrate and fire neurons (QIF):

$$\boxed{\tau_m \frac{dV_j}{dt} = V_j^2 + \mu(t) + \Delta\xi_j + gS}$$

where μ is global (to every neuron) drive, Δ is a heterogeneity strength, ξ_j is taken from the Cauchy distribution, $q(\xi)$ centered at 0 with half width 1, but fixed in time, and S satisfies:

$$\boxed{\tau_s \frac{dS}{dt} = -S + \boxed{\frac{1}{N}\sum_{j=1}^{N}\sum_{k}\delta\left(t - t_j^k\right)\tau_m\,.}}$$

Infinite dimensional to 3D model

Hence from QIF model

$$\tau_m \frac{dV_j}{dt} = V_j^2 + \mu(t) + \Delta\xi_j + gS,\ j = 1(1)N.$$

Now if we take $N \to \infty$,

Infinite dimensional to 3D model

we get

$$\tau_m a_t = 2ab + \Delta,$$
$$\tau_m b_t = b^2 - a^2 + \mu + gS$$
$$\tau_s S_t = -S + \frac{a}{\pi}$$

Where, $\frac{a}{\pi} =$ mean firing rate

$b =$ population mean voltage

$S =$ synapse

Ref. Montbrió, Ernest, Diego Pazó, and Alex Roxin. "Macroscopic description for networks of spiking neurons." Physical Review X 5.2 (2015): 021028.

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Some observations using mathematical analysis for bifurcation

Theorem: Let $(\bar{a}, \bar{b}, \widehat{S})$ be a fixed point. Then we have some observations

- If $\tau_s \to \infty$ then we won't have any limit cycle
- If $\tau_s \to 0$ then all possible equilibrium points are stable or saddle. Hence we can't have a Hopf bifurcation. Using Dulac's criterion, we can't have a limit cycle. Moreover if $g < 0$ we have stable equilibrium point only.

Simulation(Full 3 population model)

$$a_l' = \frac{1}{\tau_{ml}}(2a_l b_l + \delta_l)$$

$$b_l' = \frac{1}{\tau_{ml}}(b_l^2 - a_l^2 + g_{el}s_e - \gamma g_{il}s_i - \lambda g_{sl}s_s + \mu_l)\text{where } k, l \in \{e, i, s\}$$

$$s_l' = \frac{1}{\tau_l}\left(-s_l + \frac{a_l}{\pi}\right)$$

Simulation(Full 3 population model)

$$a_l' = \frac{1}{\tau_{ml}}(2a_l b_l + \delta_l)$$

$$b_l' = \frac{1}{\tau_{ml}}(b_l^2 - a_l^2 + g_{el}s_e - \gamma g_{il}s_i - \lambda g_{sl}s_s + \mu_l) \text{where } k, l \in \{e, i, s\}$$

$$s_l' = \frac{1}{\tau_l}\left(-s_l + \frac{a_l}{\pi}\right)$$

- μ_l: External input to the l-type population
- δ_l: heterogenity constant for l-type population
- λ: Parameter that influences how much SOM affects others
- γ: Parameter that influences how much PV affects others

- τ_l: Decay time constant for the l-type population
- τ_{ml}: Membrane time constant for l-type population
- g_{kl}: Interaction strengths/ connectivity coefficients(if $k = l$) or coupling parameter(if $k \neq l$) between k-type and l-type population

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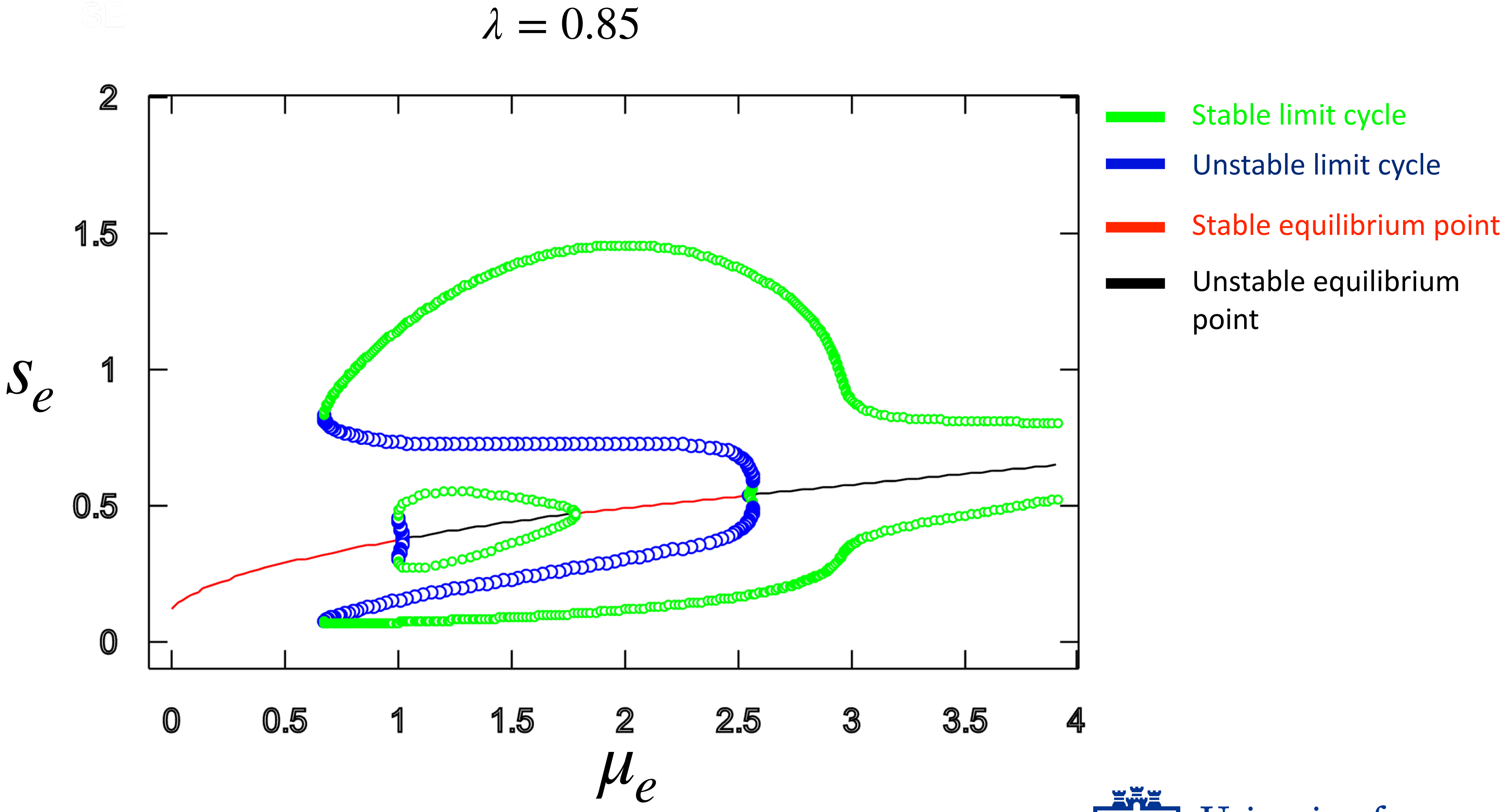
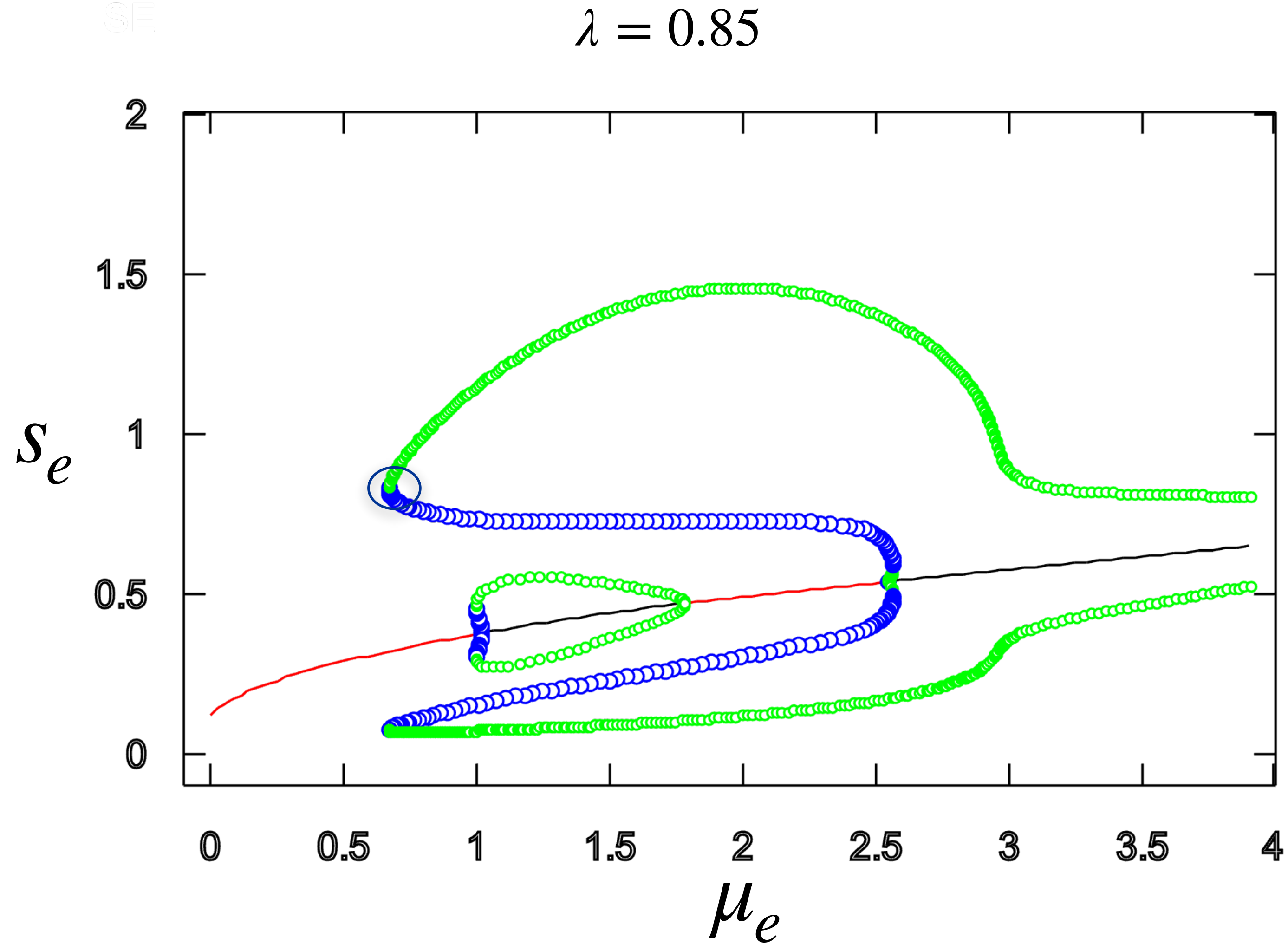
Bifurcation Plots μ_e vs λ (effect of SOM)



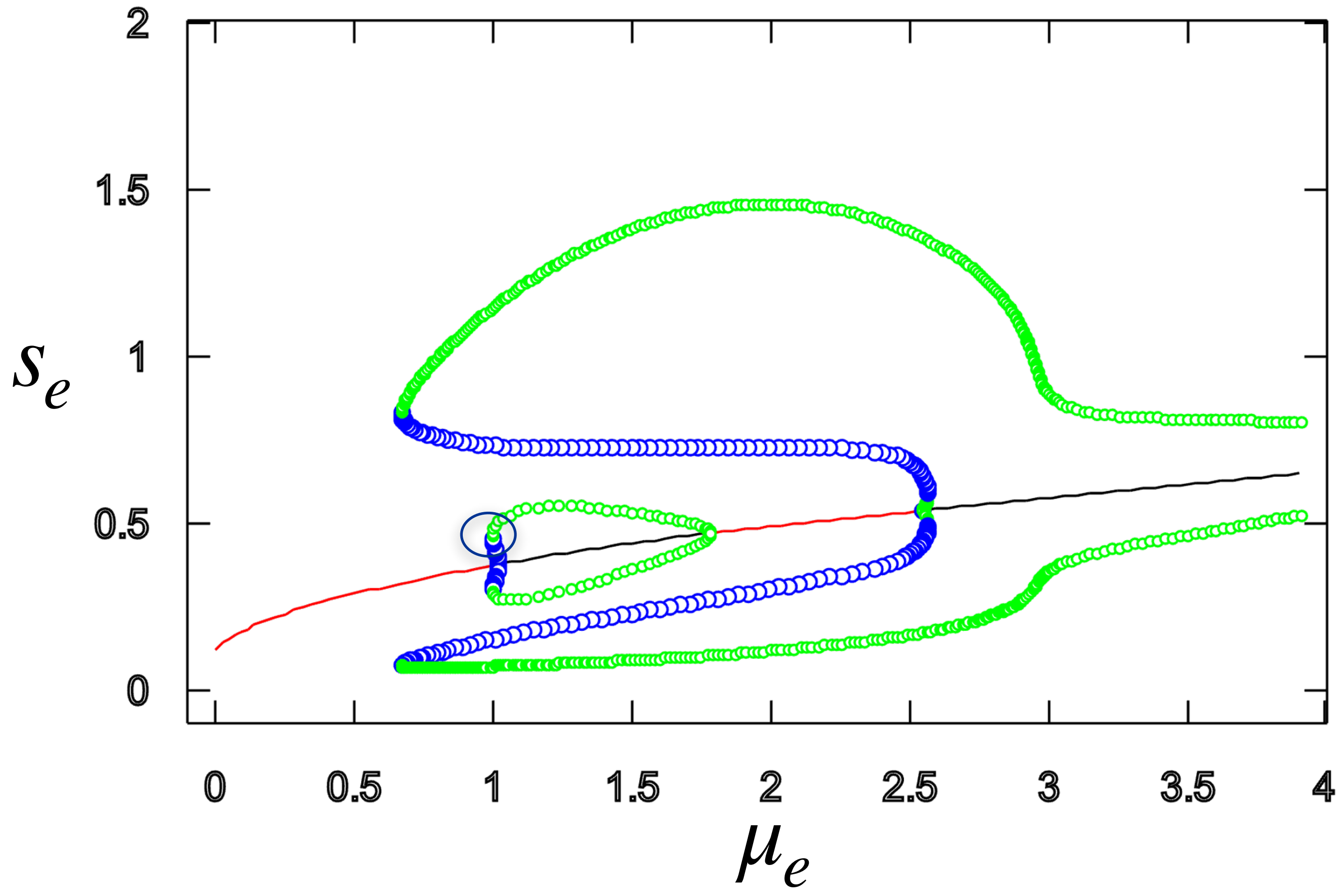
Bifurcation Plots μ_e vs λ



Fold limit cycle bifurcation

Bifurcation Plots μ_e vs λ

$\lambda = 0.85$



Fold limit cycle bifurcation

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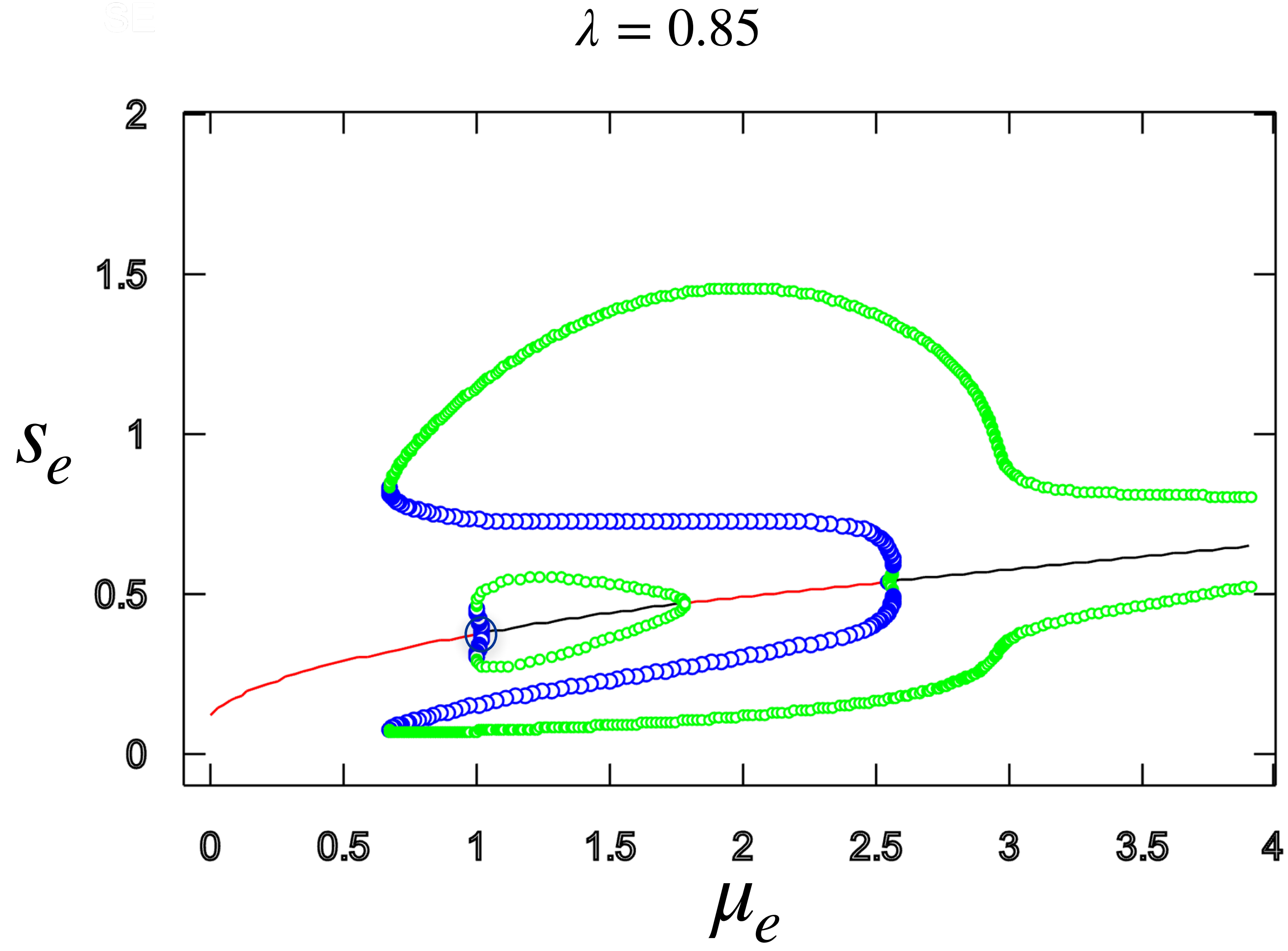
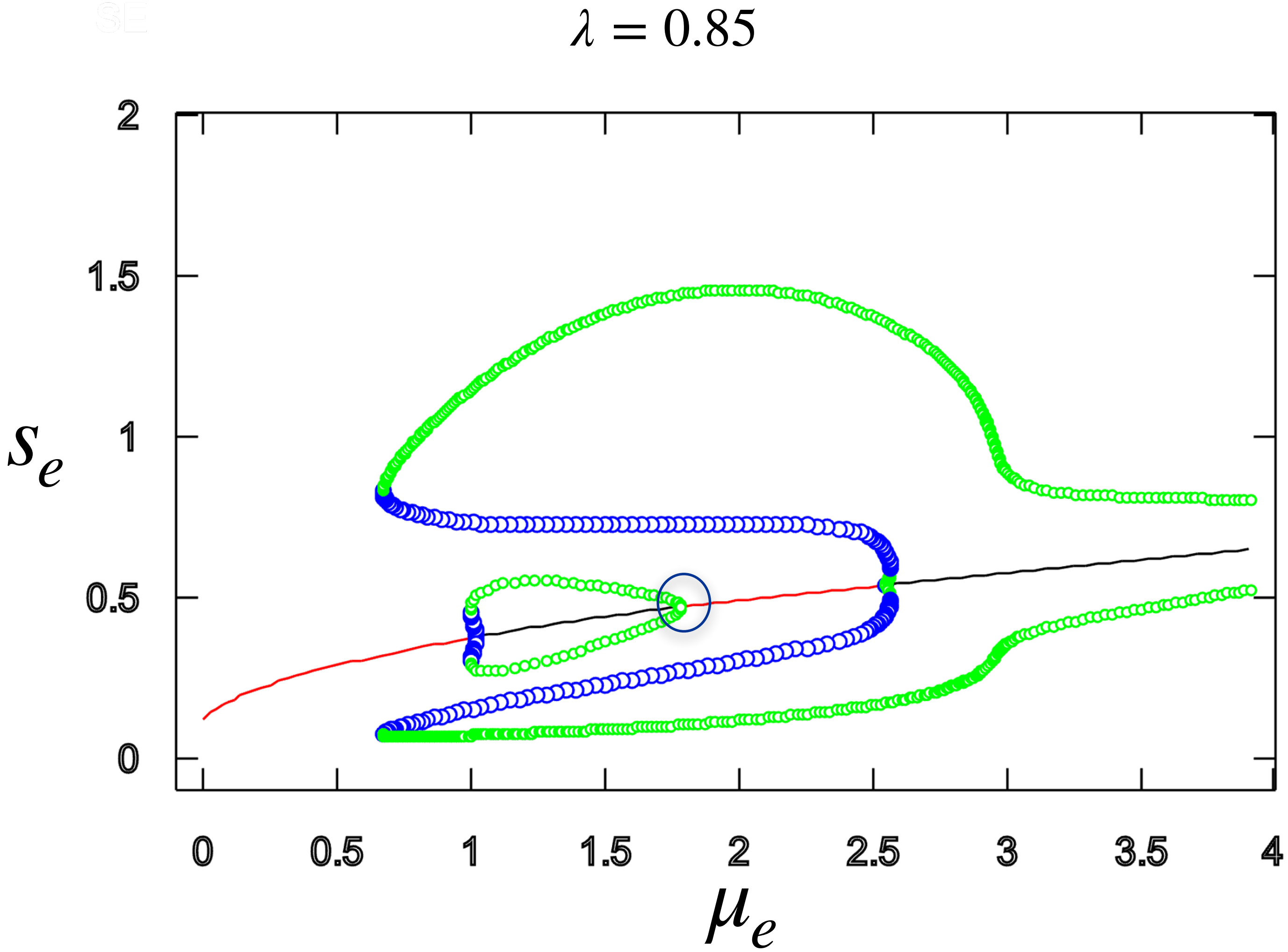
Bifurcation Plots μ_e vs λ



Subcritical Hopf bifurcation

Bifurcation Plots μ_e vs λ



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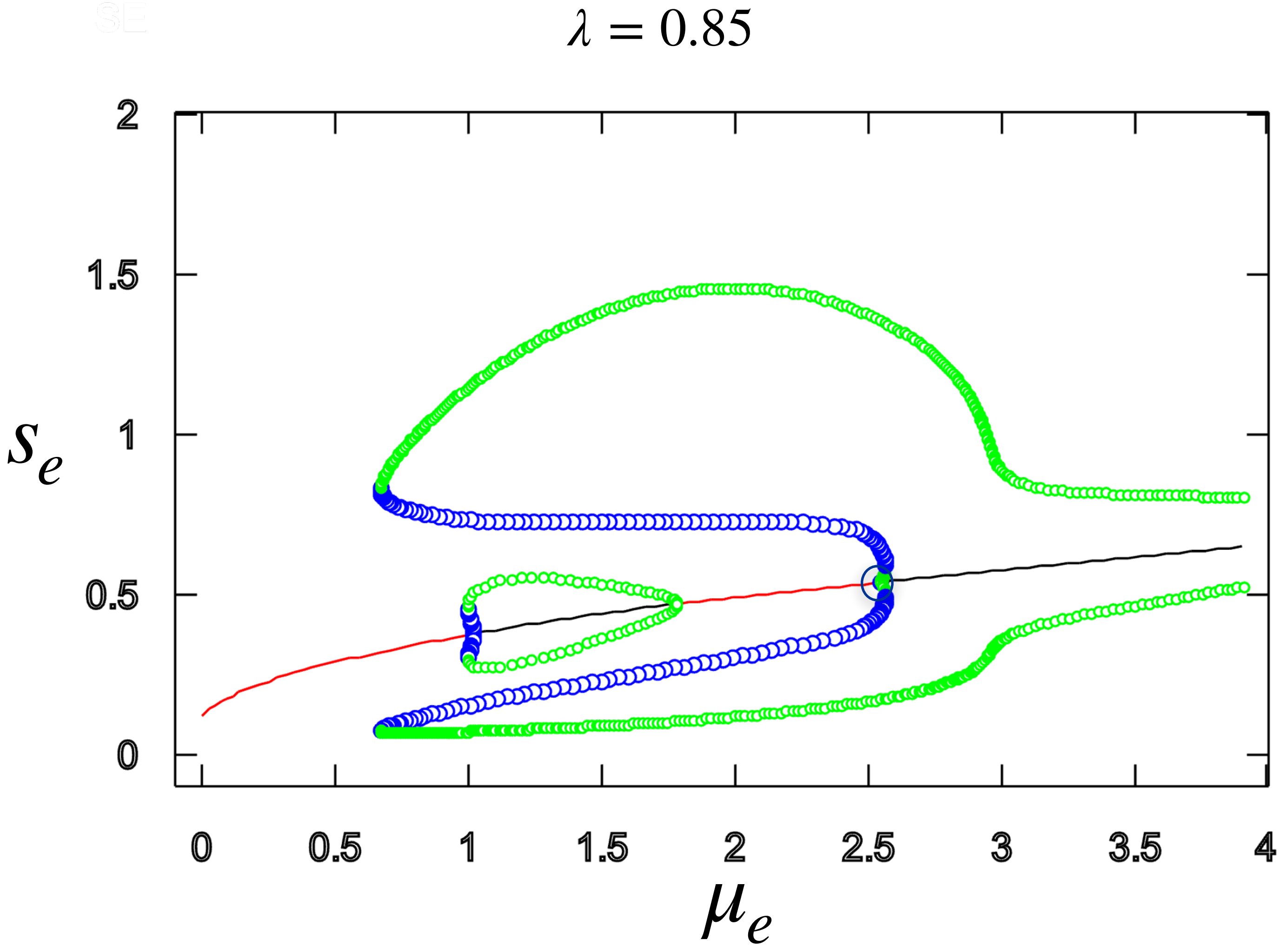

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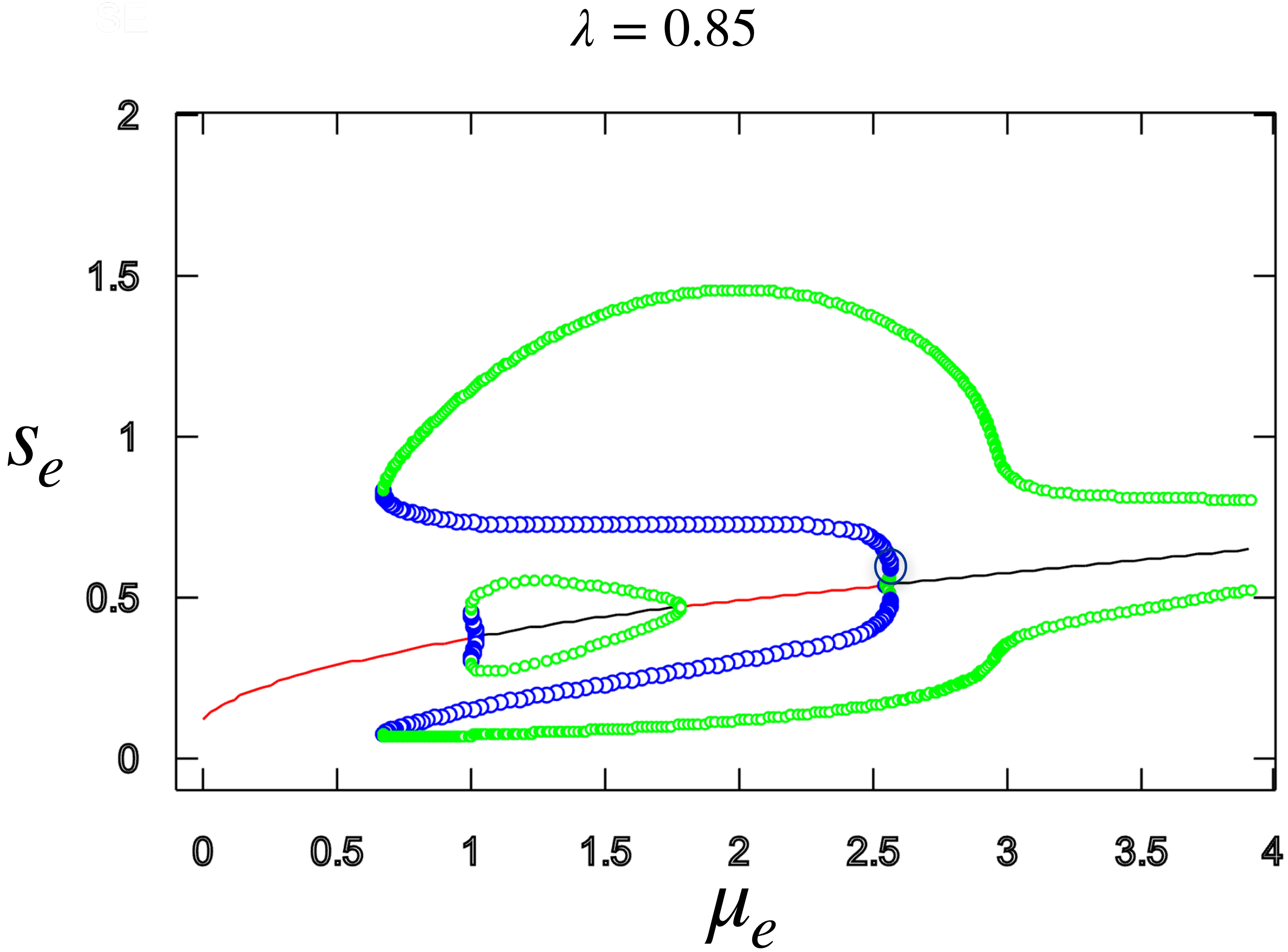

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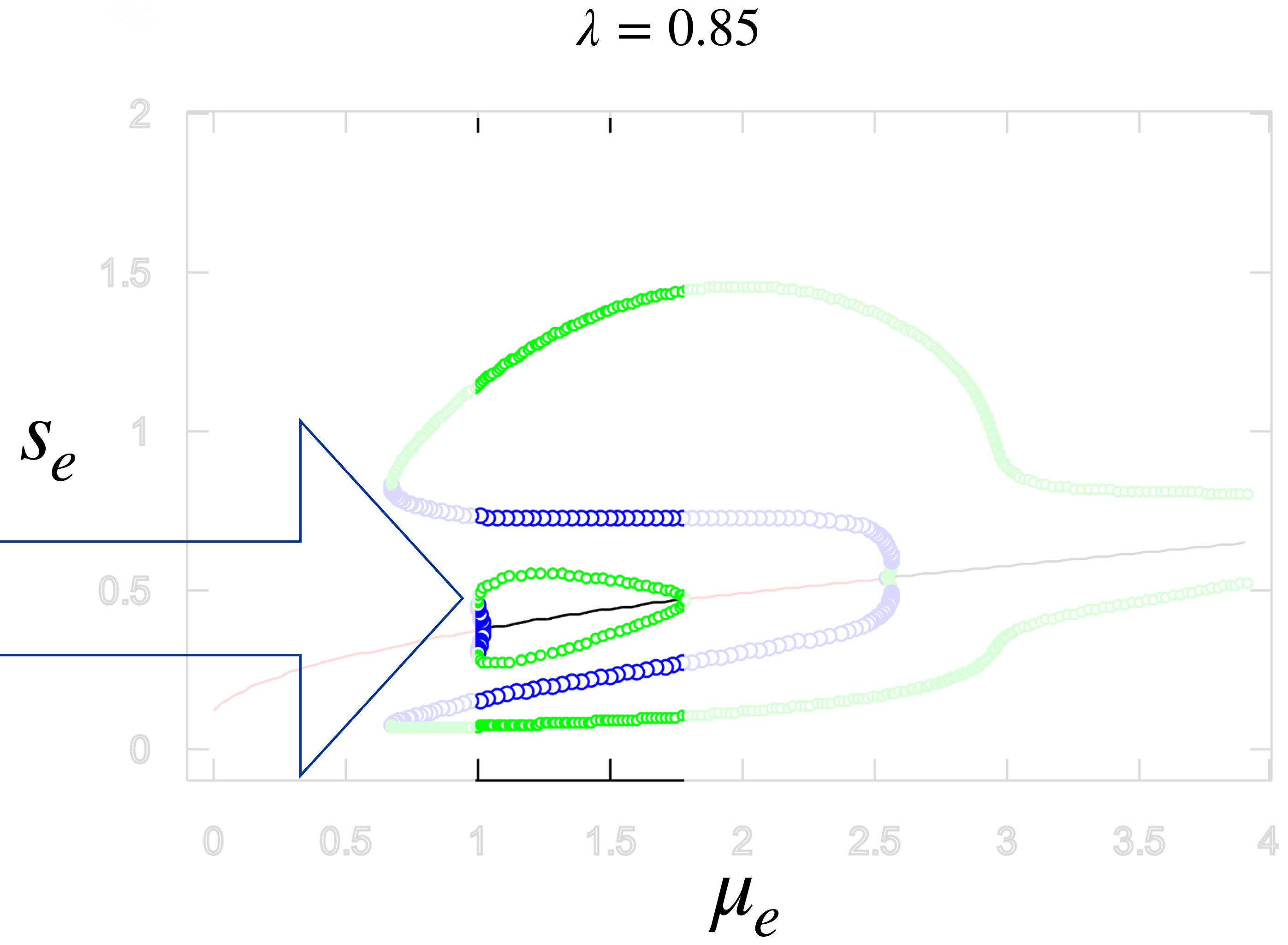
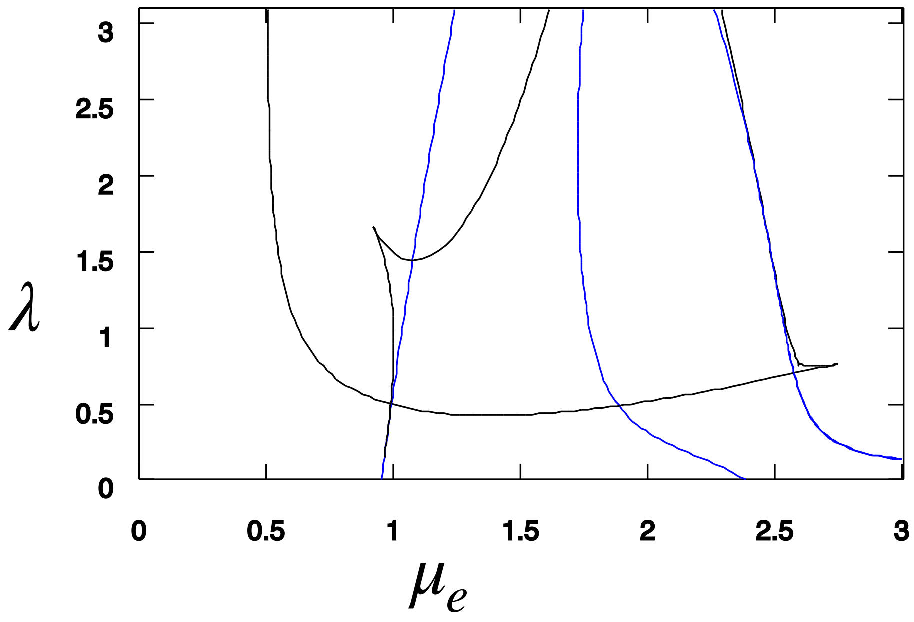
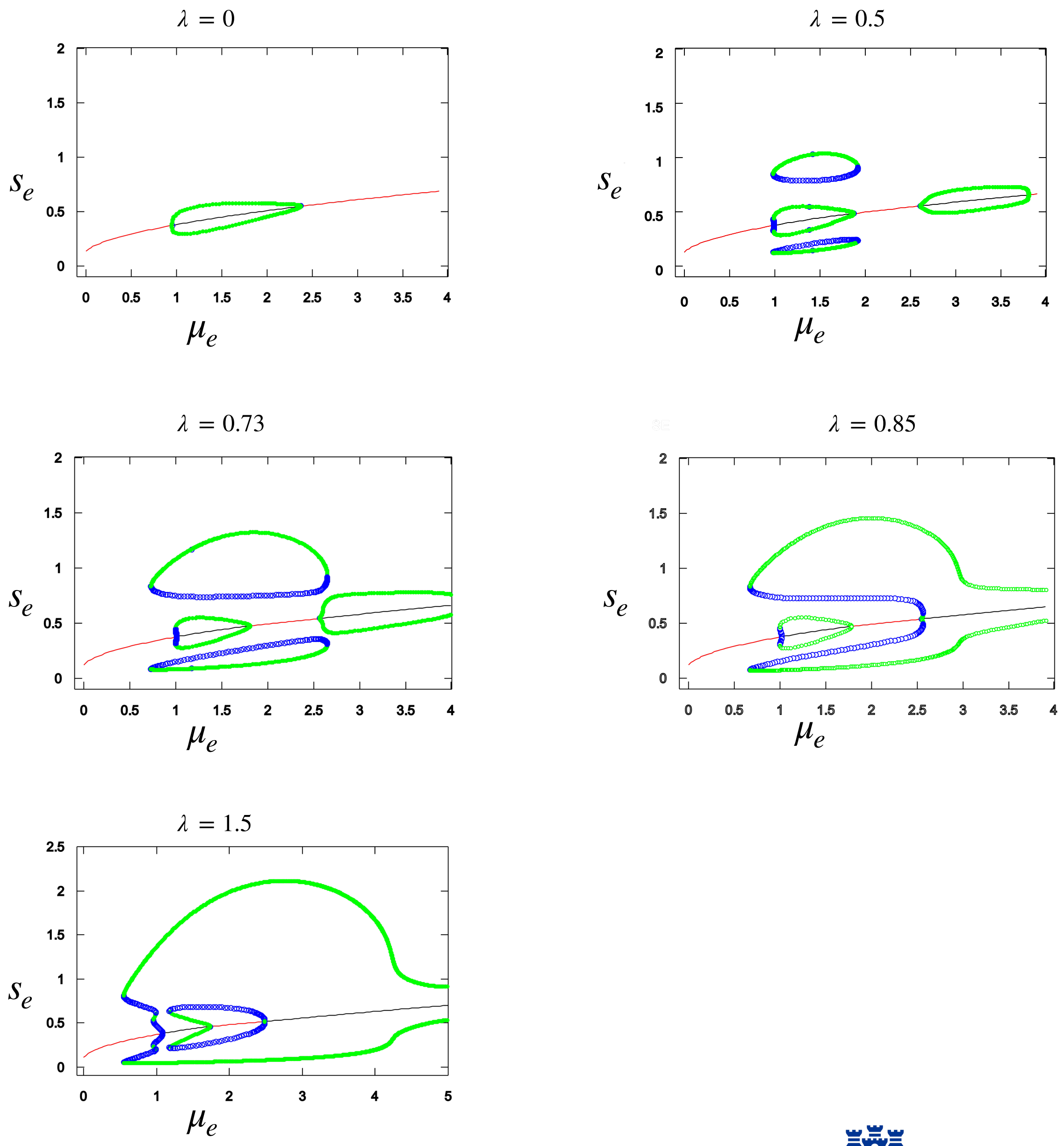


Region of
Bi-rhythmicity

Bifurcation Plots μ_e vs λ





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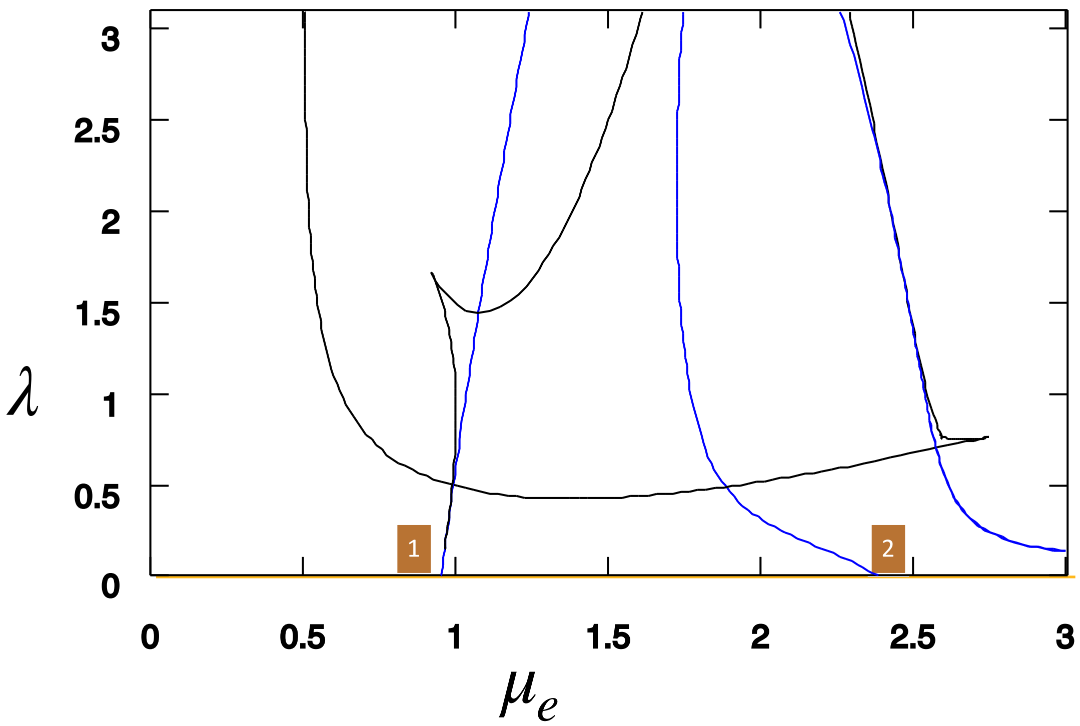

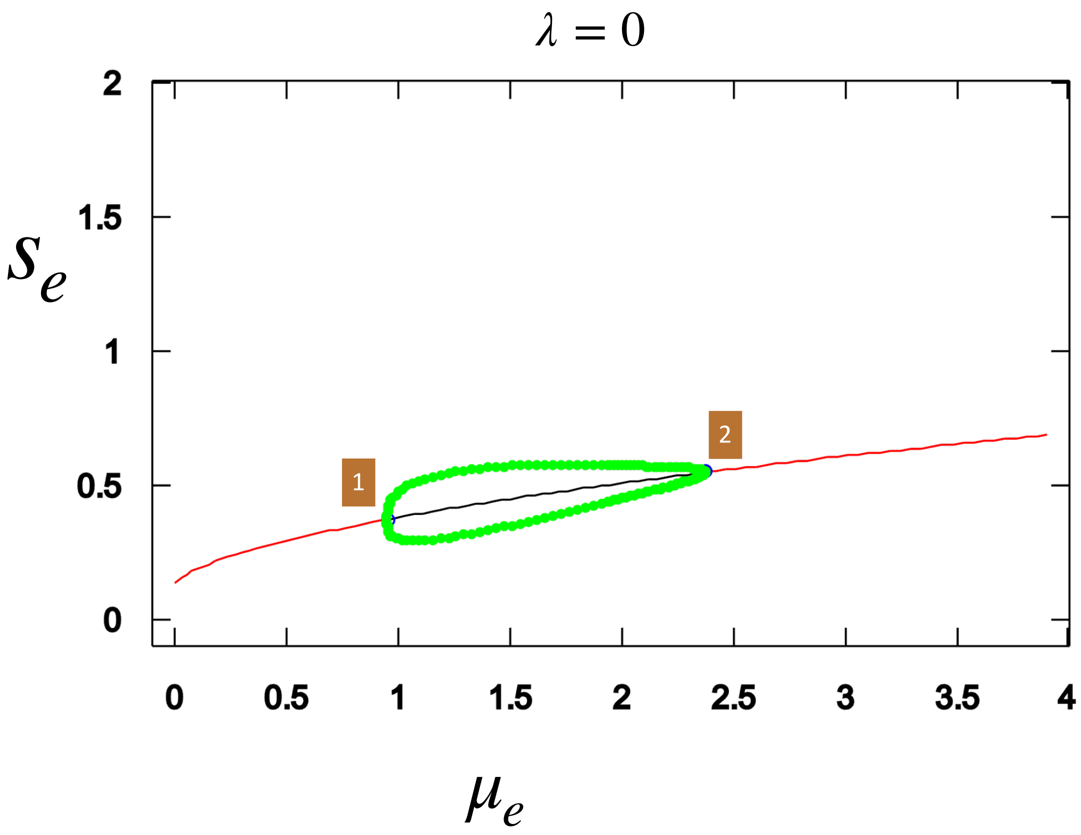

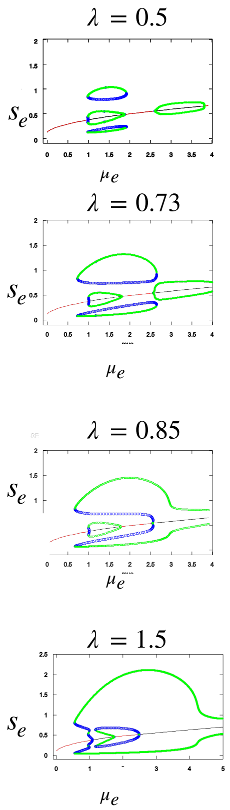

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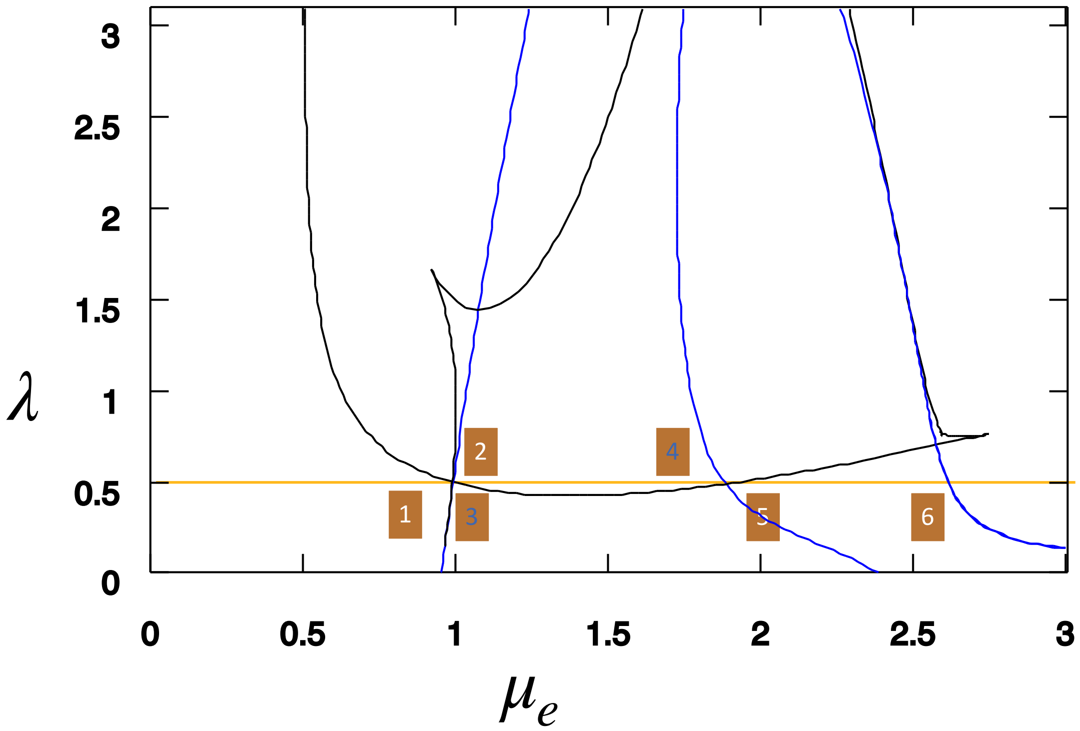

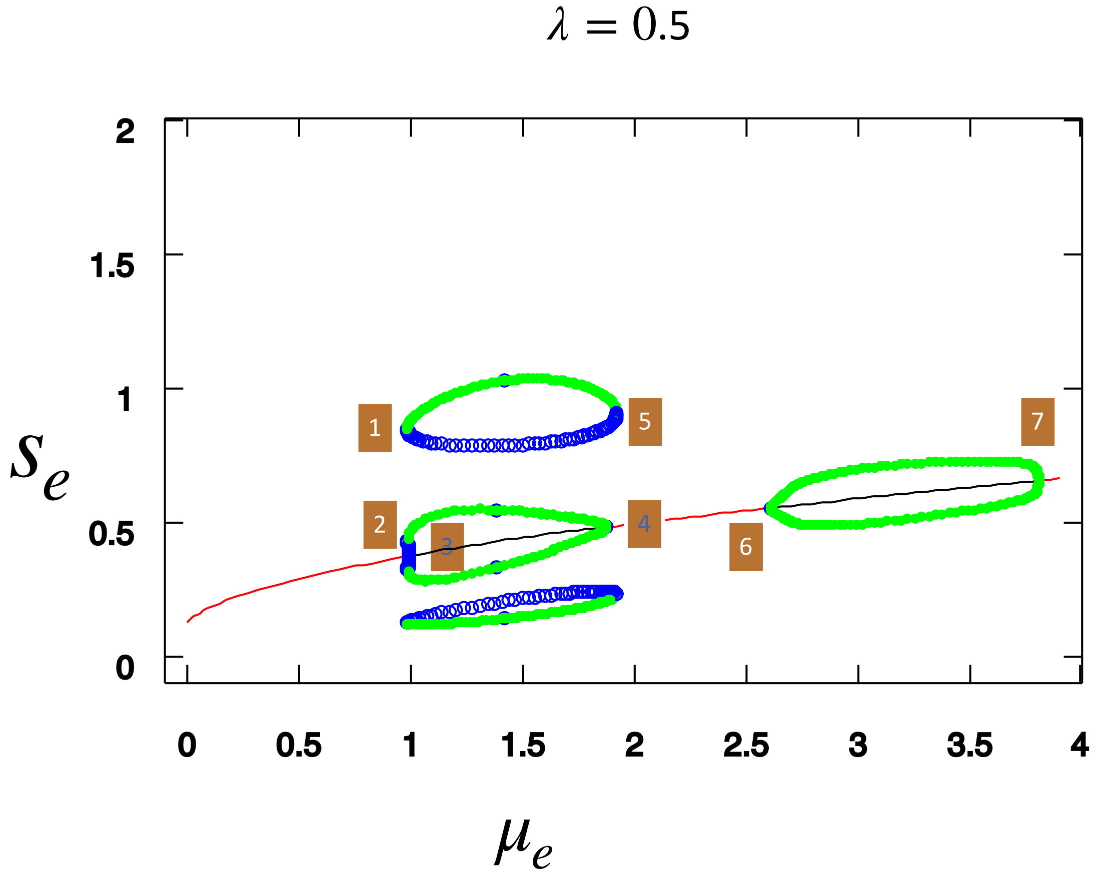

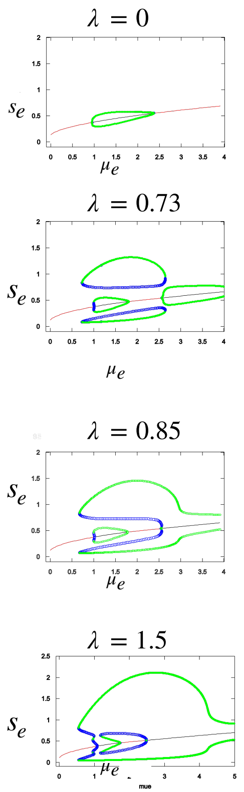

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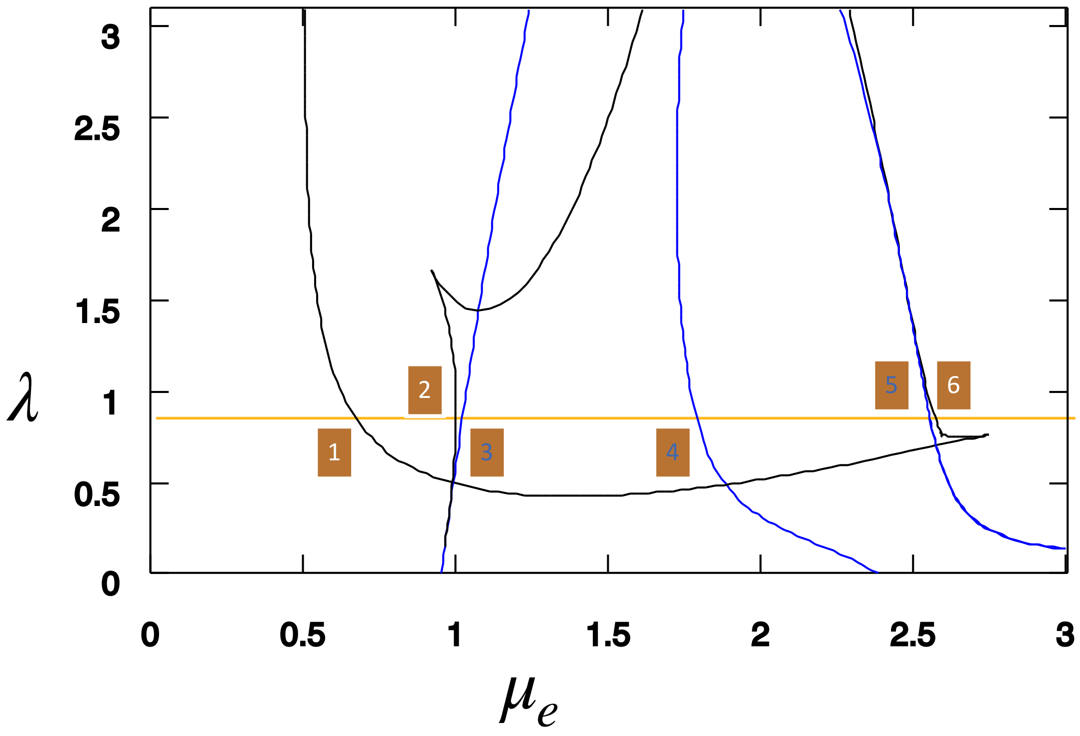

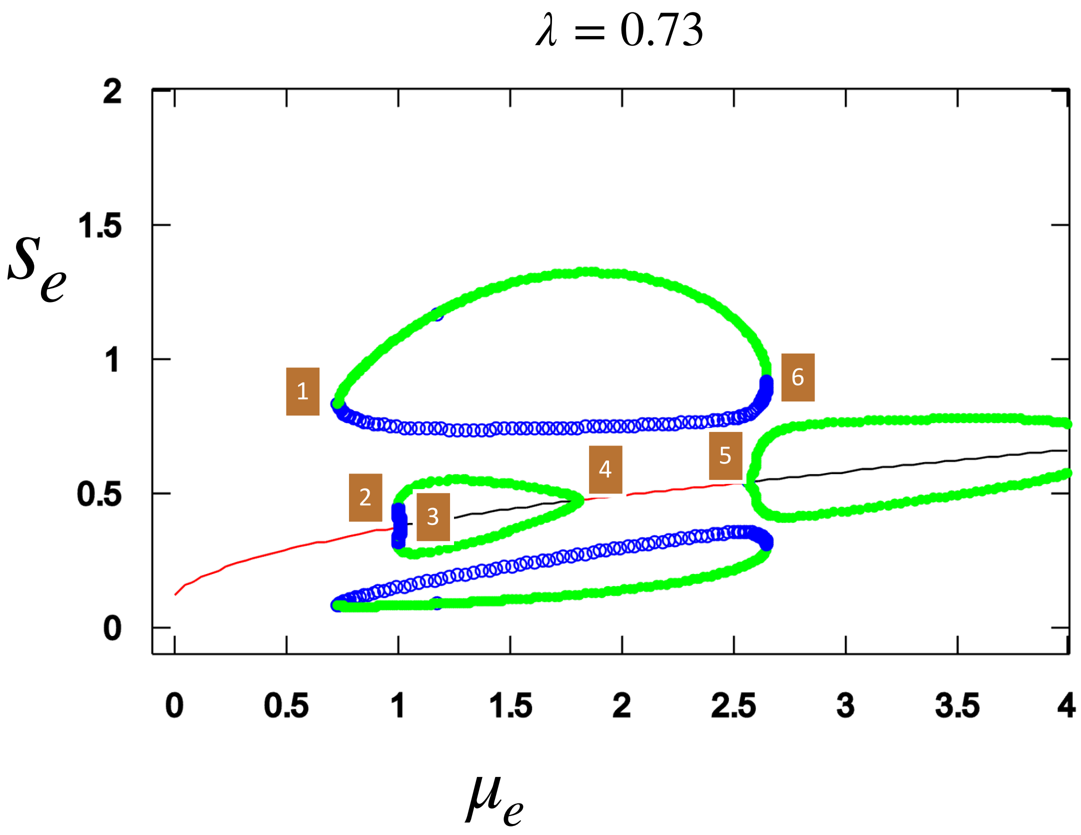

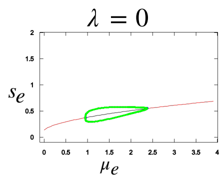

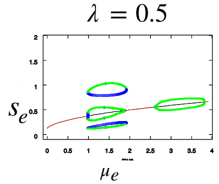

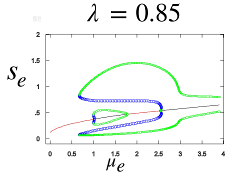

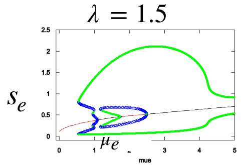

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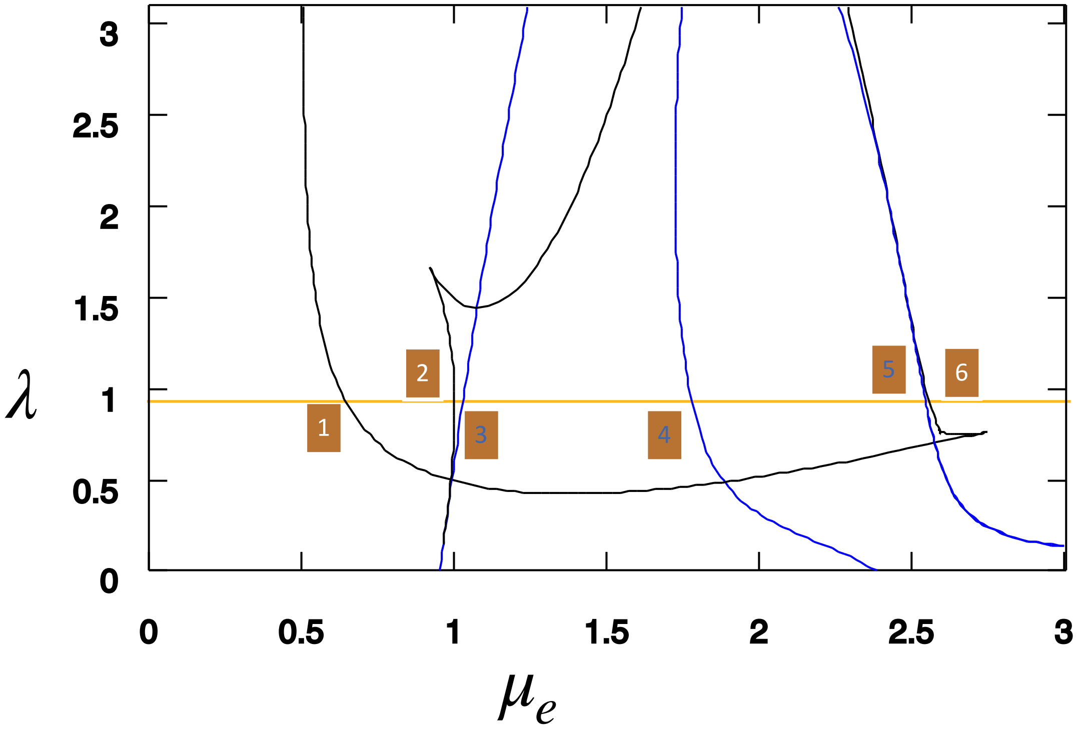
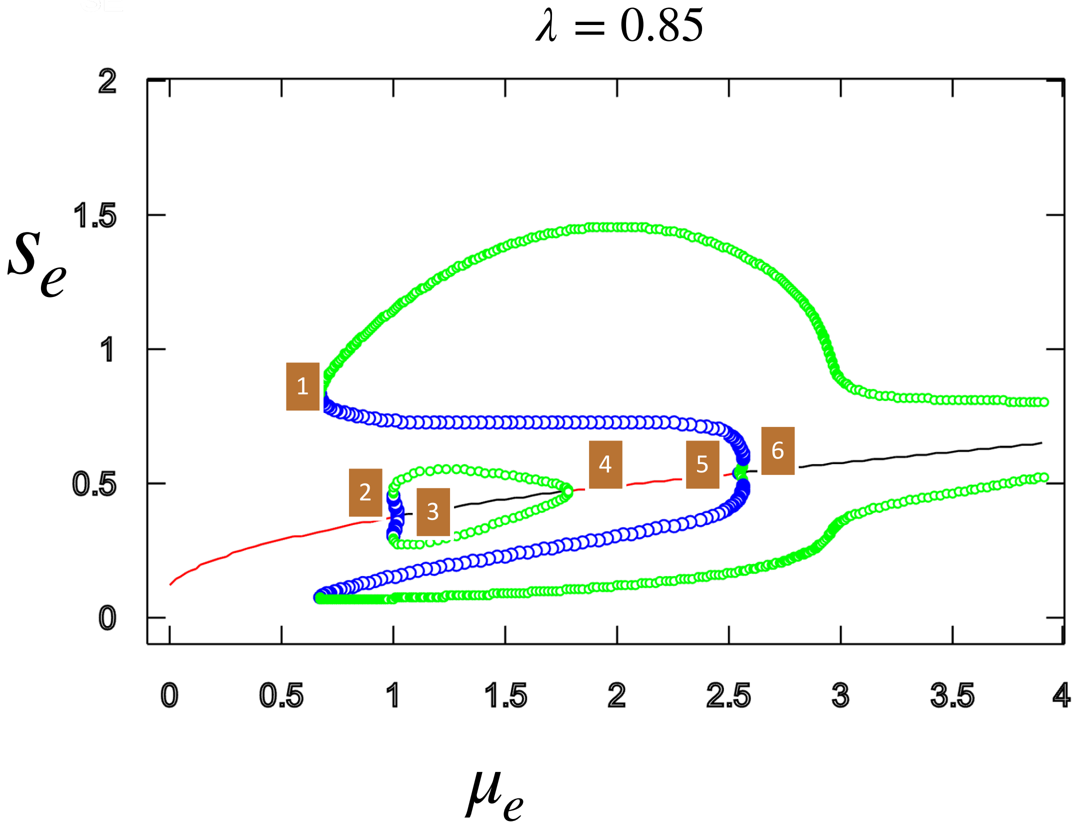
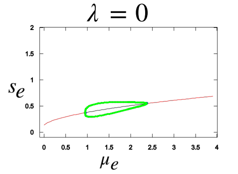
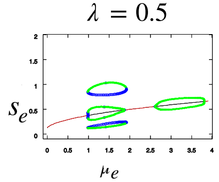
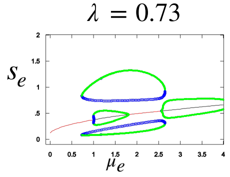
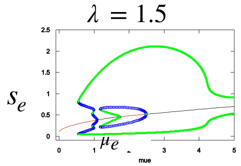




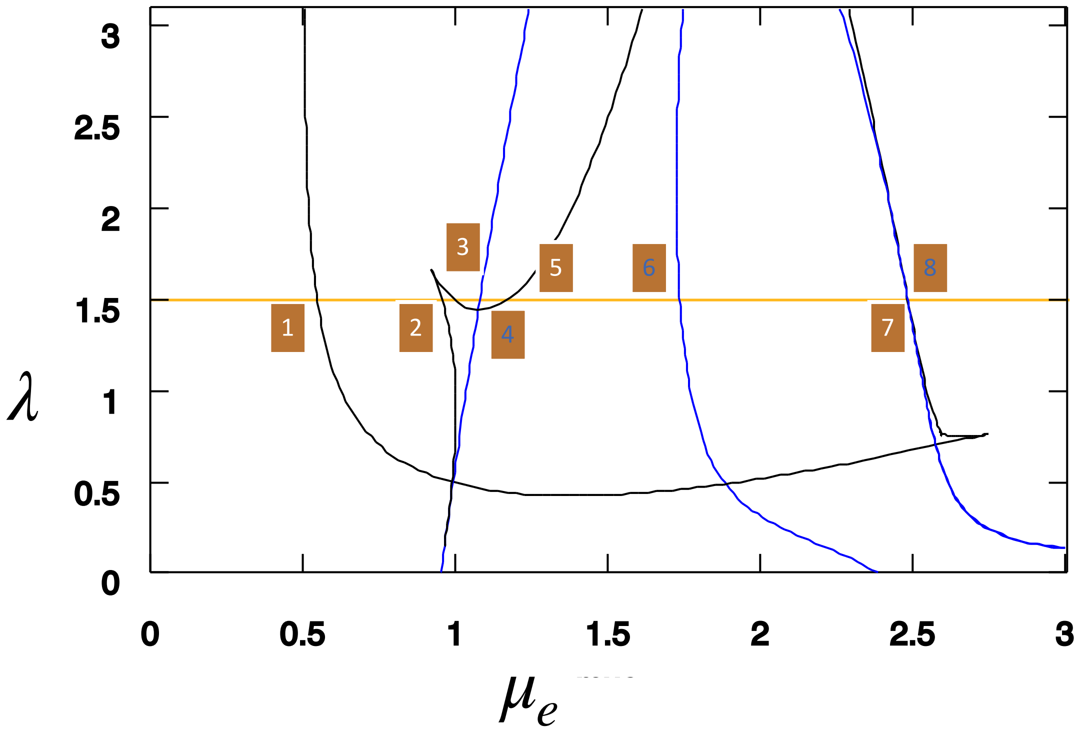


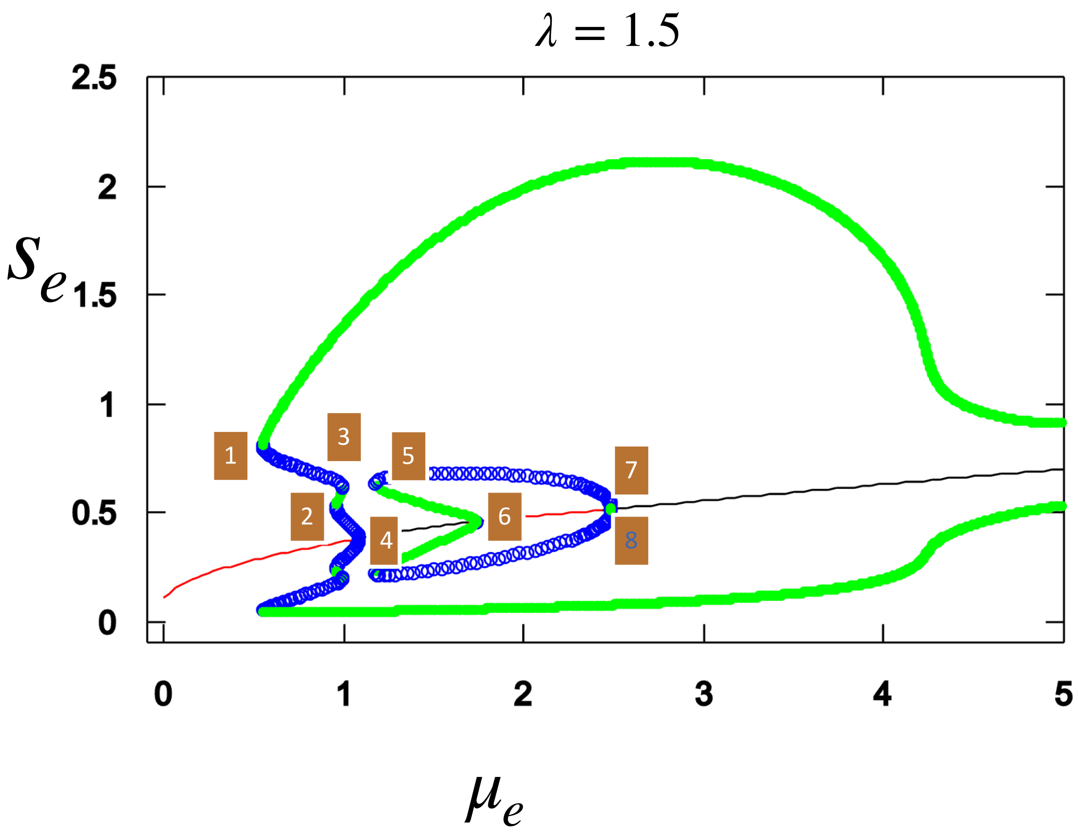


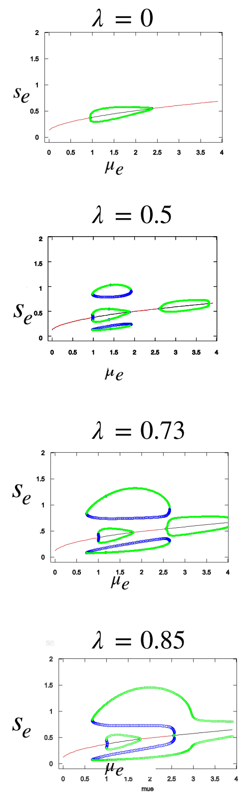




Bifurcation Plots μ_e vs λ







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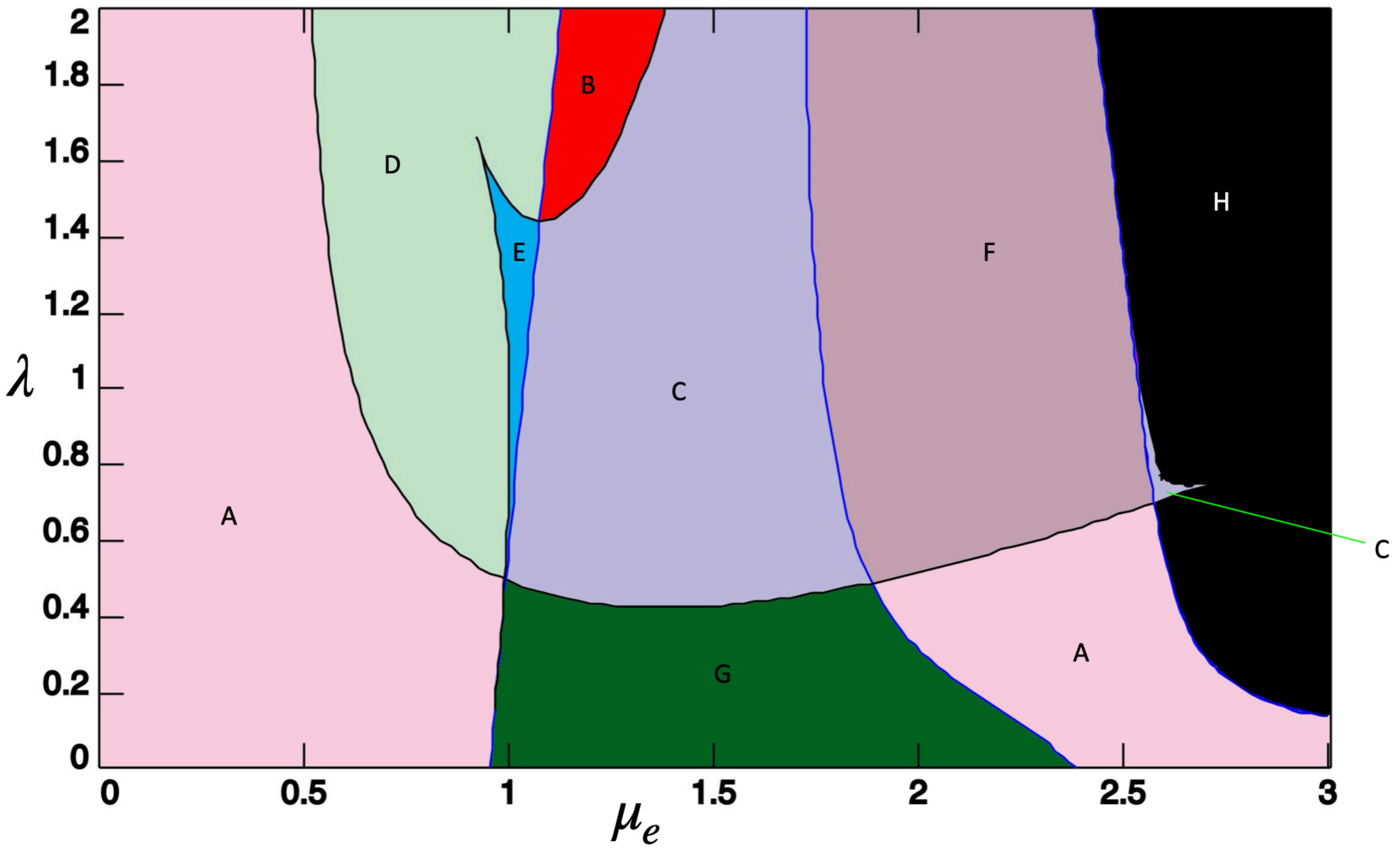
Regions in 2 par bifurcation



A: Stable fixed point, no limit cycle

B: Unstable fixed point, one stable limit cycle(bigger)

C: Unstable fixed point, Birhythmicity

D: Stable fixed point, One stable limit cycle

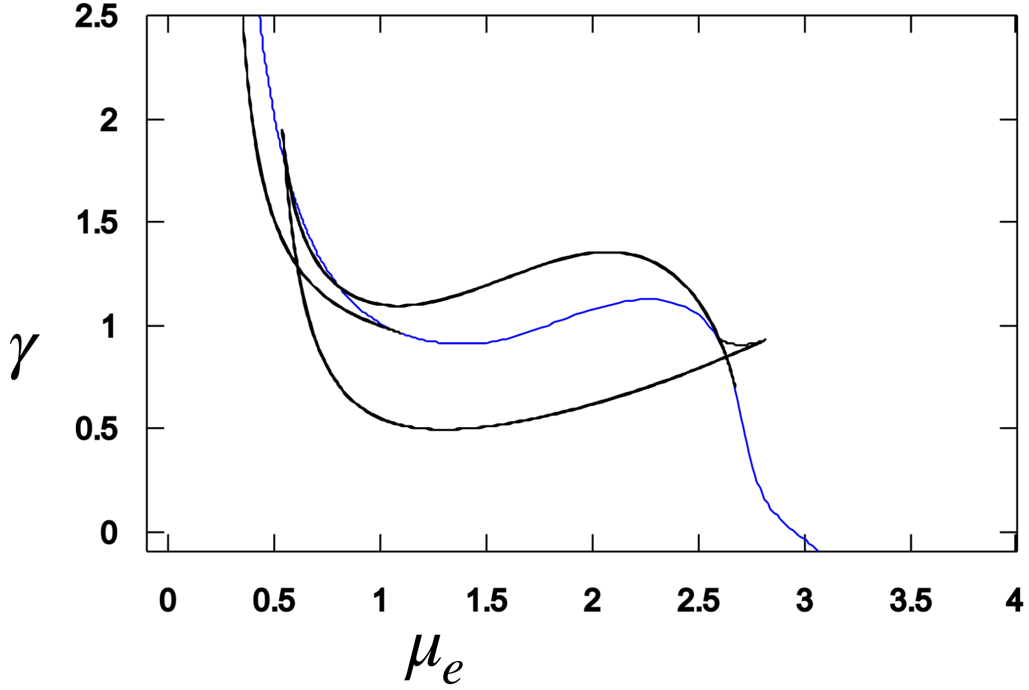
E: Stable fixed point, Birhythmicity

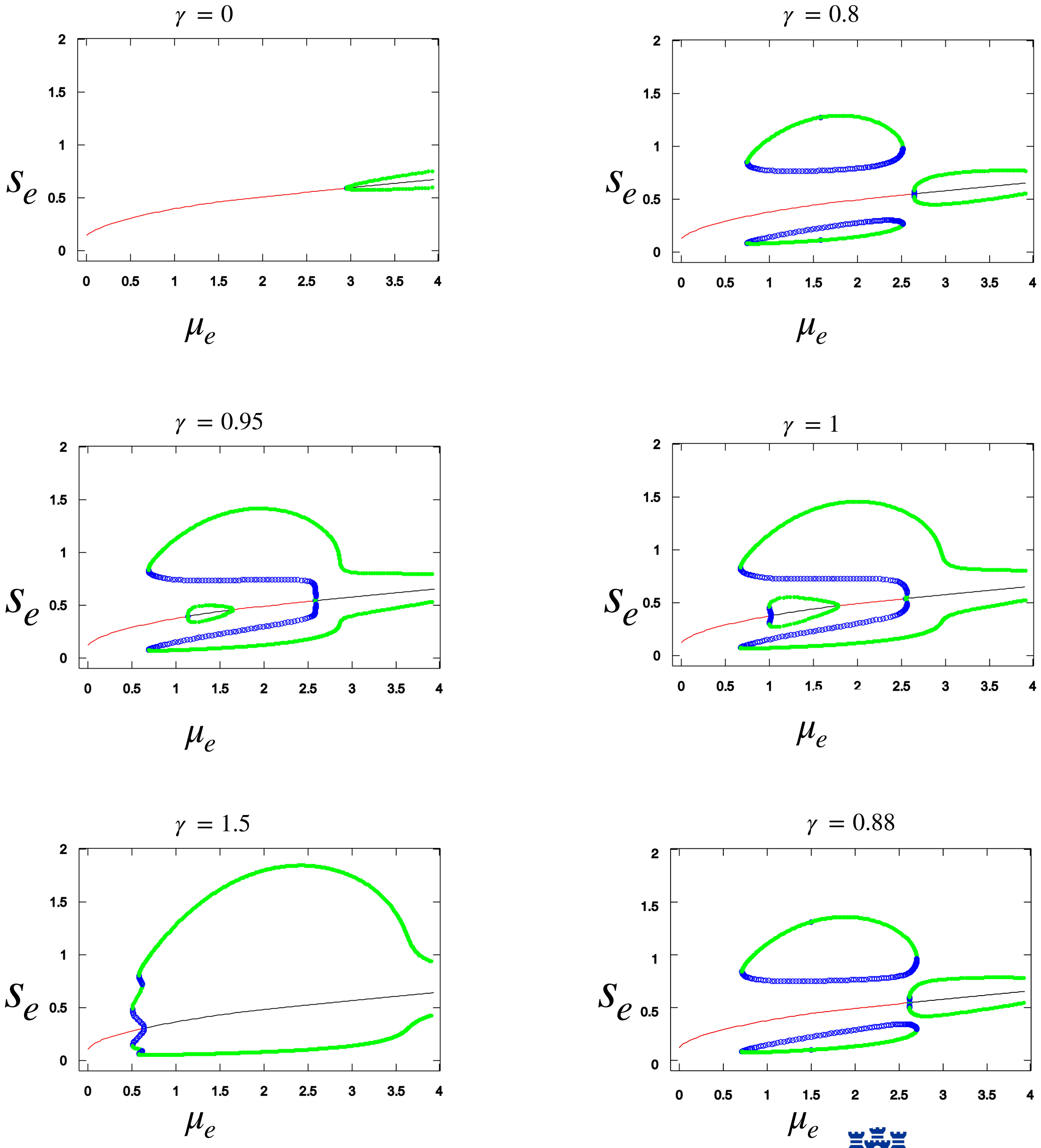
F: Stable fixed point, One stable limit cycle(bigger)

G: Unstable fixed point, One stable limit cycle(smaller)

H: Unstable fixed point, one stable limit cycle(smaller) 0.925, 0.88

Bifurcation Plots μ_e vs γ (effect of PV)





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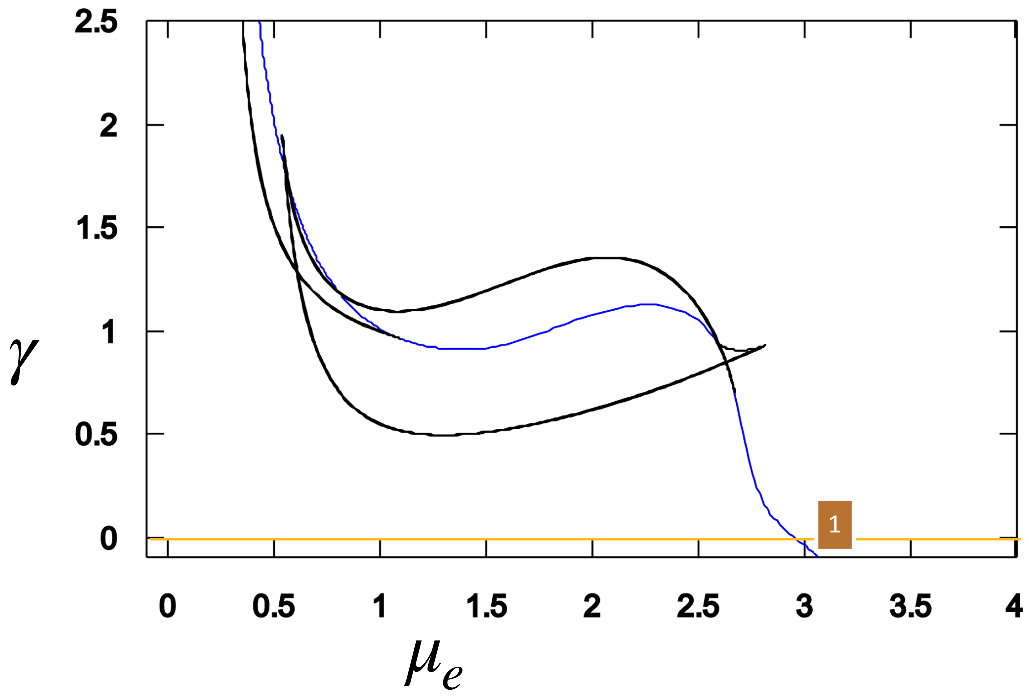
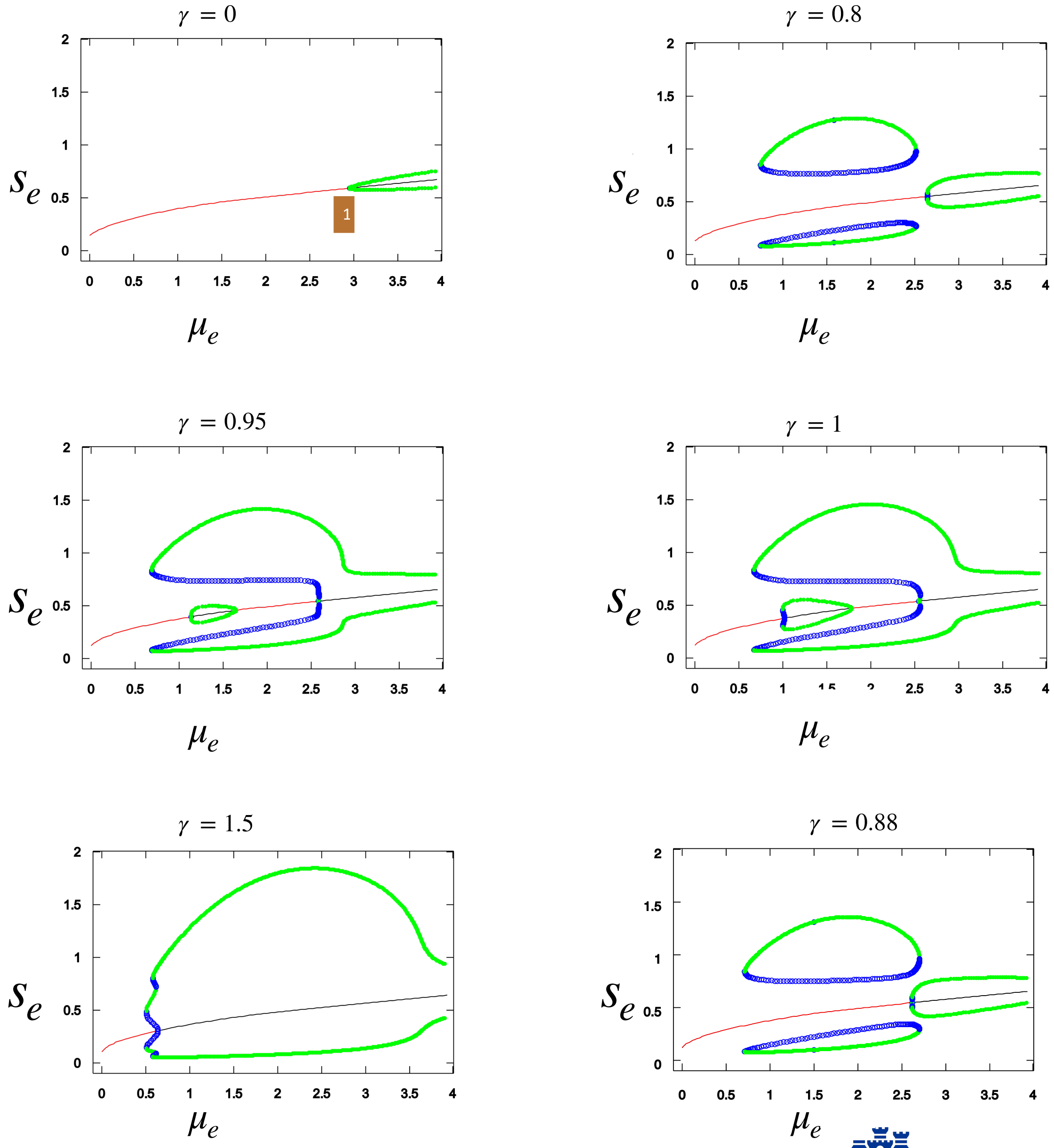
Bifurcation Plots μ_e vs γ





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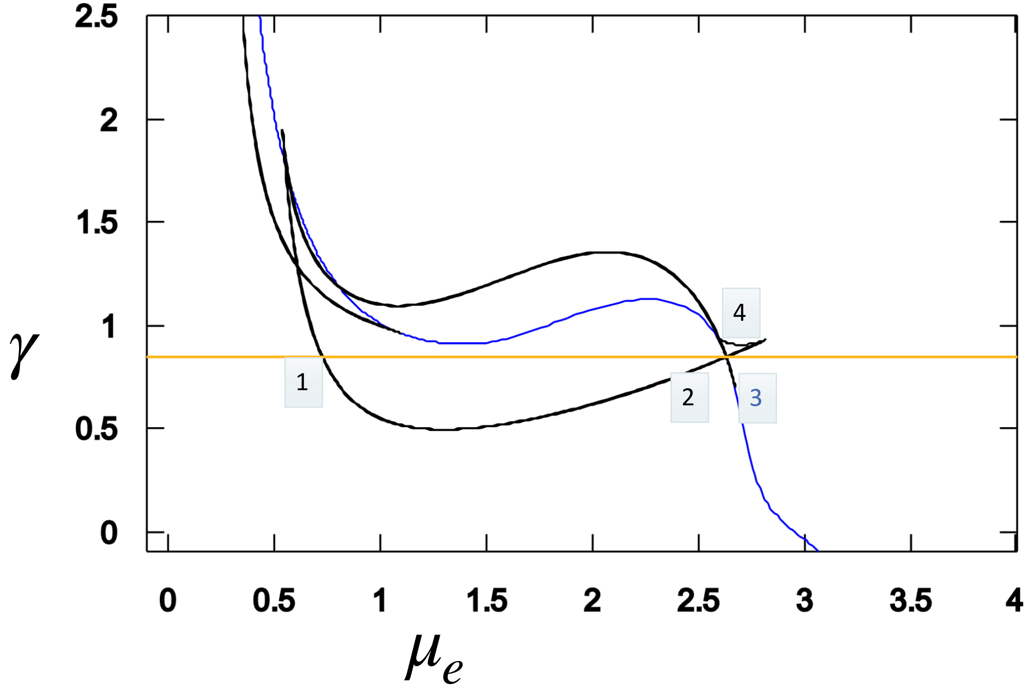
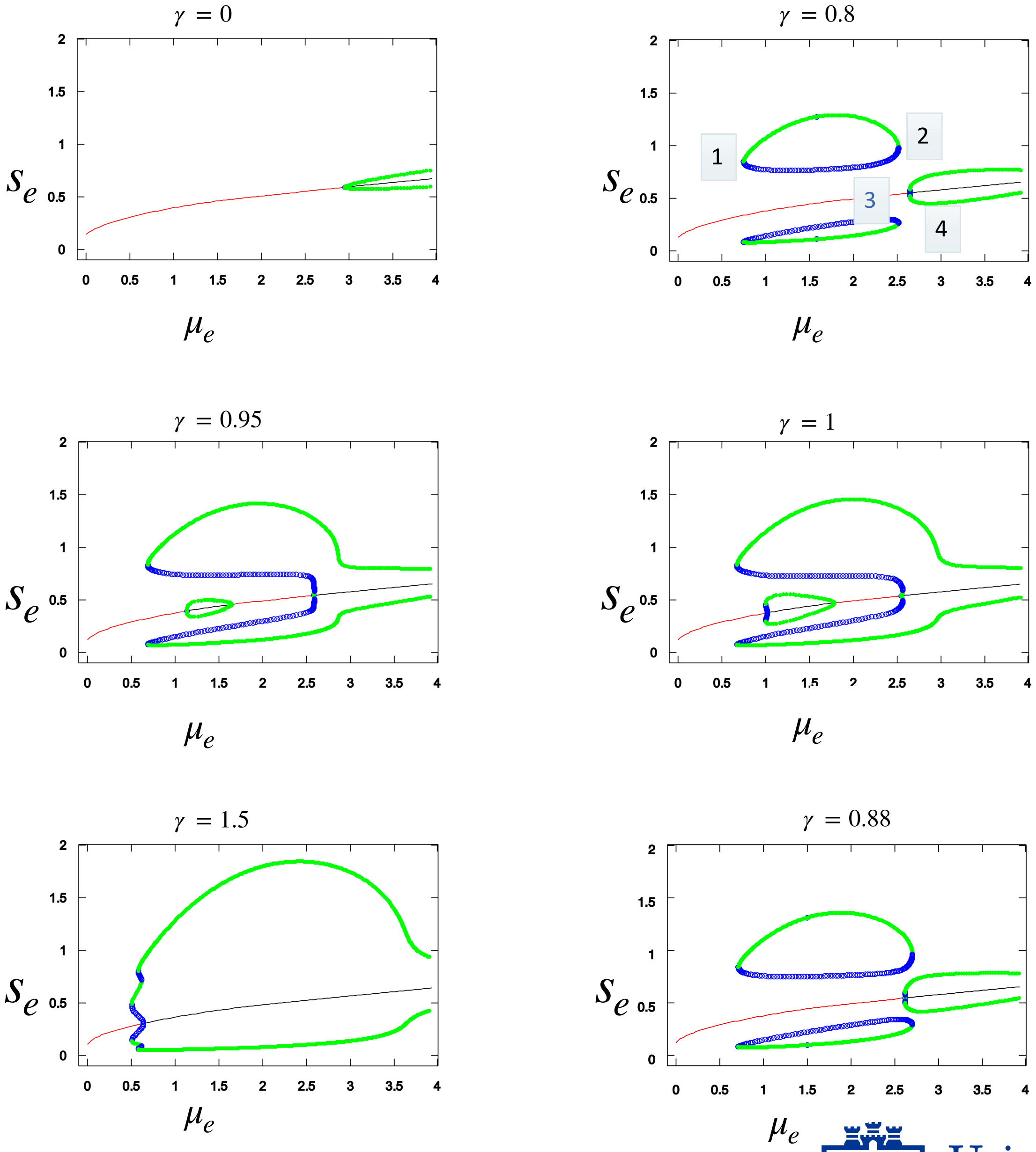
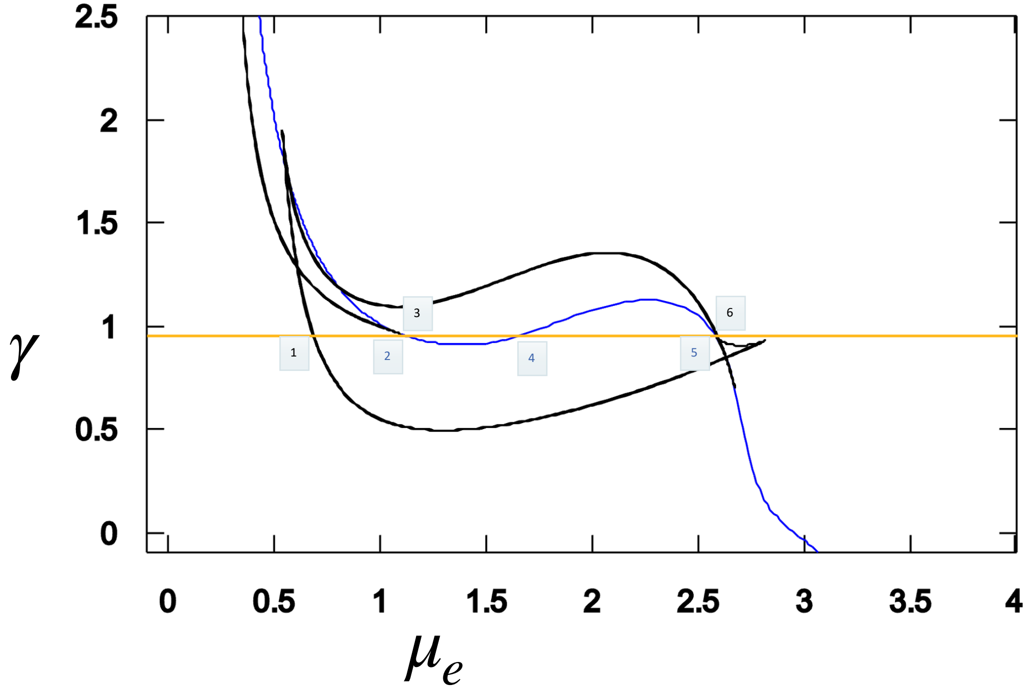
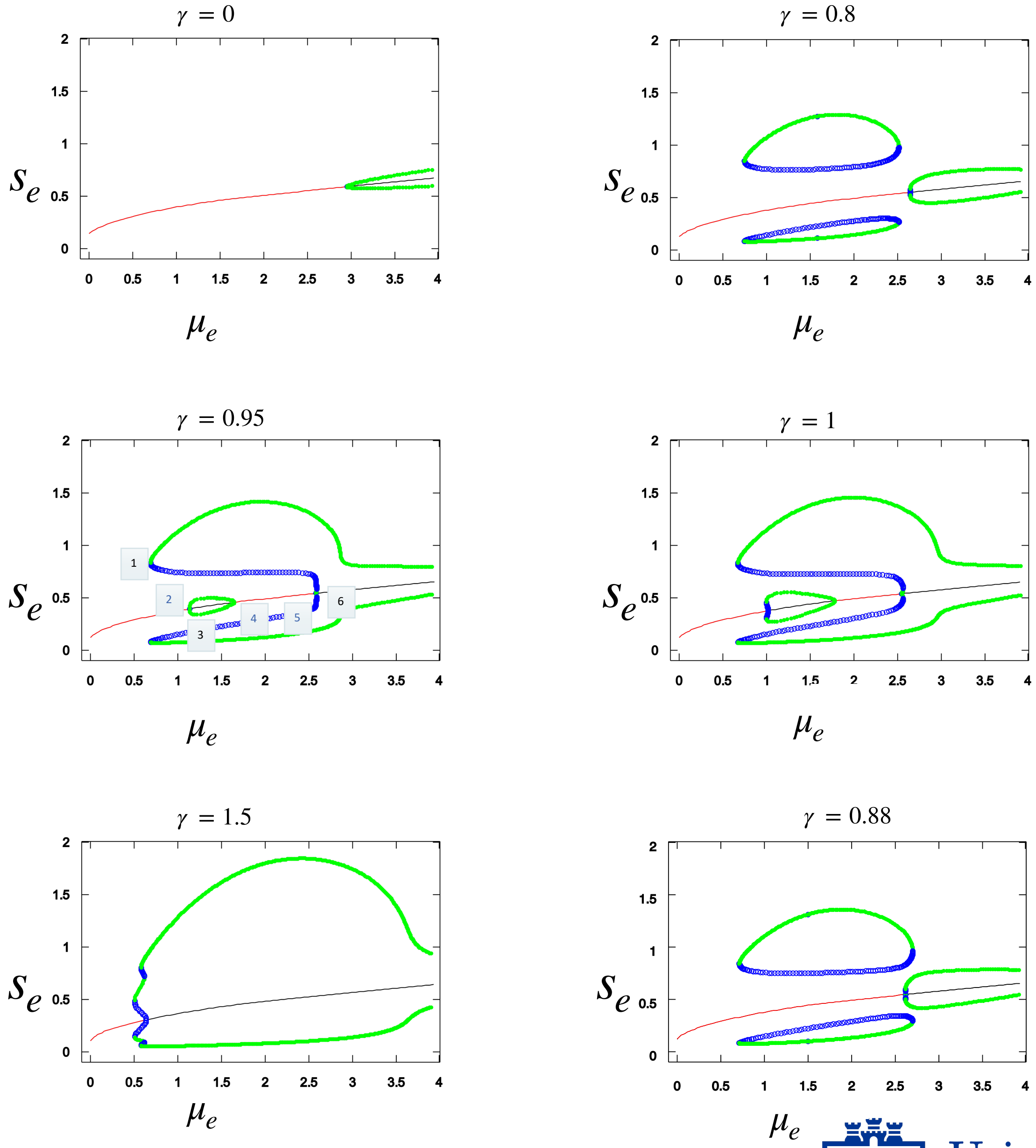
Bifurcation Plots μ_e vs γ



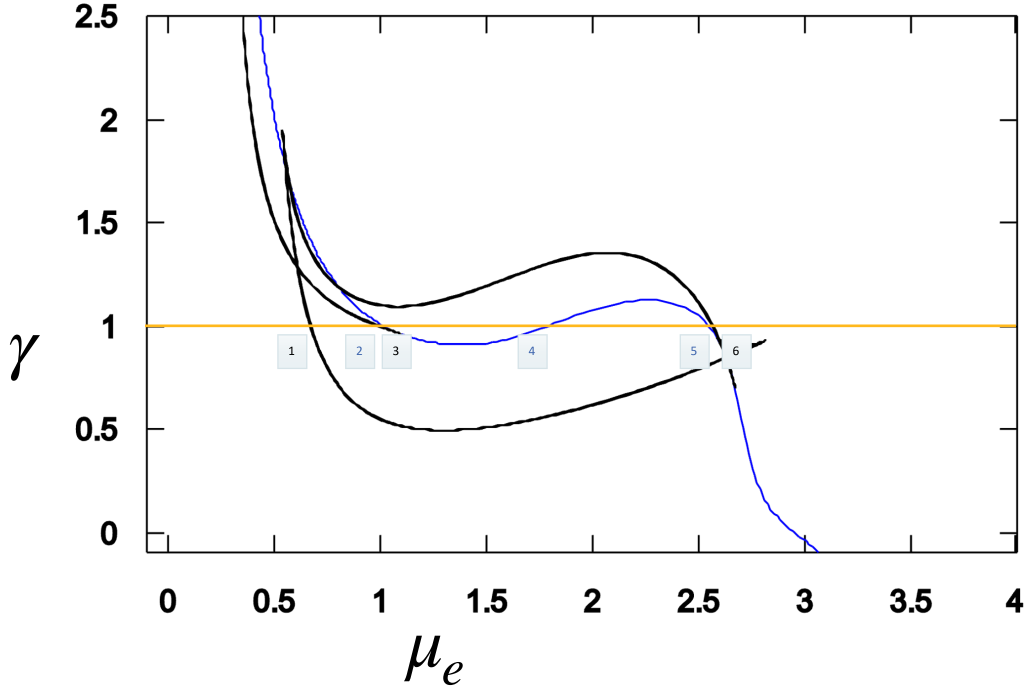


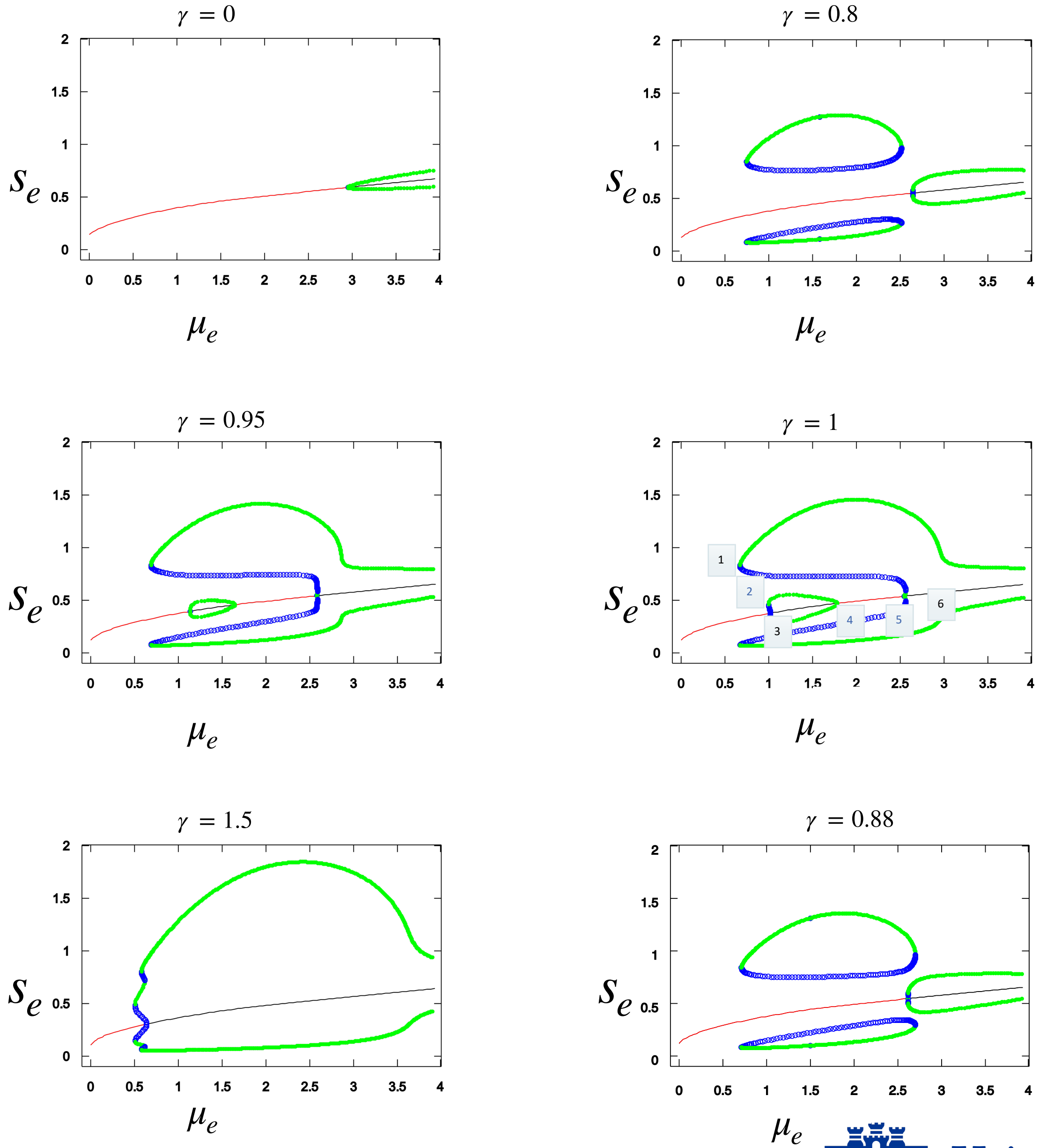
Bifurcation Plots μ_e vs γ





Bifurcation Plots μ_e vs γ





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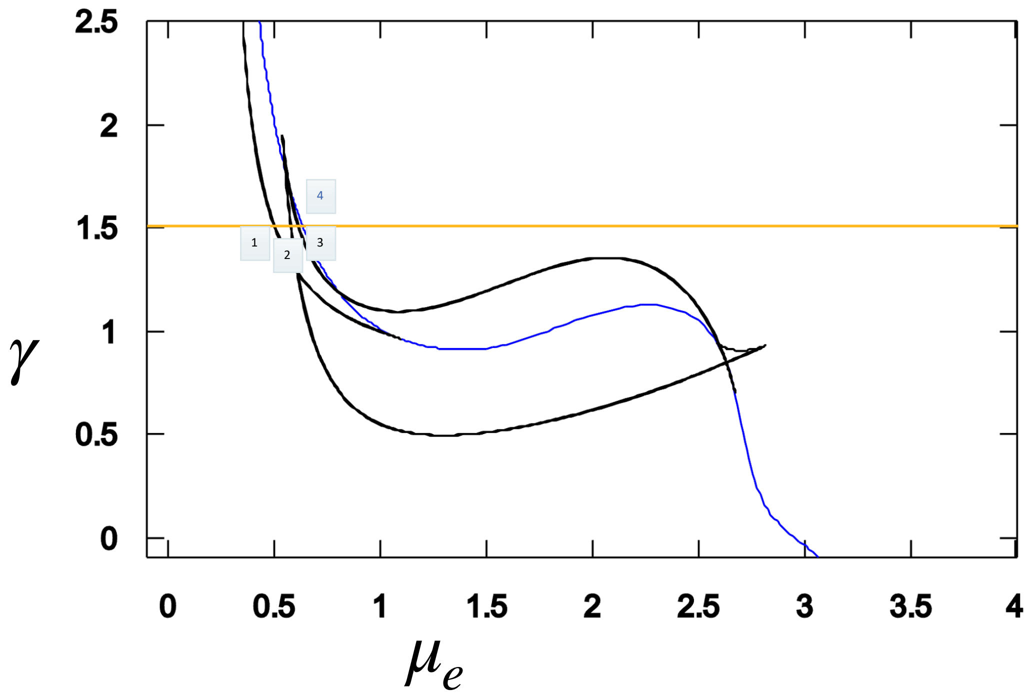
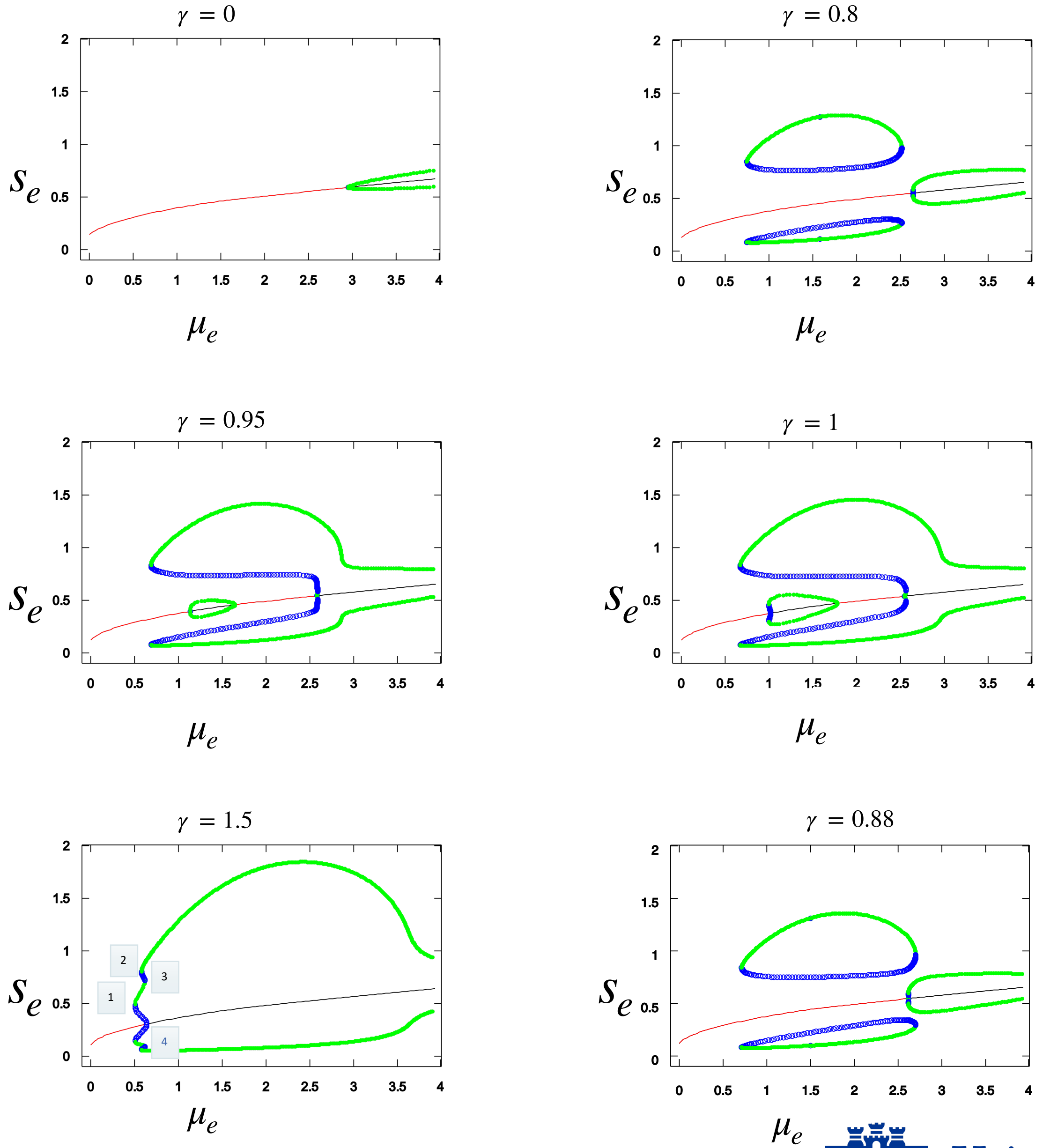
Bifurcation Plots μ_e vs γ





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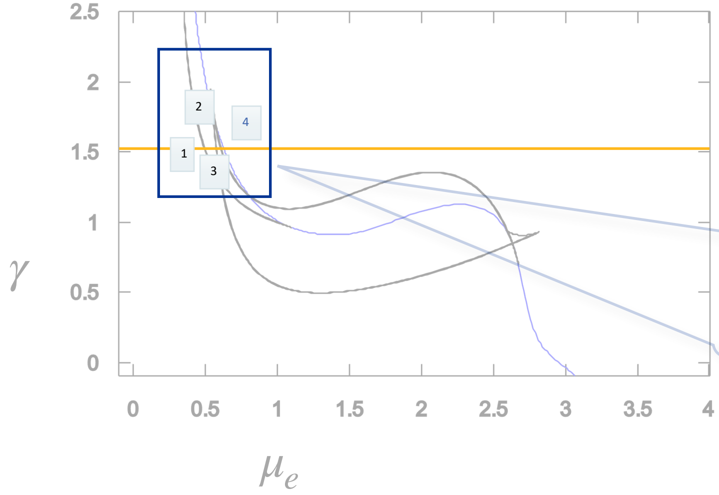
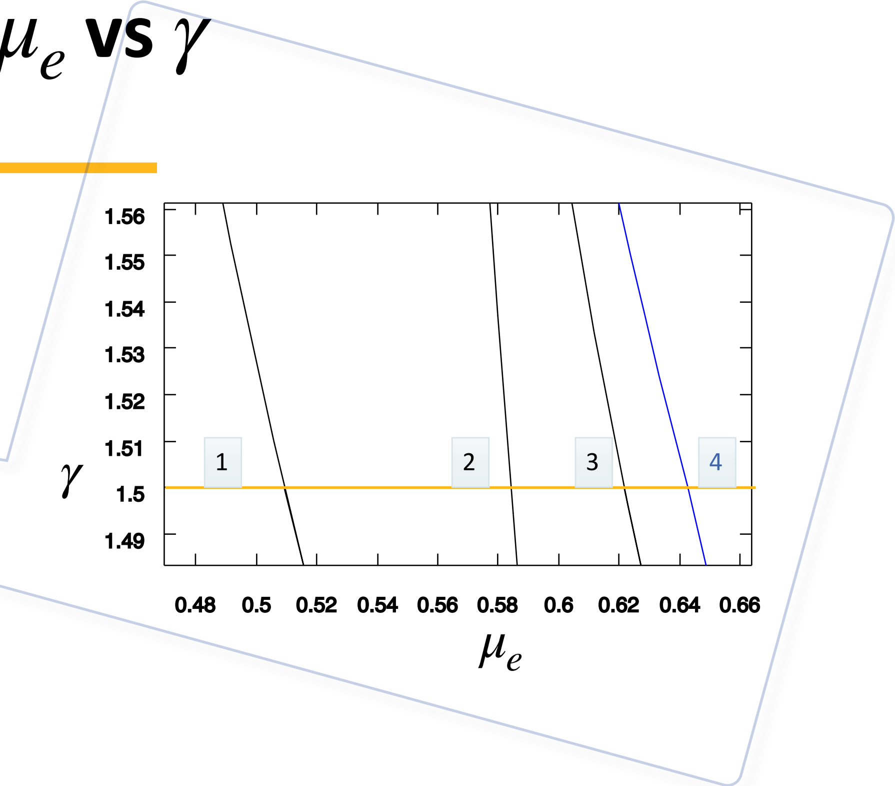
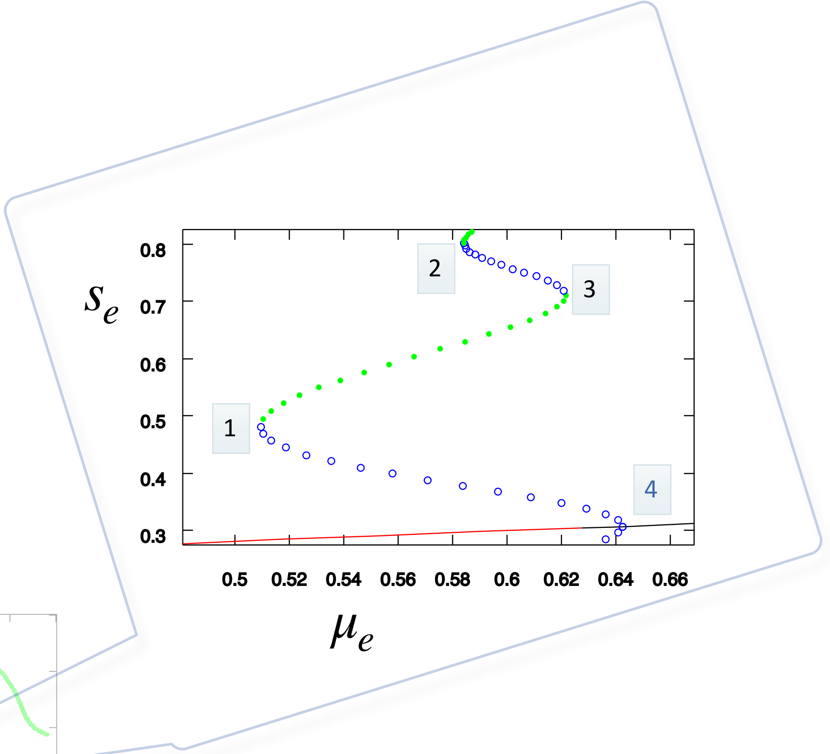
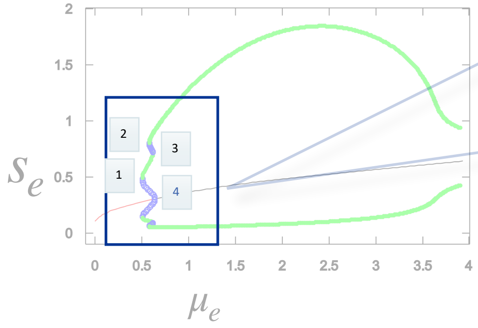
Bifurcation Plots μ_e vs γ

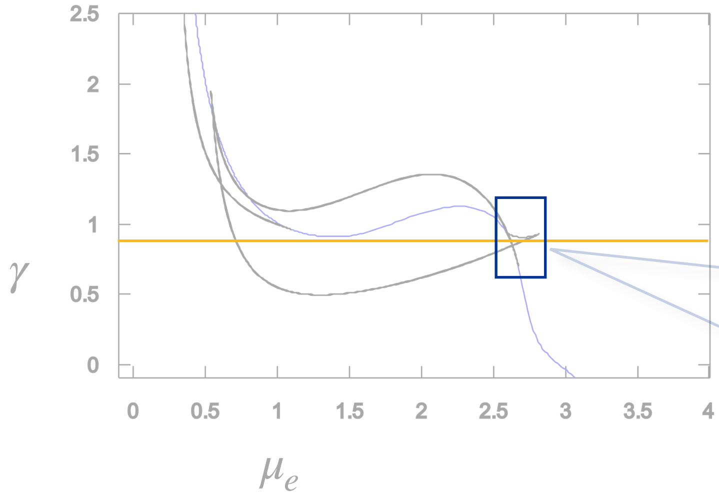


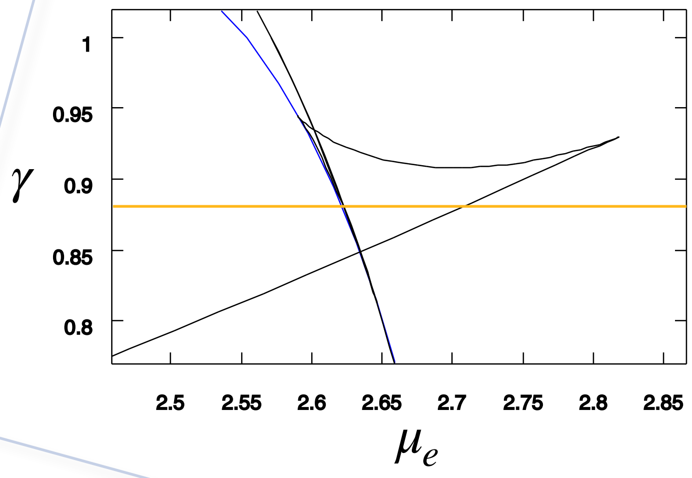




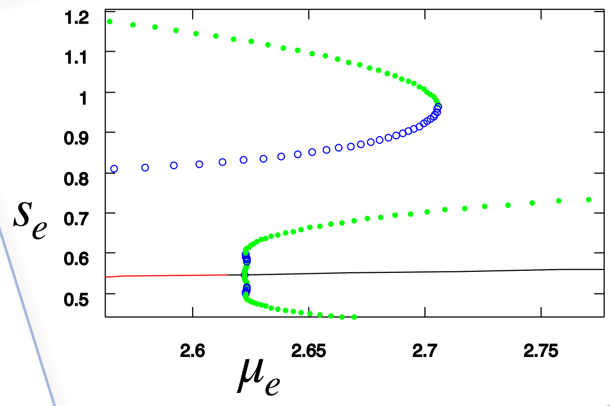


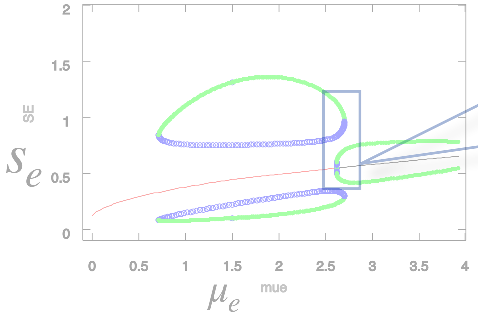
Bifurcation Plots μ_e vs γ





Trirhythmicity!!





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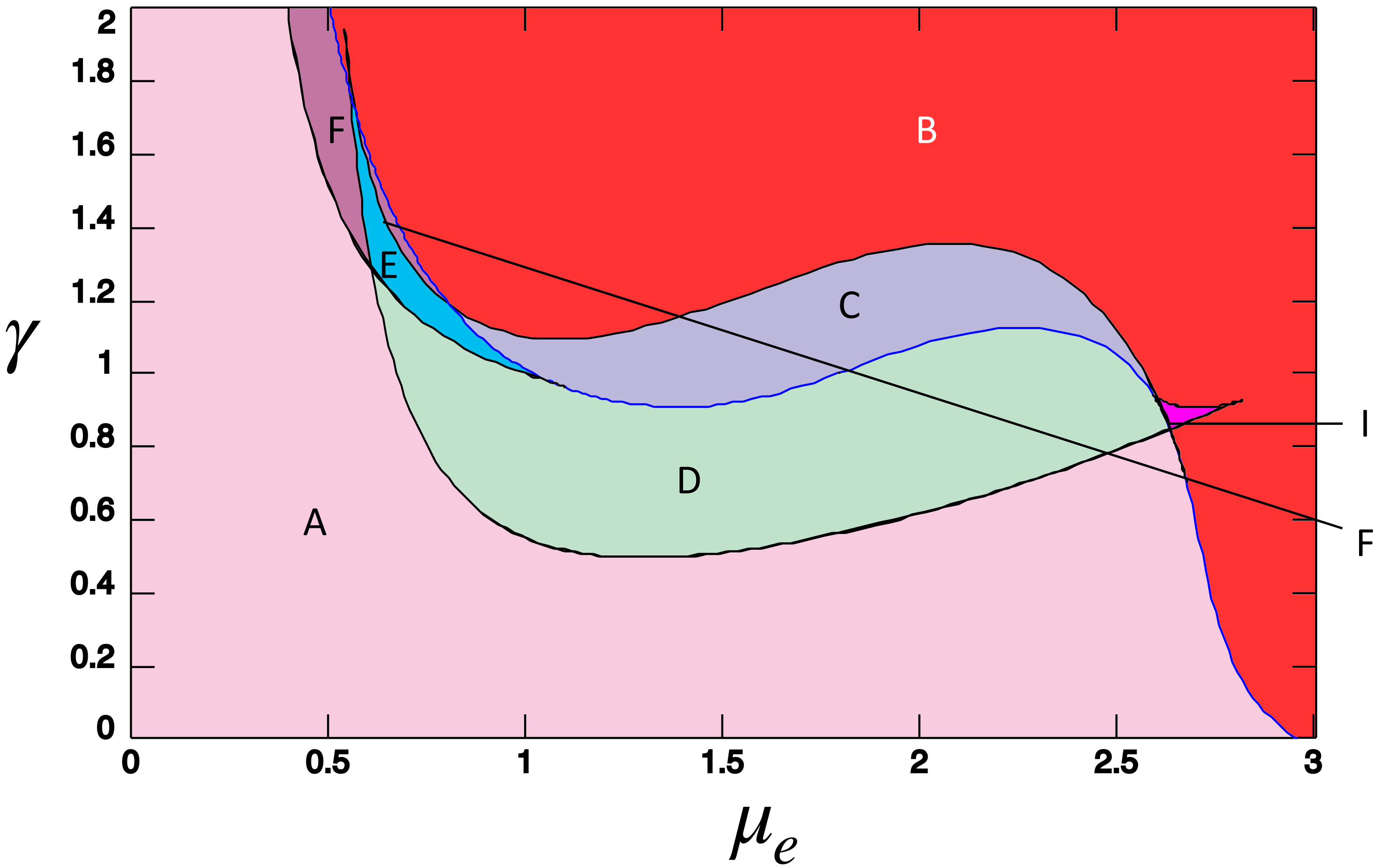
Regions in 2 par bifurcation



A: Stable fixed point, no limit cycle

B: Unstable fixed point, one stable limit cycle(bigger)

C: Unstable fixed point, Birhythmicity

D: Stable fixed point, One stable limit cycle

E: Stable fixed point, Birhythmicity

F: Stable fixed point, One stable limit cycle(bigger)

I: Unstable fixed point, Trirhythmicity

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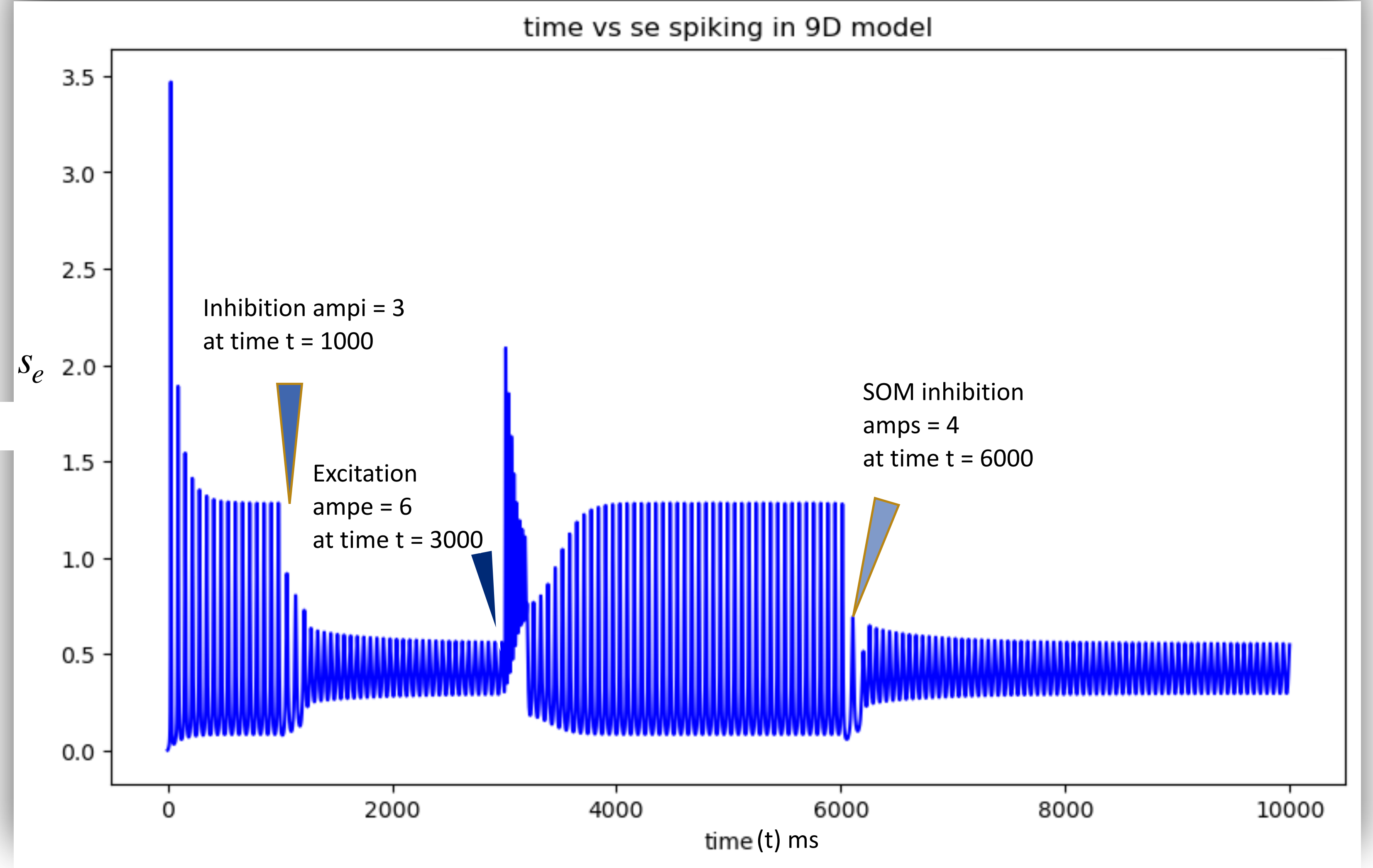
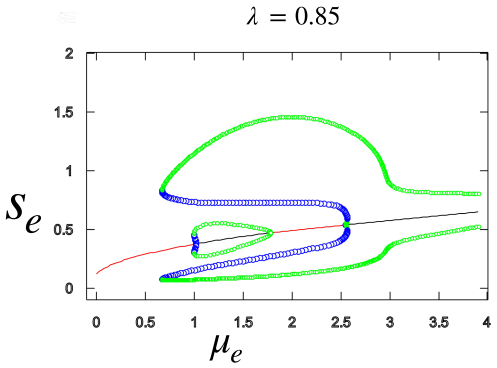
Spiking Plot





Everywhere the input current was effected for 100 msec

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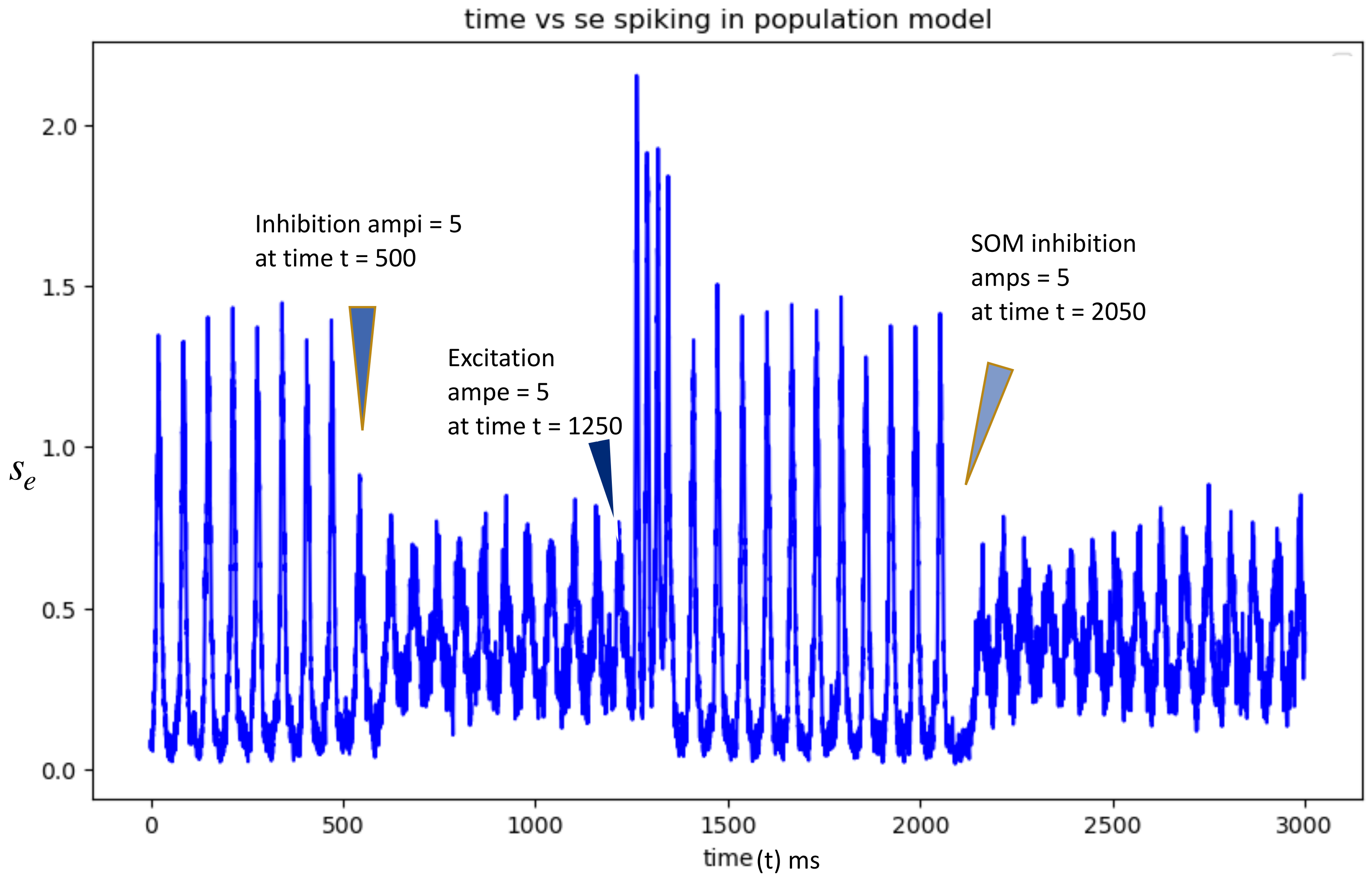
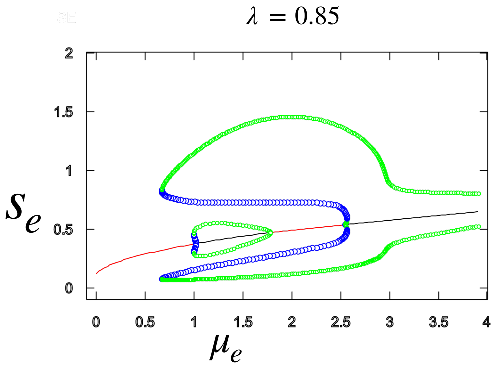
Spiking Plot





It is a spiking model consisting
400 neurons for each population and
Everywhere the input current
was effected for 100 msec

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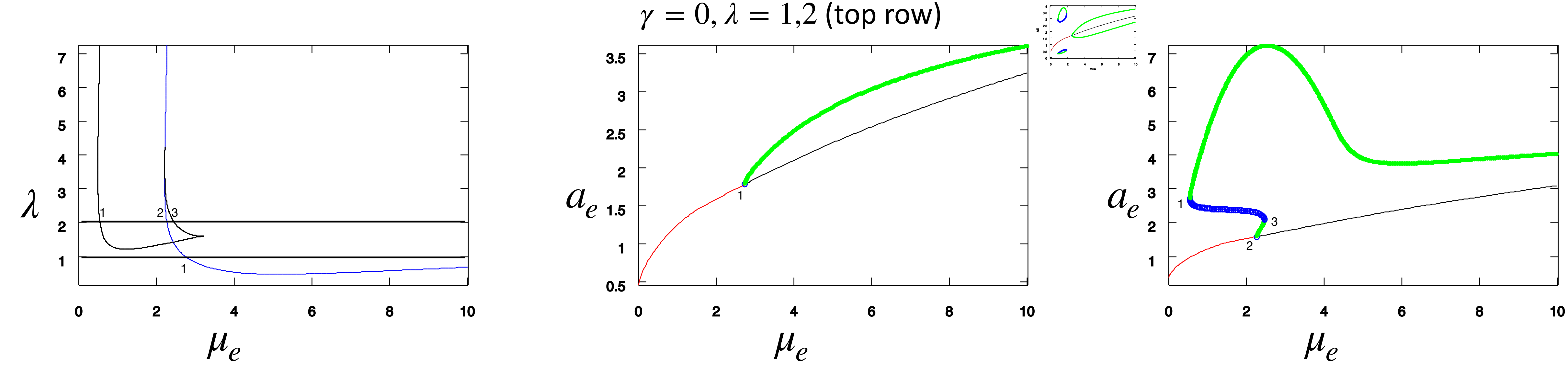
Part 2

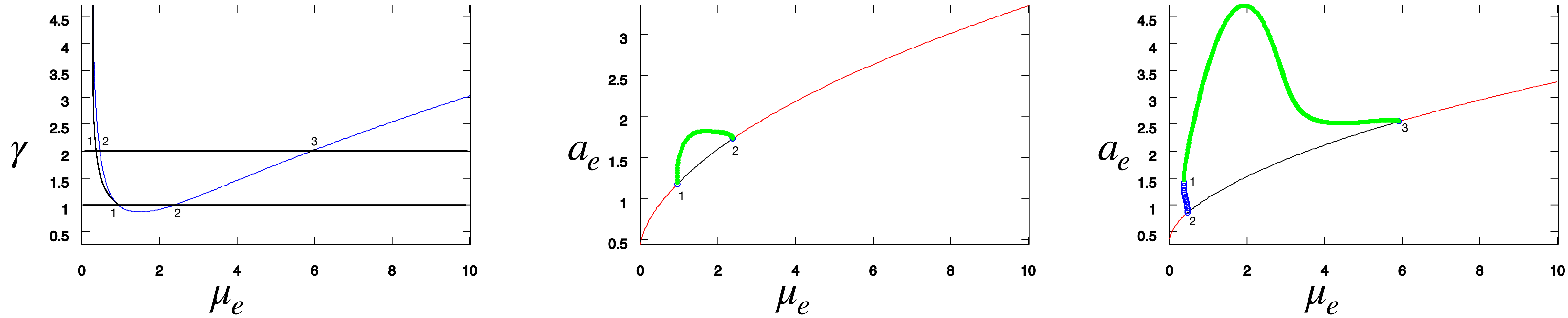
Question

What will happen if we shut down one input of inhibition μ_e or μ_s?

$\gamma = 0, \lambda = 1{,}2$ (top row)

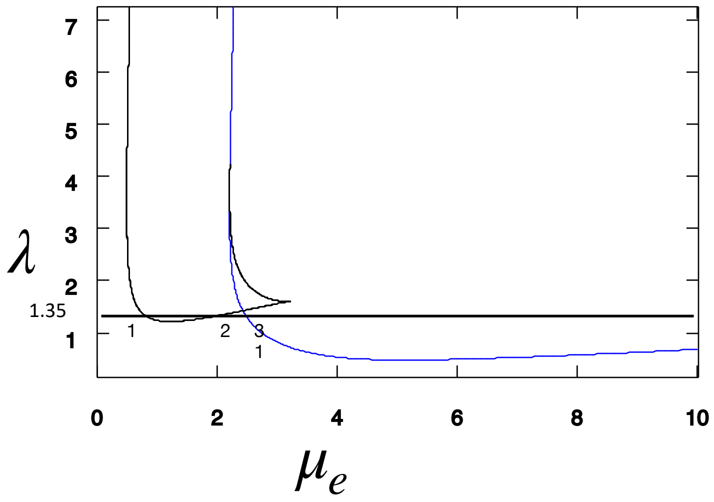


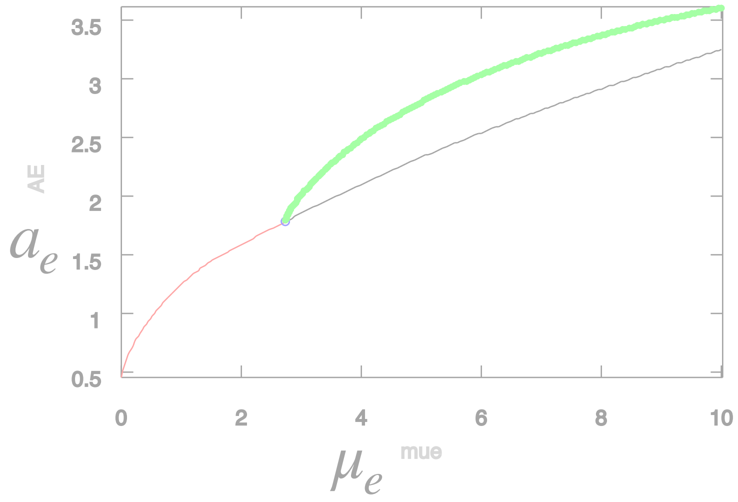
$\lambda = 0, \gamma = 1{,}2$ (bottom row)

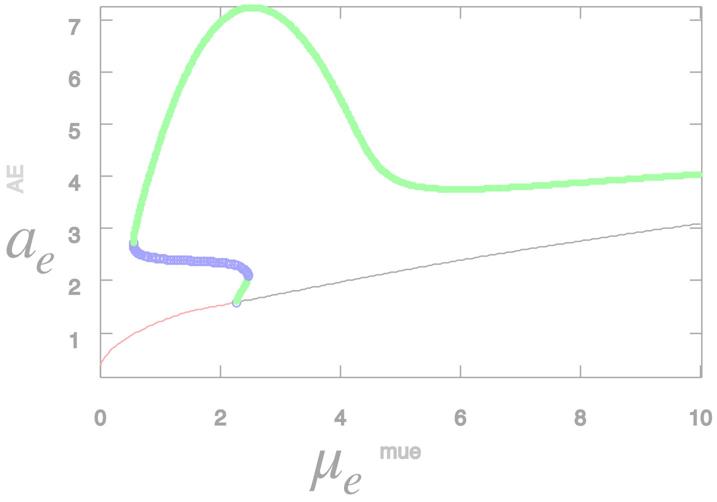


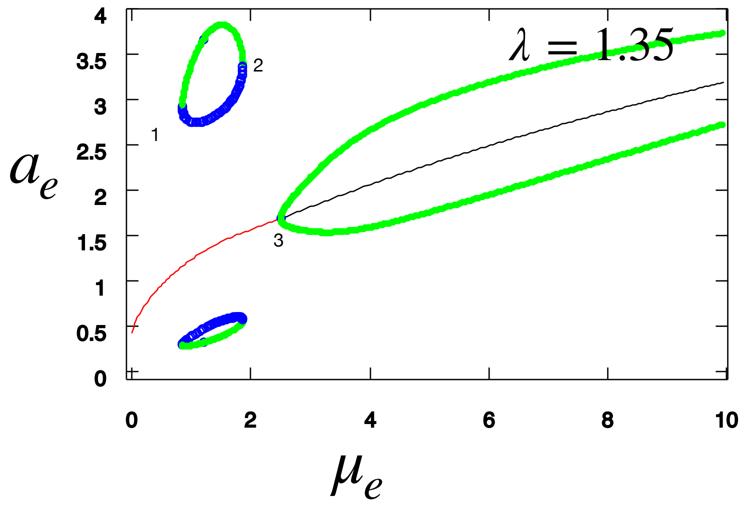
2par bifurcation for the 9D model, γ vs μ_e for $\gamma = 0, \lambda = 1{,}2$ (top row) and bifurcation for $\lambda = 0, \gamma = 1{,}2$ (bottom row). This lets us see what happens without one type of inhibition

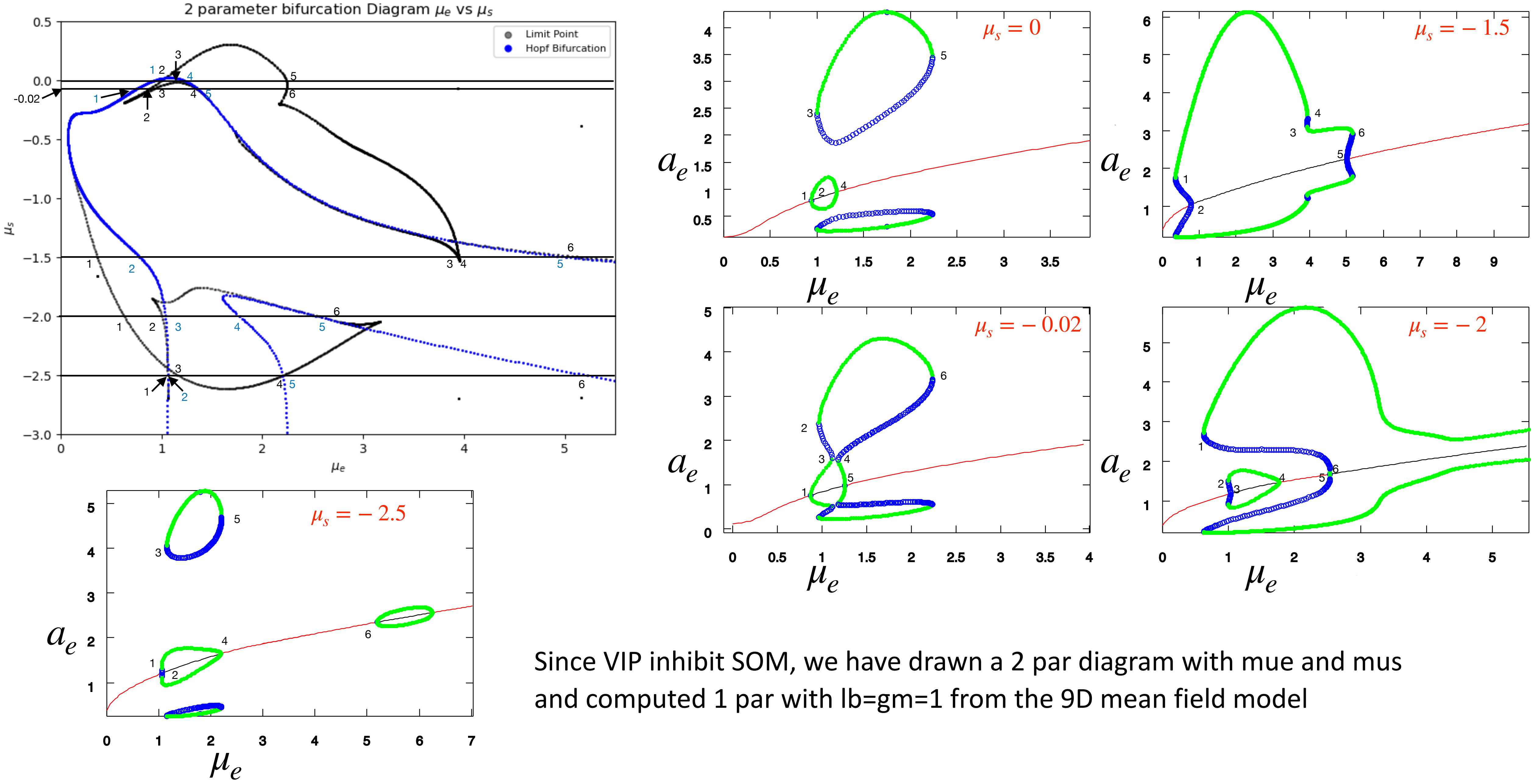
$\gamma = 0, \lambda = 1{,}2$ (top row)



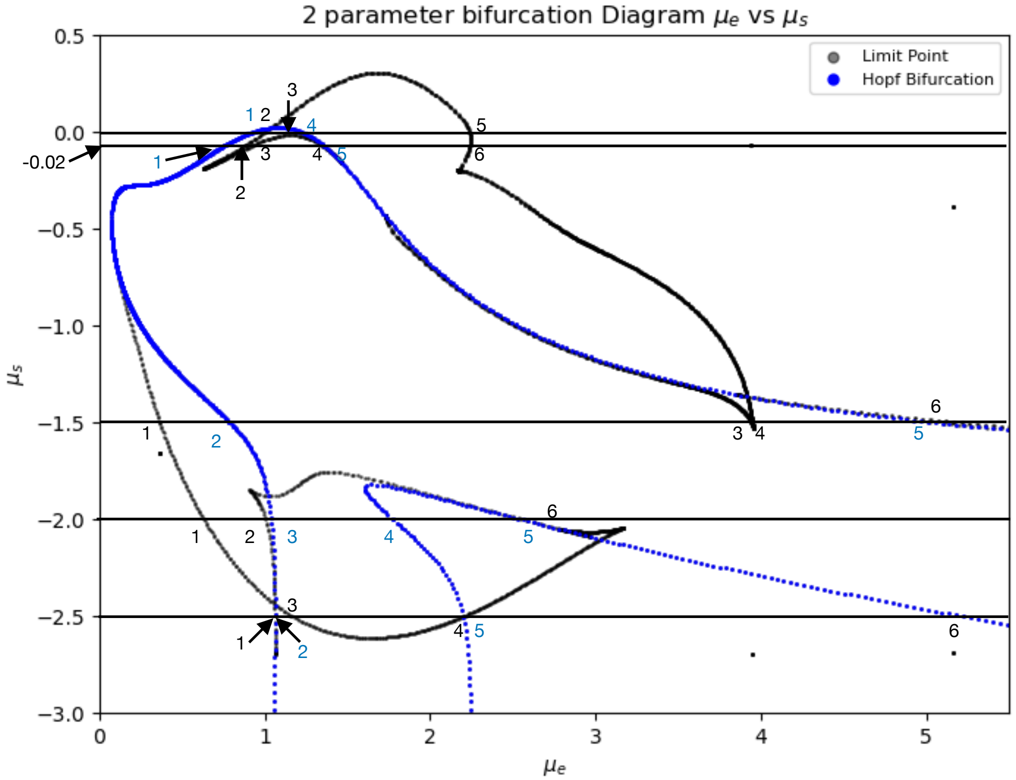


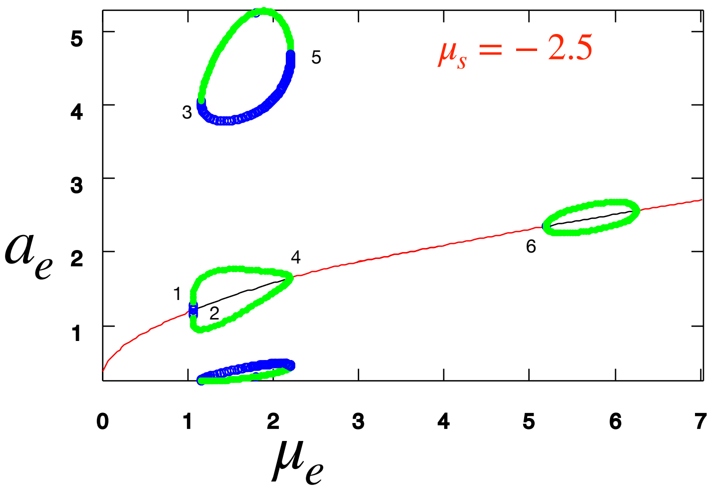






Since VIP inhibit SOM, we have drawn a 2 par diagram with mue and mus and computed 1 par with lb=gm=1 from the 9D mean field model





$$\mu_s = \mu_{s_0} - \mu_{s_{\text{VIP}}}$$

$$\mu_{s_{\text{VIP}}} > 0$$

Qn: Can we reduce the 9D model to 3D?

- Do we need all 9 populations to get birhythmicity?
- Can we consider a limiting case when the membrane time constant go to zero or the synaptic time constant go to infinity?

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The 3D model

Remember that we had,

$$\tau_m a_t = 2ab + \Delta,$$
$$\tau_m b_t = b^2 - a^2 + \mu + gS,$$
$$\tau_s S_t = -S + \frac{a}{\pi}$$

Now consider $\tau_m = 1$ and make a change in time scale $\tau = \epsilon t$ where $\epsilon = \dfrac{1}{\tau_s}$; we get

$$\epsilon a_\tau = 2ab + \Delta,$$
$$\epsilon b_\tau = b^2 - a^2 + \mu + gS$$
$$S_\tau = -S + \frac{a}{\pi}$$

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The 3D model

Now take $\tau_s \to \infty$ or $\epsilon \to 0$, we get

$$2ab + \Delta = 0,$$
$$b^2 - a^2 + \mu + gS = 0$$

Substituting b in terms of a and solving for a we get, $a = \sqrt{0.5(\eta + \sqrt{\eta^2 + \Delta^2})} := f(\eta)$, where $\eta = \mu + gS$.

Hence, we have

$$\frac{ds}{d\tau} = -s + \frac{f(\eta)}{\pi}.$$

Now, we can consider 3 populations E, I and S version of it.

The 3D model

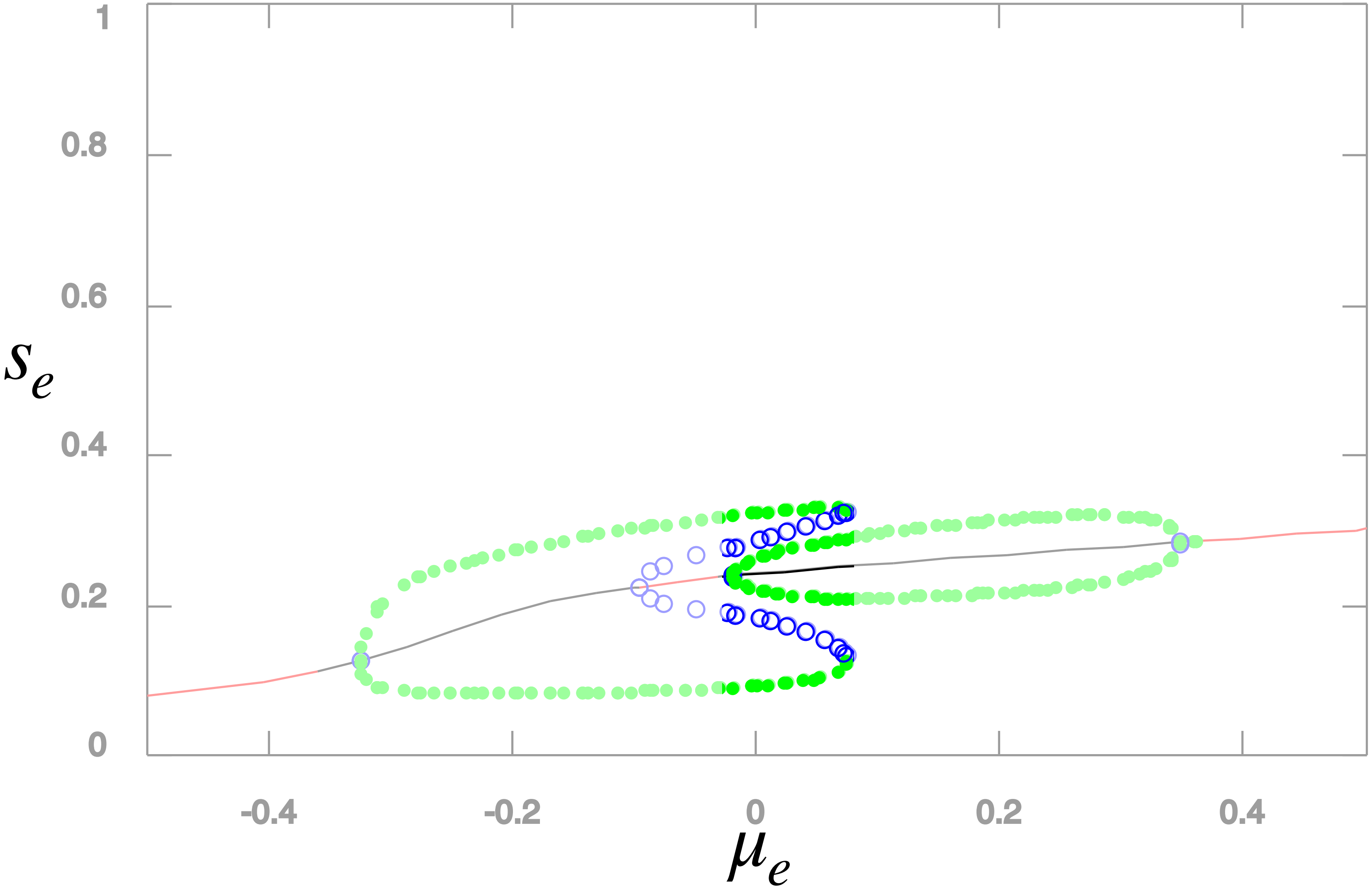
$$s_e' = \frac{-s_e + f(g_{ee}s_e - \gamma g_{ie}s_i - \lambda g_{se}s_s + \mu_e)}{\tau_e}$$

$$s_i' = \frac{-s_i + f(g_{ei}s_e - g_{ii}\gamma s_i - \lambda g_{si}s_s + \mu_i)}{\tau_i} \quad \text{where, } f(x) = \frac{\sqrt{.5(x + \sqrt{x^2 + d})}}{\pi}$$

$$s_s' = \frac{-s_s + f(g_{es}s_e + \mu_s)}{\tau_s}$$

- This is a simple fire rate model. We need to understand the range of parameters where we will see birhythmicity.

3 dimensional model



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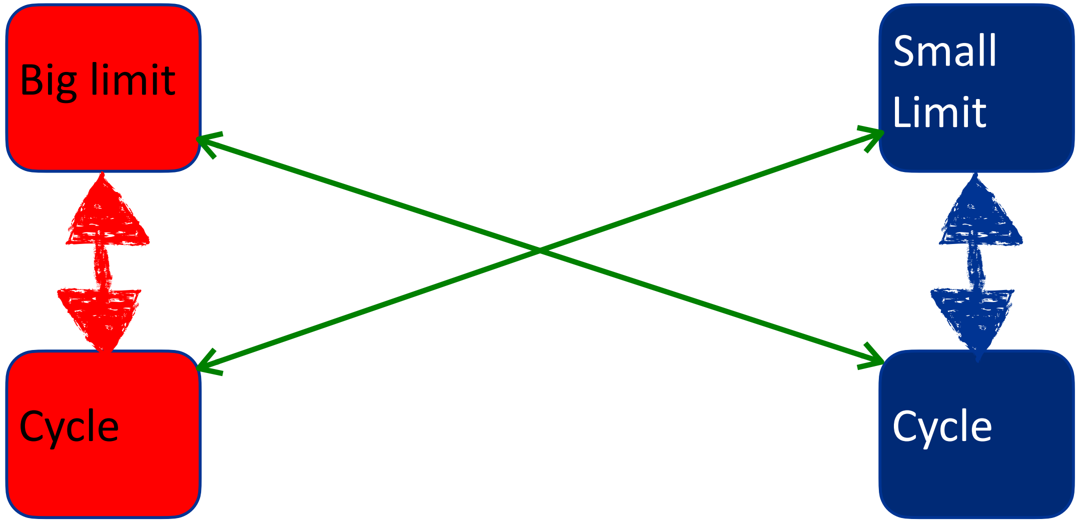
Qn: How about mixing population

Now we have seen that there are regions where we see birhythmicity. Can we have 9 populations starting from the bigger limit cycle and other 9 population starting from the lower limit cycle and mix them up?



Simulation(Full 3 population model)

$$a_l[j]' = \frac{1}{\tau_{ml}}(2a_l[j]b_l[j] + \delta_l)$$

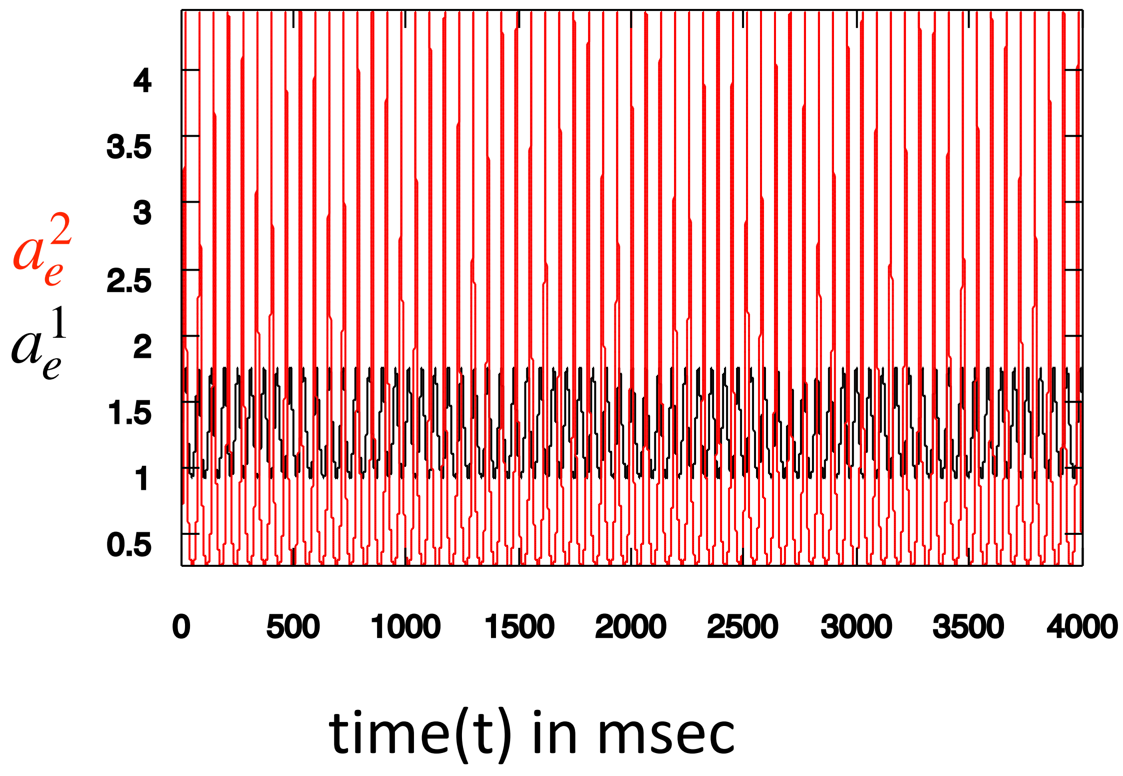
$$b_l[j]' = \frac{1}{\tau_{ml}}(b_l[j]^2 - a_l[j]^2 + g_{el}s_{eb}[j] - \gamma g_{il}s_{ib}[j] - \lambda g_{sl}s_{sb}[j] + \mu_l)$$

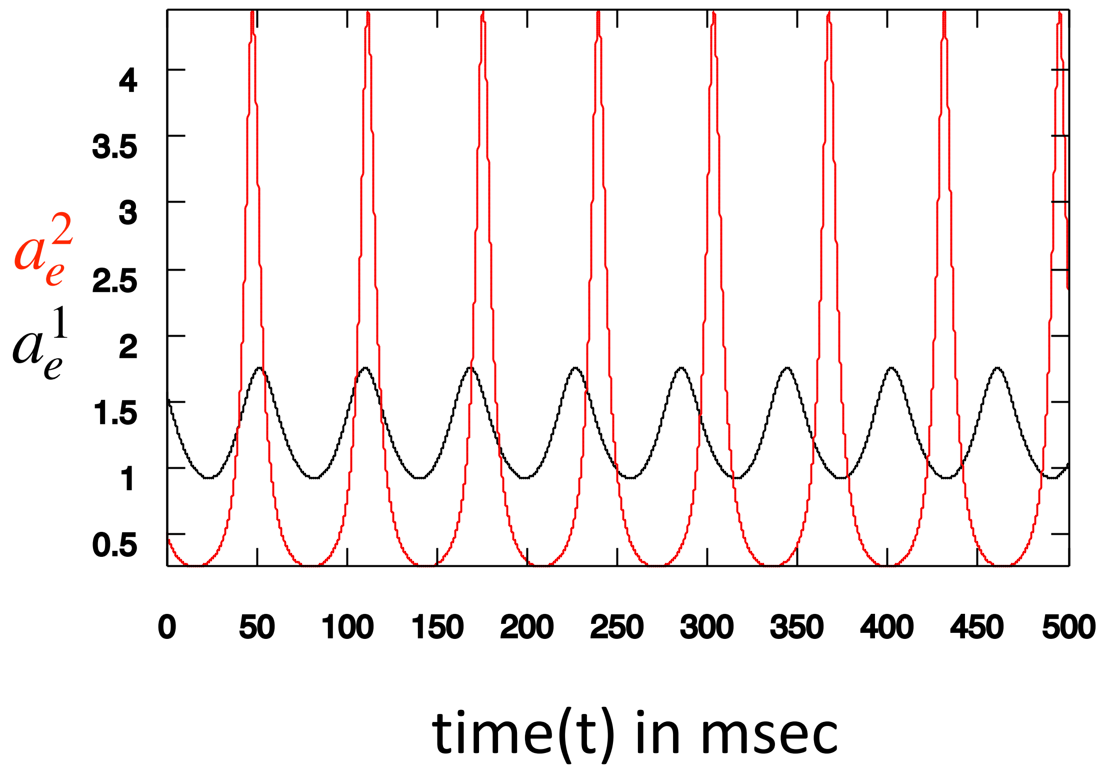
$$s_l[j]' = \frac{1}{\tau_l}\left(-s_l[j] + \frac{a_l[j]}{\pi}\right)$$

where, $\begin{aligned} s_{lb}[1]' &= ps_l[1] + (1-p)s_l[2] \\ s_{lb}[2]' &= ps_l[2] + (1-p)s_l[1] \end{aligned}$ and $k, l \in \{e, i, s\};\ j \in \{1,2\};\ p \in [0,1]$

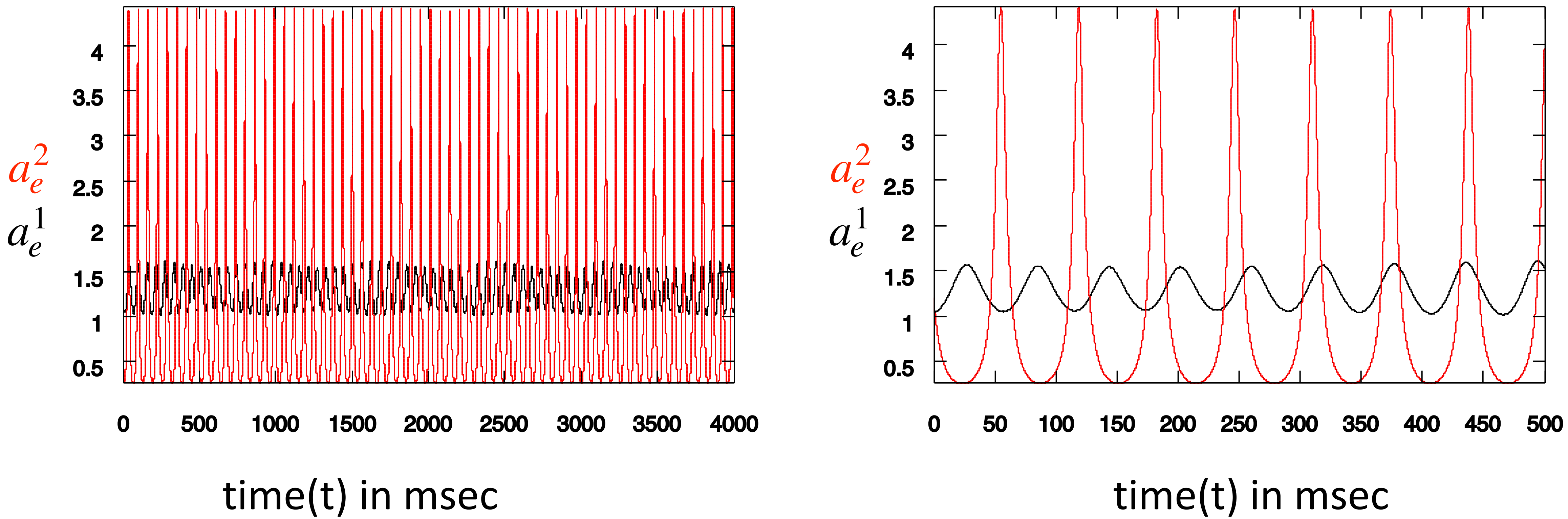
At $p = 1, \mu_e = 1.25$

a_e^2 is big and a_e^1 is small

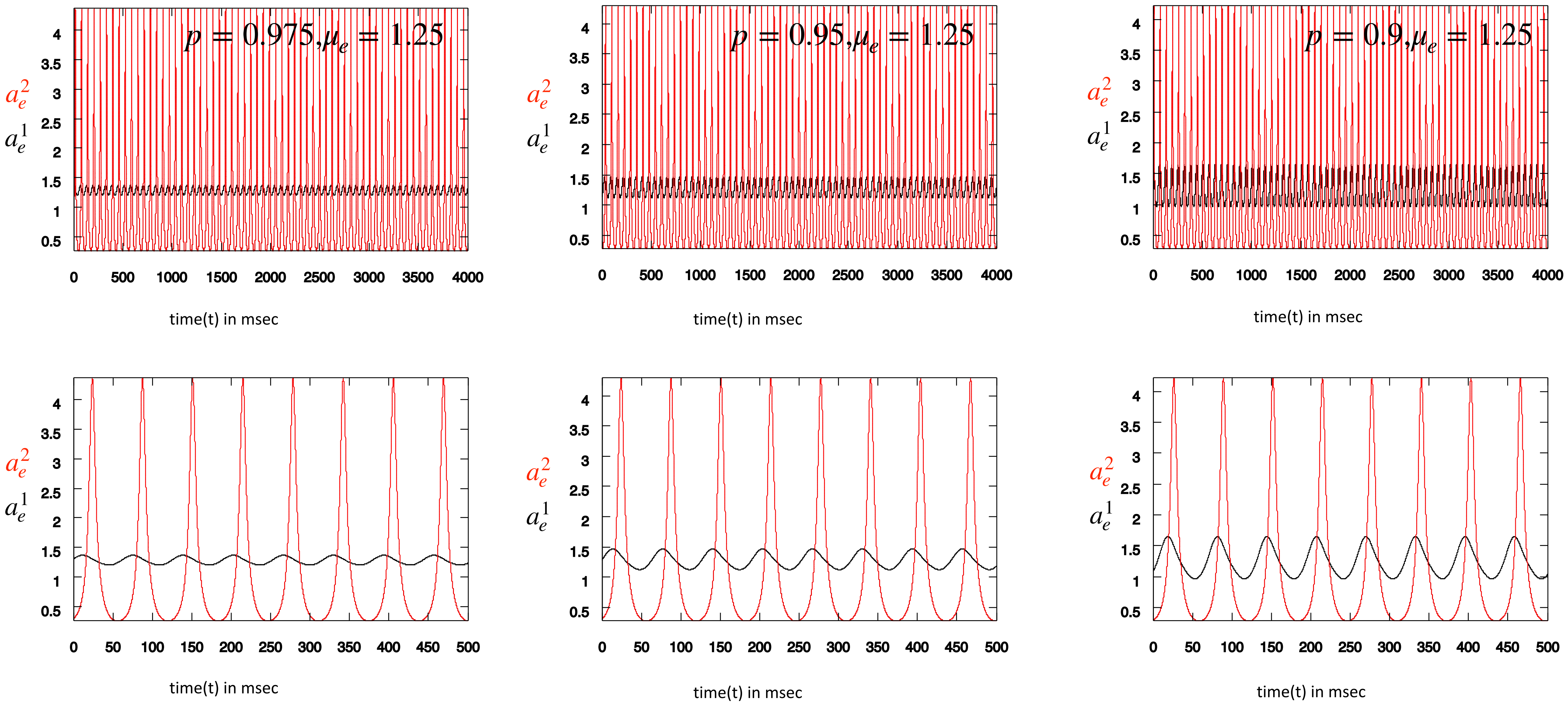




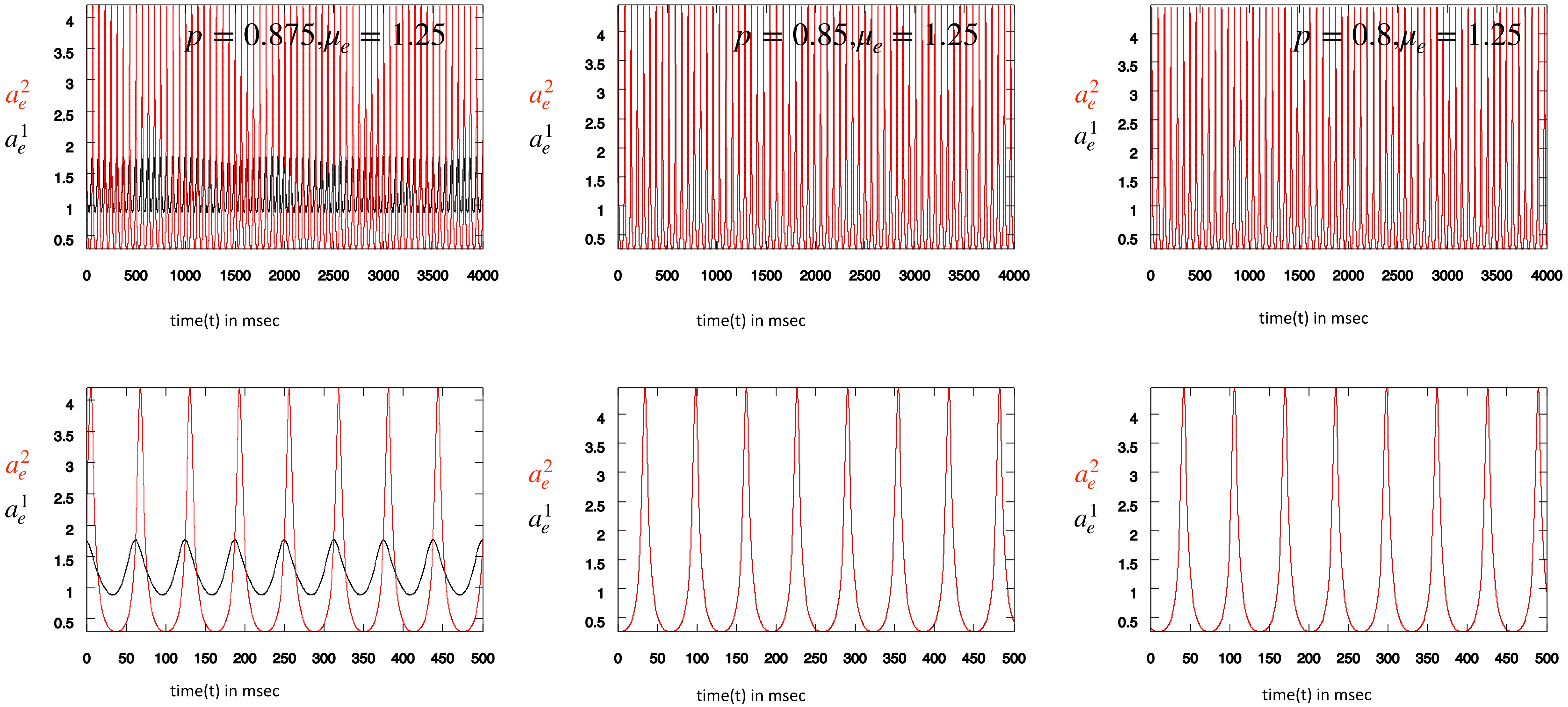
$p = 0.99,\ \mu_e = 1.25$



a_e^2
a_e^1
4
3.5
3
2.5
2
1.5
1
0.5
0
500
1000
1500
2000
2500
3000
3500
4000
time(t) in msec
a_e^2
a_e^1
4
3.5
3
2.5
2
1.5
1
0.5
0
50
100
150
200
250
300
350
400
450
500
time(t) in msec

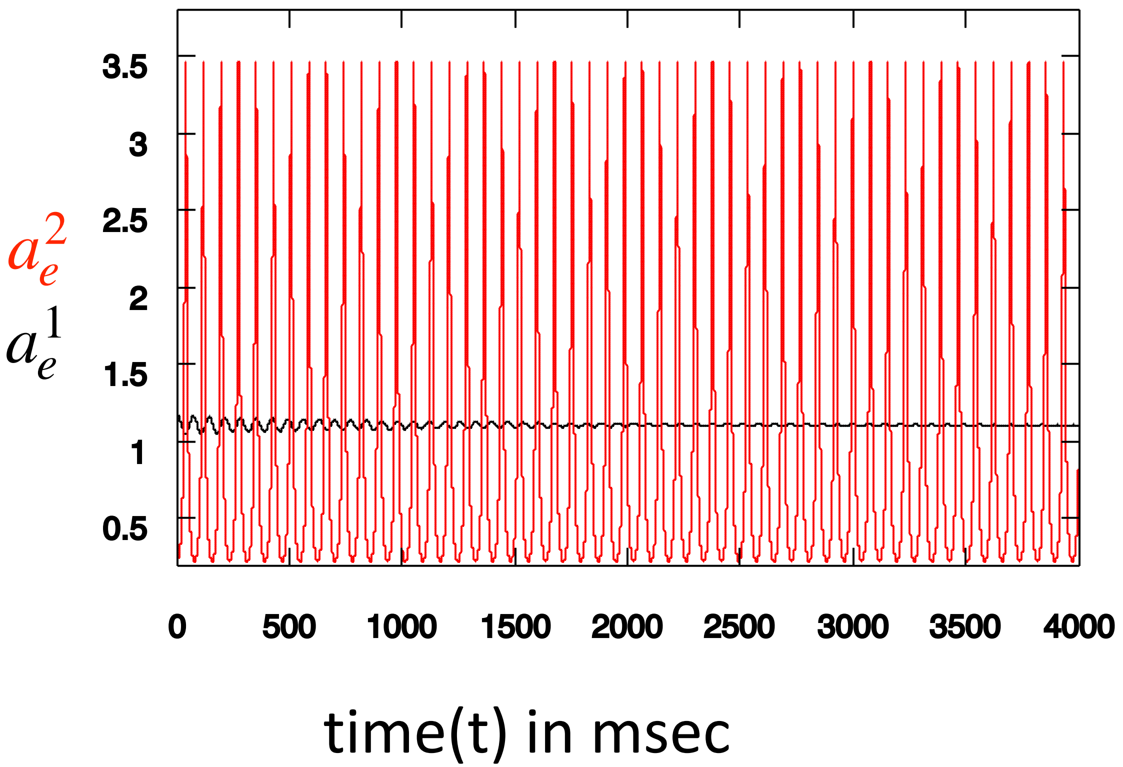


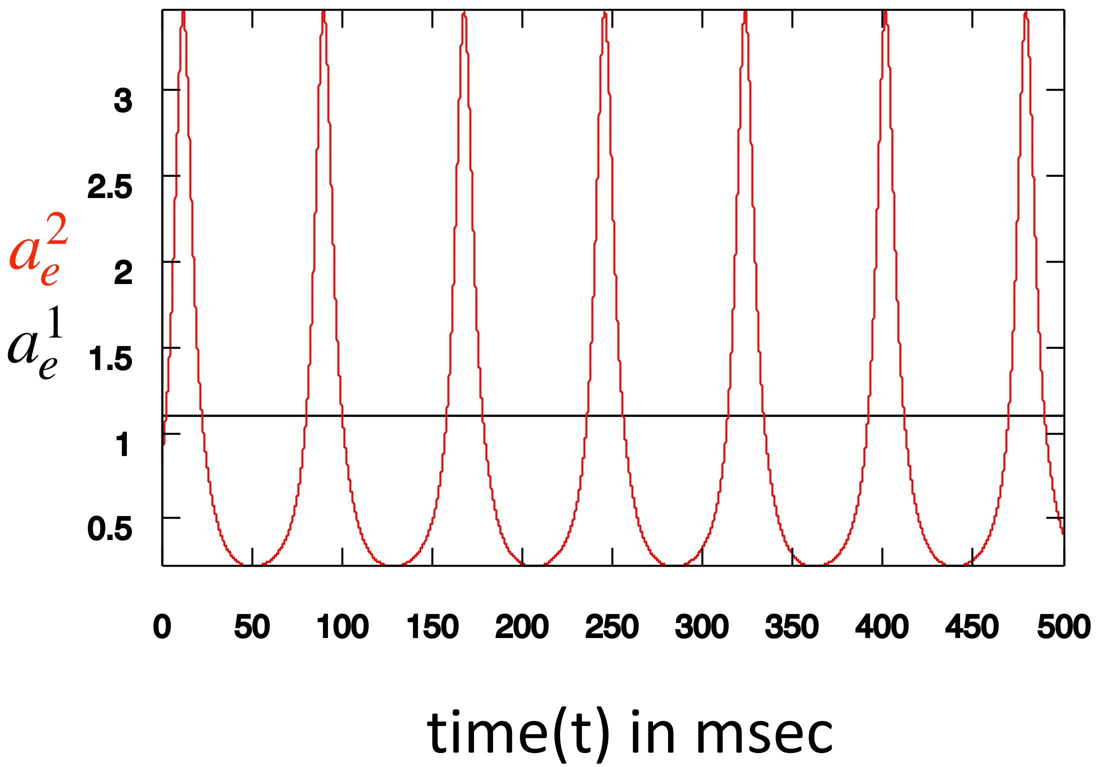
$p = 0.975, \mu_e = 1.25$
$p = 0.95, \mu_e = 1.25$
$p = 0.9, \mu_e = 1.25$
a_e^2
a_e^1
time(t) in msec

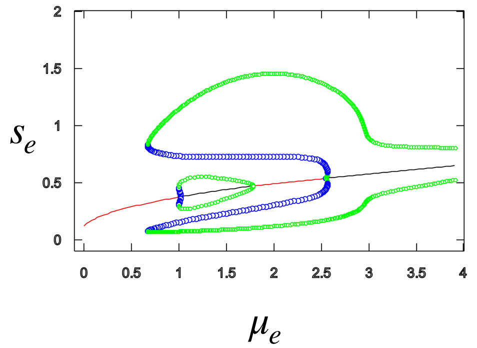


$p = 0.875, \mu_e = 1.25$
$p = 0.85, \mu_e = 1.25$
$p = 0.8, \mu_e = 1.25$
a_e^2
a_e^1
time(t) in msec

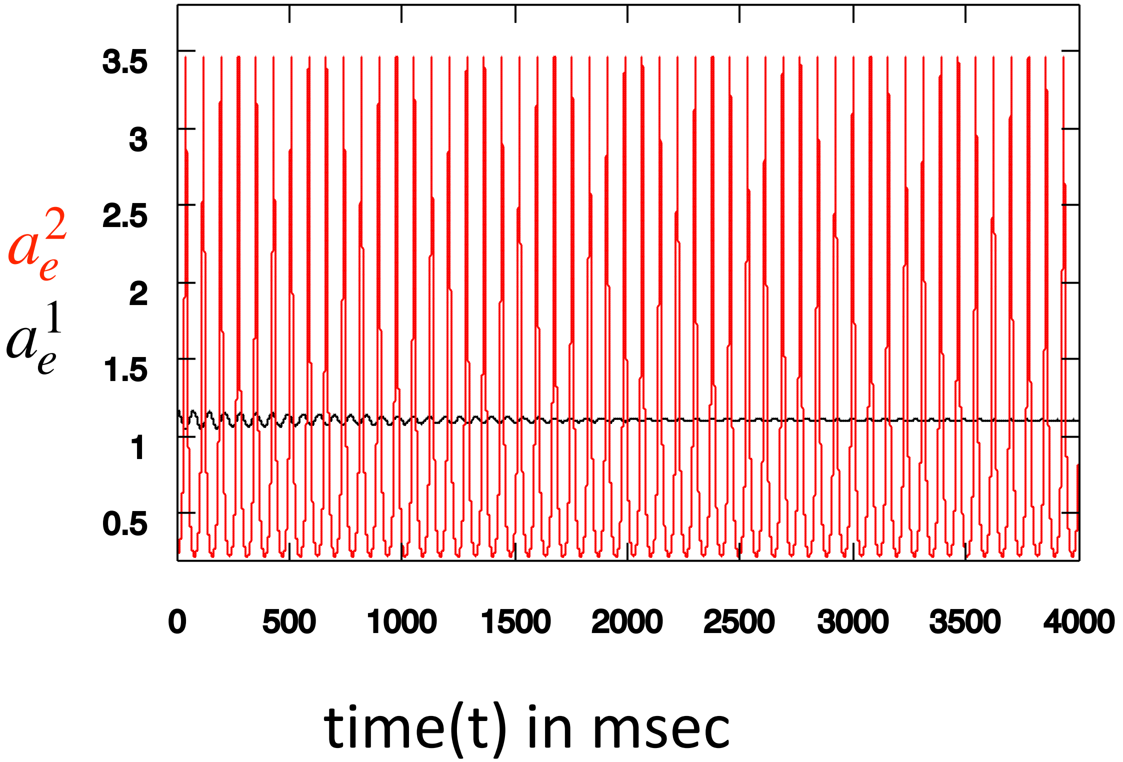
$$p = 1,\ \mu_e = 0.85$$

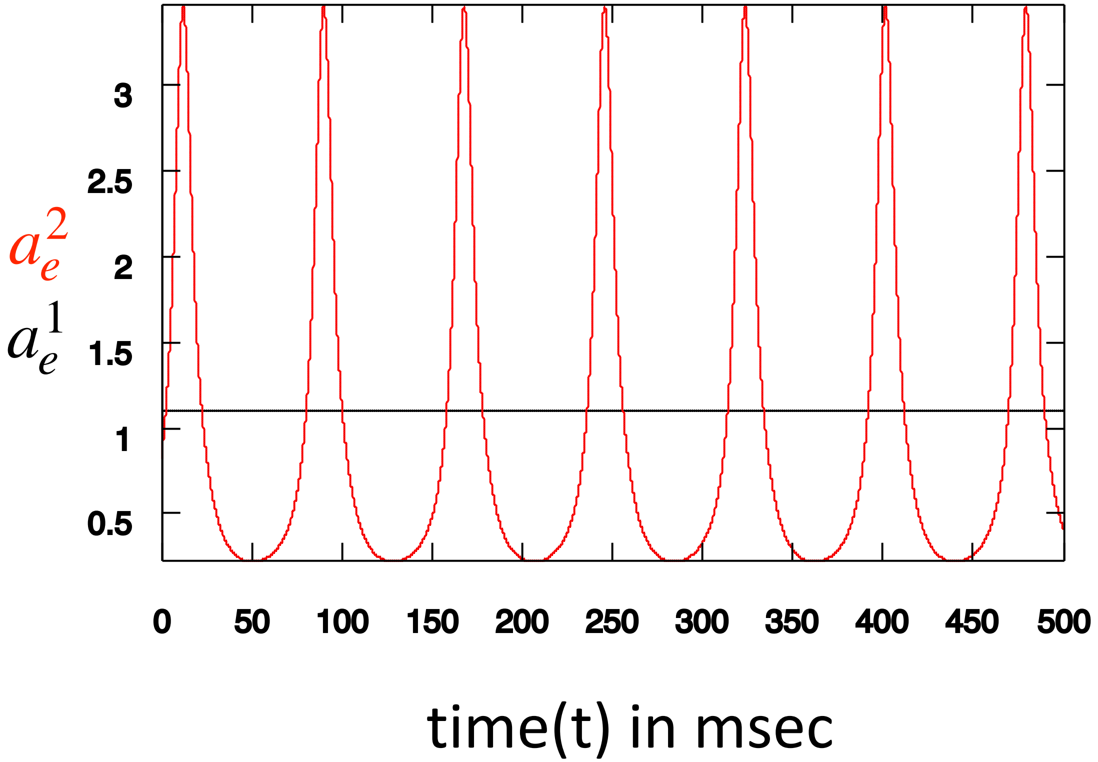

a_e^2
a_e^1
3.5
3
2.5
2
1.5
1
0.5
0
500
1000
1500
2000
2500
3000
3500
4000
time(t) in msec

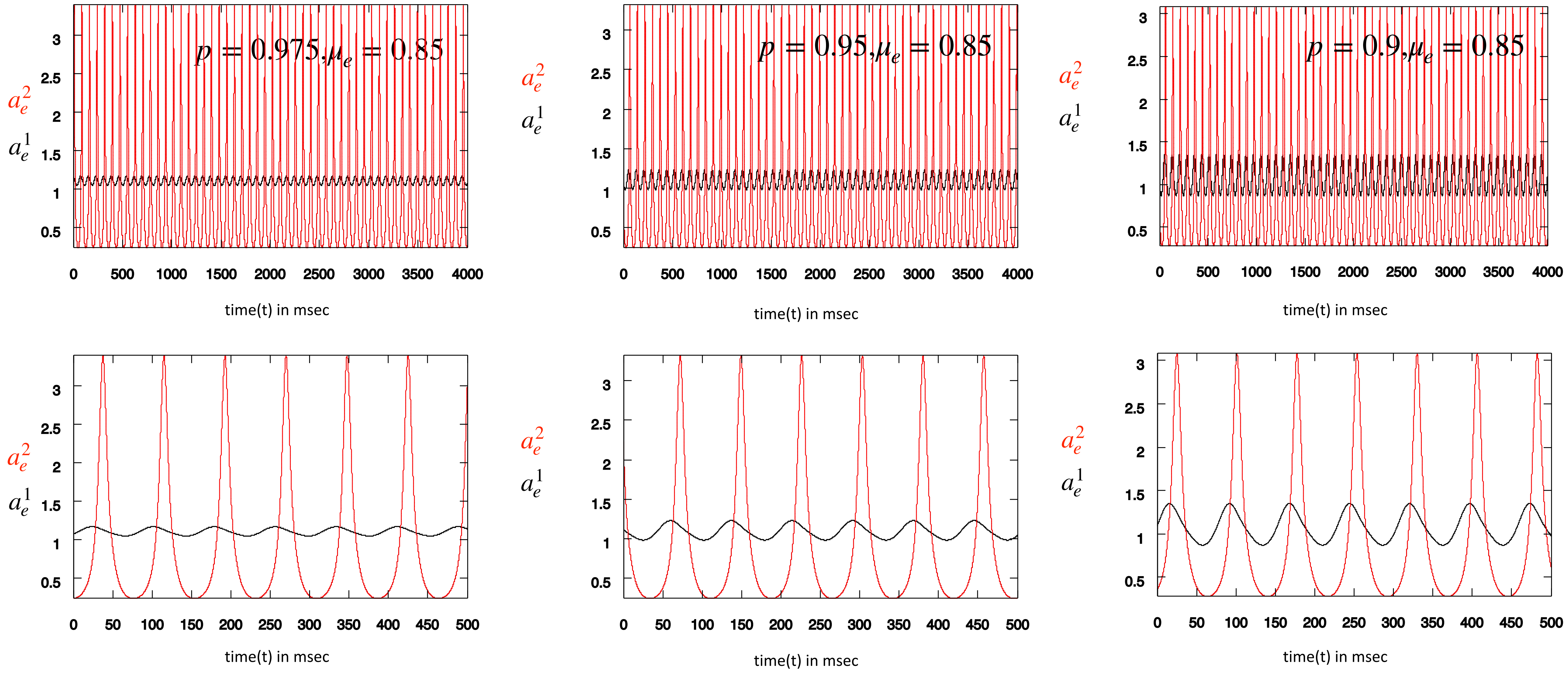

a_e^2
a_e^1
3
2.5
2
1.5
1
0.5
0
50
100
150
200
250
300
350
400
450
500
time(t) in msec



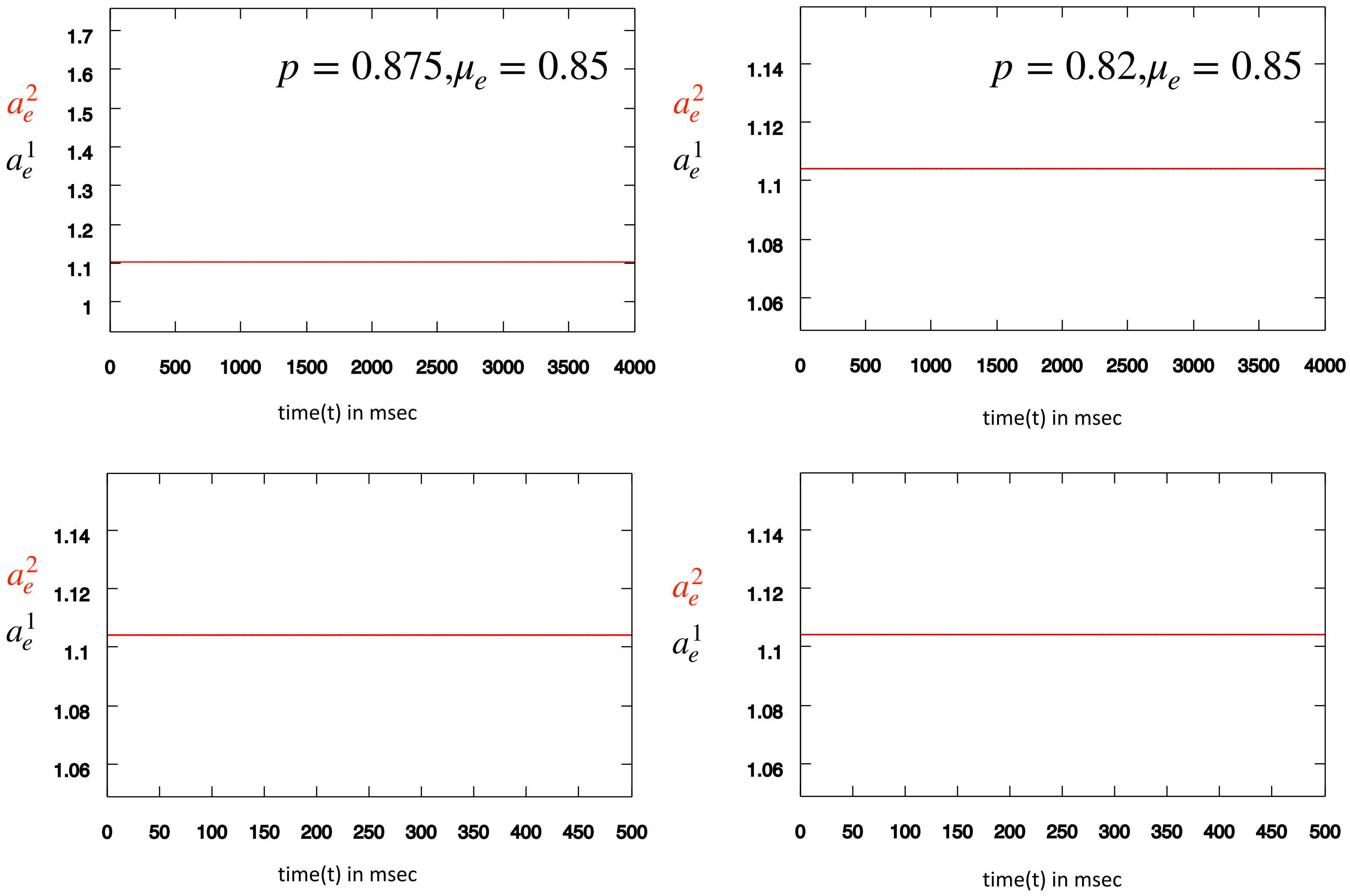
$$p = 1,\ \mu_e = 0.85$$



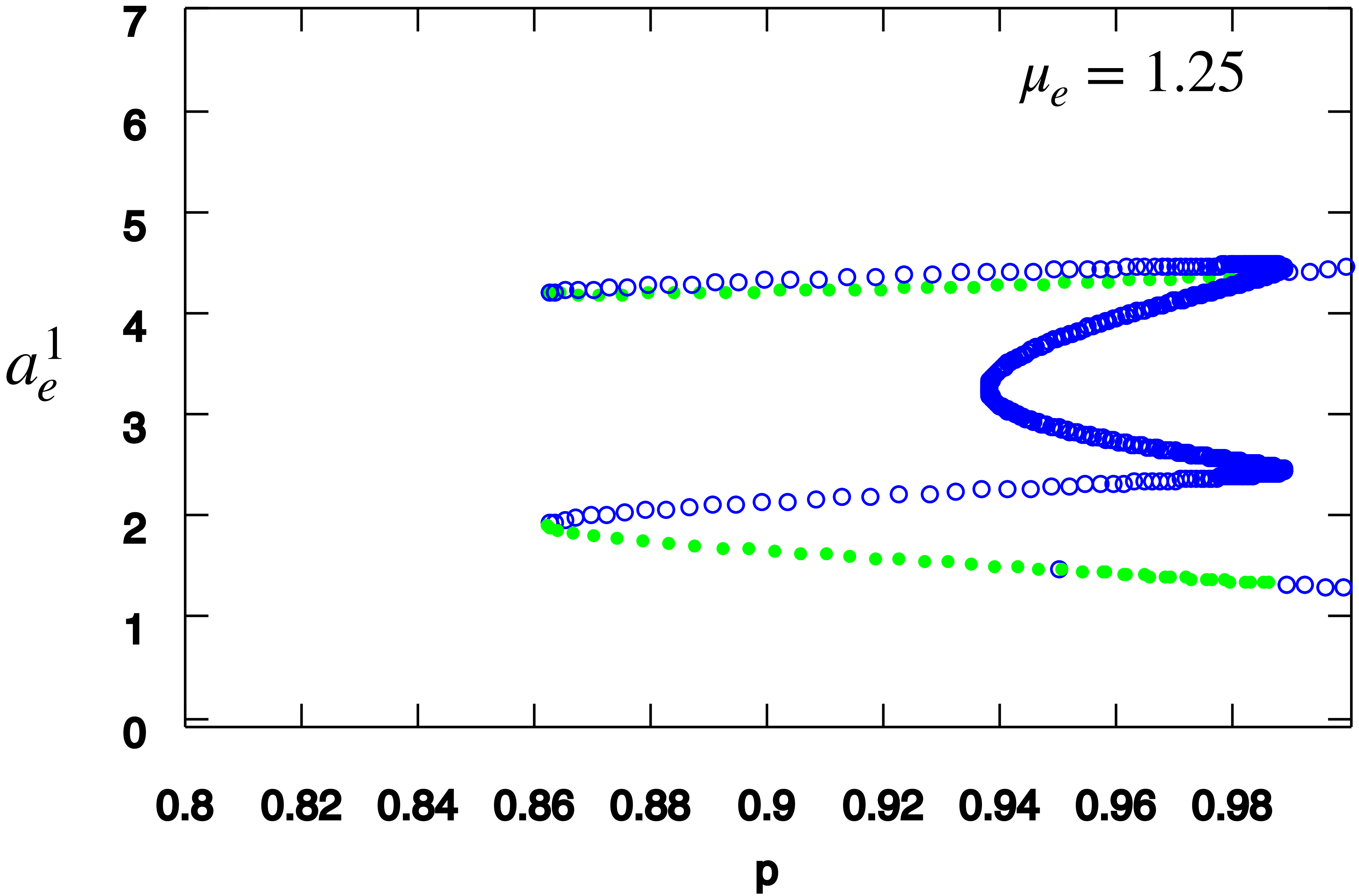




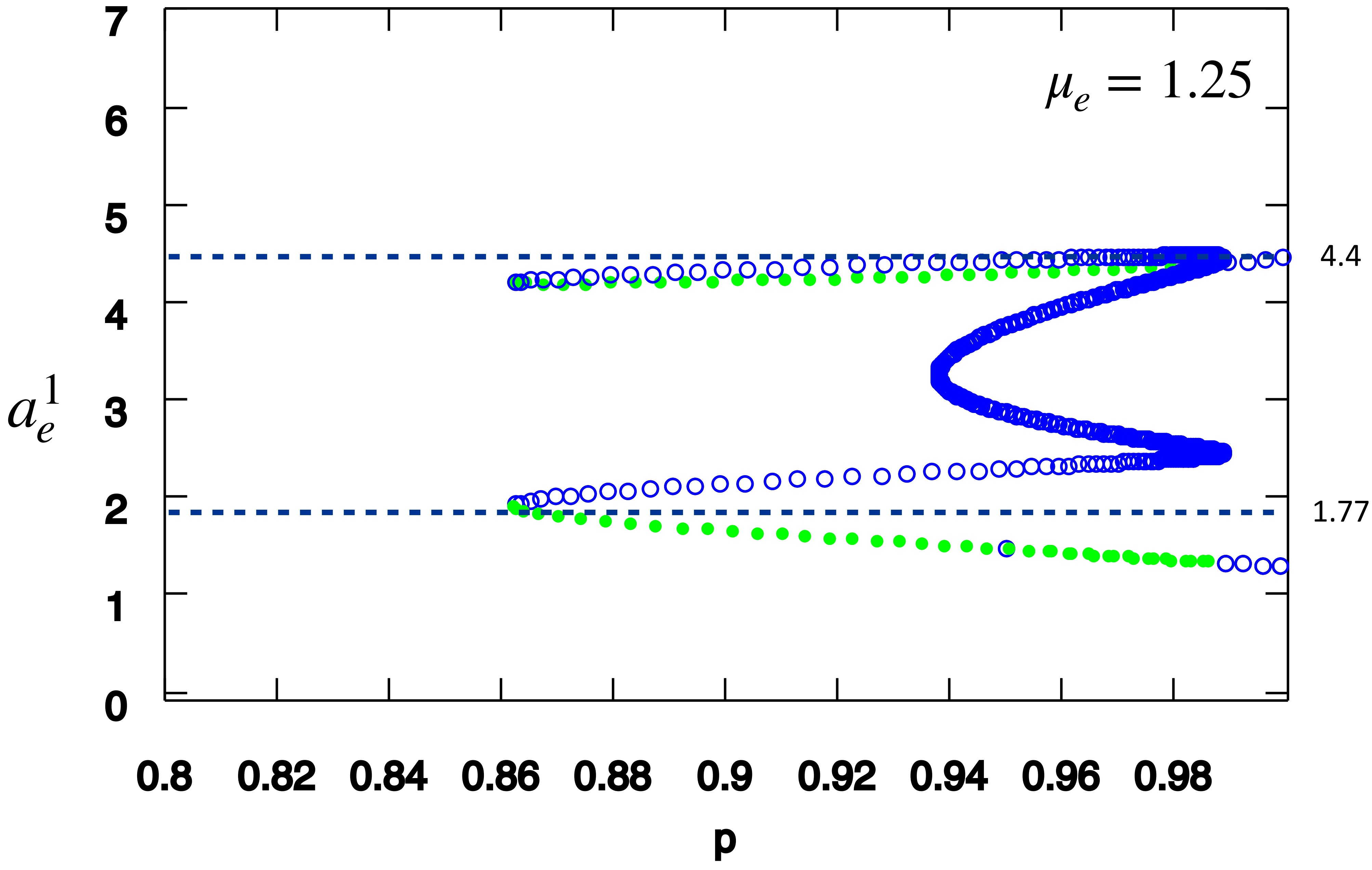
$p = 0.975, \mu_e = 0.85$
$p = 0.95, \mu_e = 0.85$
$p = 0.9, \mu_e = 0.85$
a_e^2
a_e^1
time(t) in msec



It seems when we take p = 0.89 it goes to the fixed point

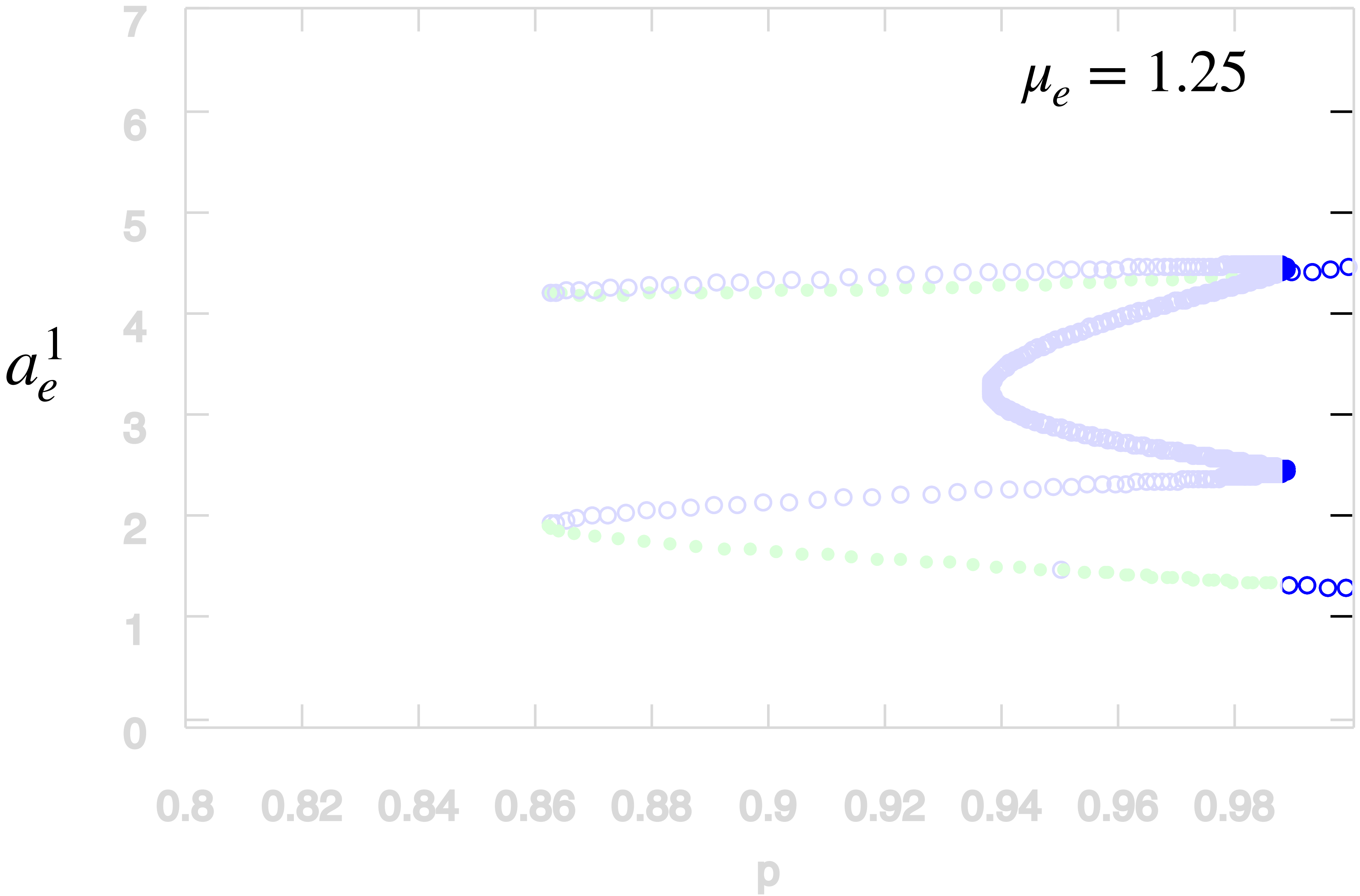


$\mu_e = 1.25$
a_e^1
p
0
1
2
3
4
5
6
7
0.8
0.82
0.84
0.86
0.88
0.9
0.92
0.94
0.96
0.98

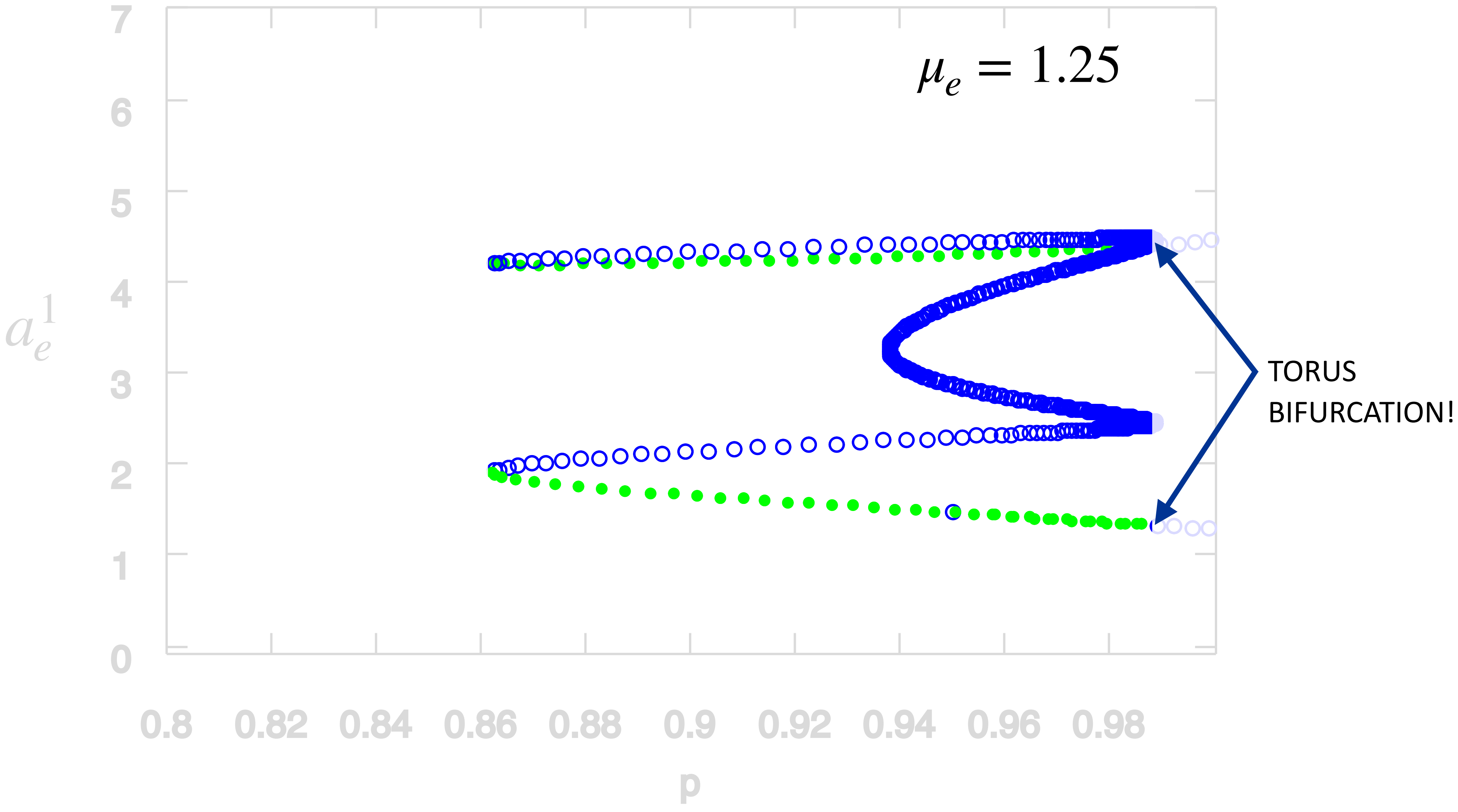


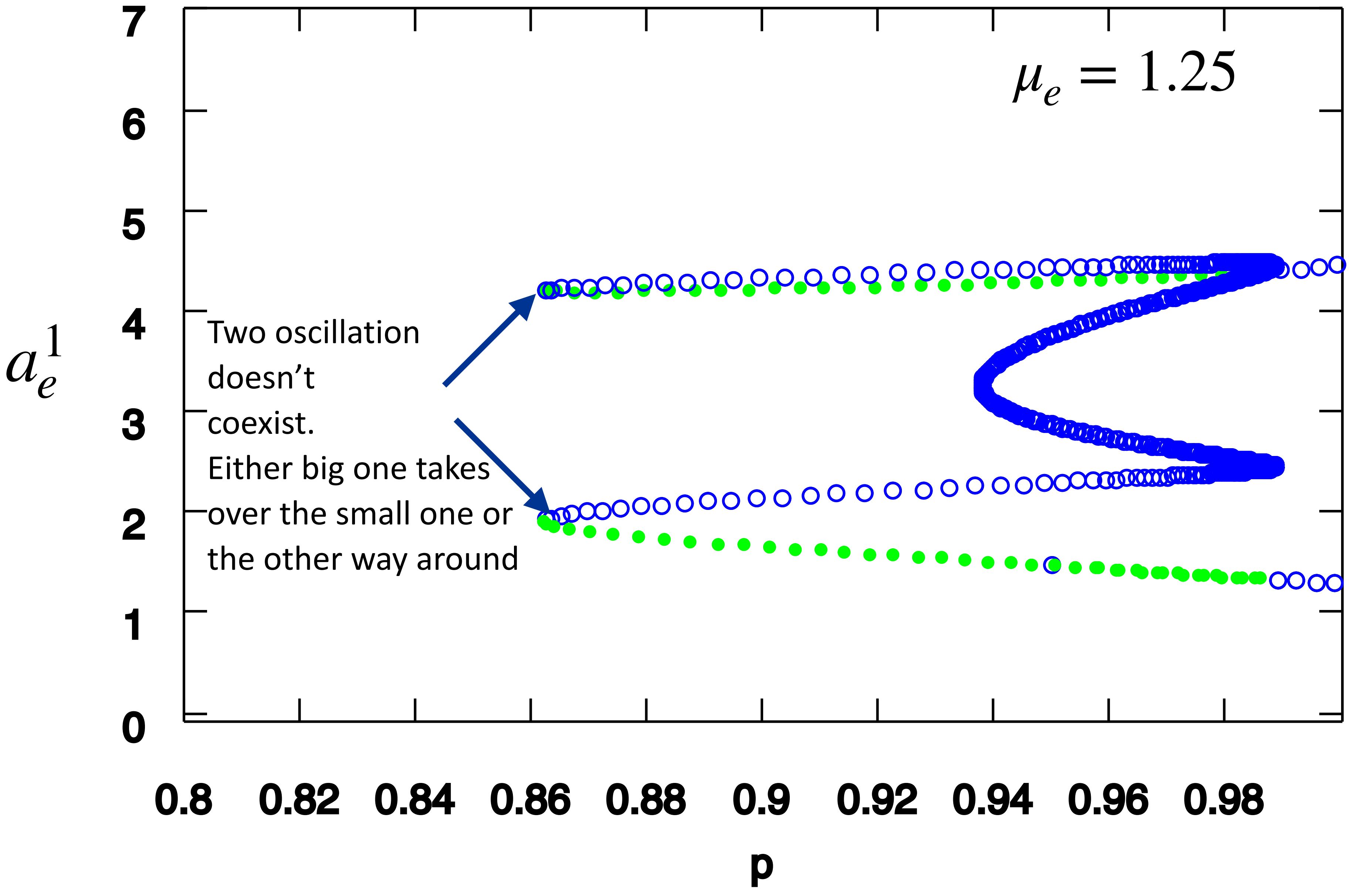
$\mu_e = 1.25$
4.4
1.77
a_e^1
p
0
1
2
3
4
5
6
7
0.8
0.82
0.84
0.86
0.88
0.9
0.92
0.94
0.96
0.98

We can see two distinct oscillations but they are not phase locked!



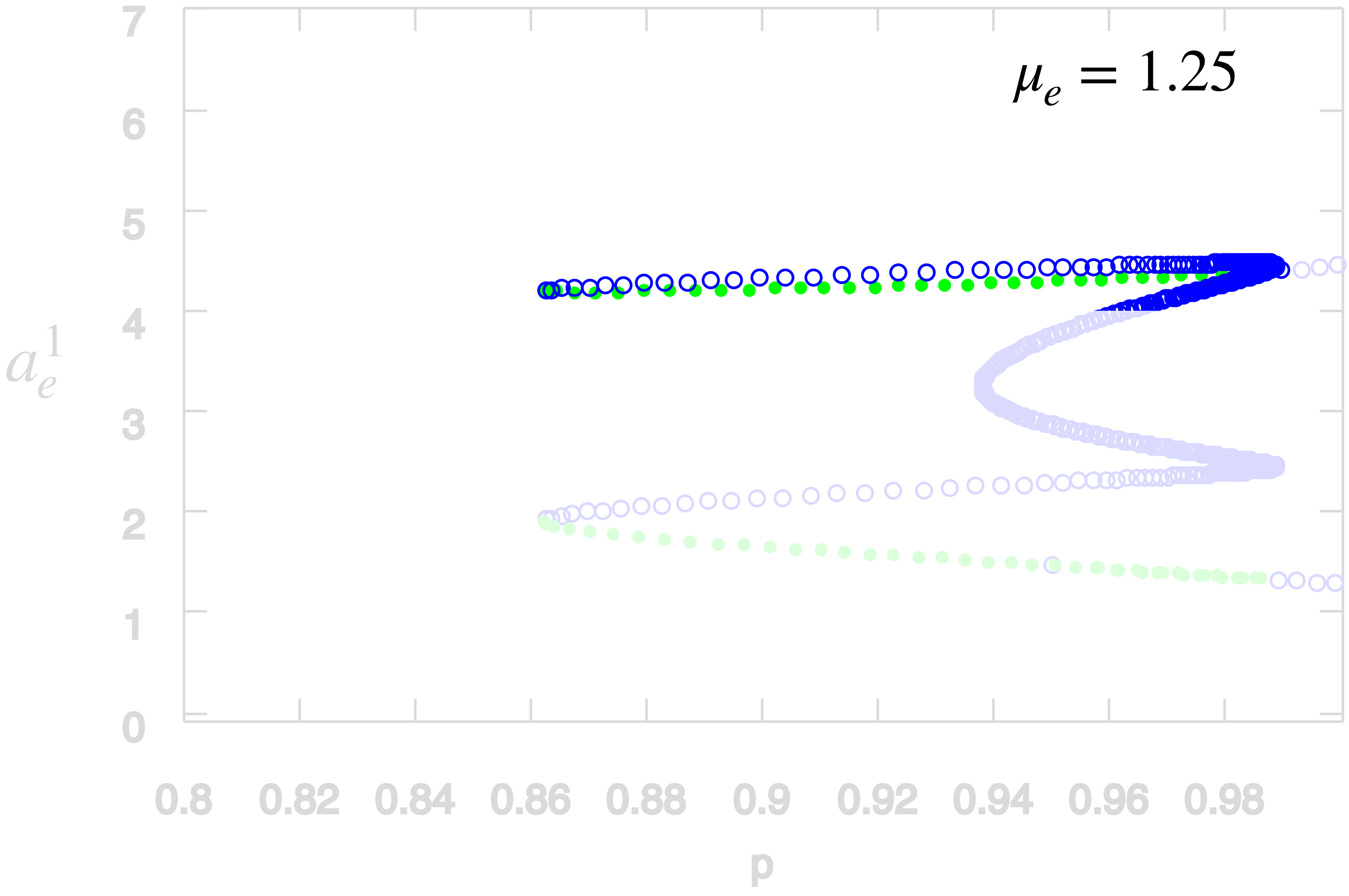
We can see two distinct oscillations but they are phase locked!



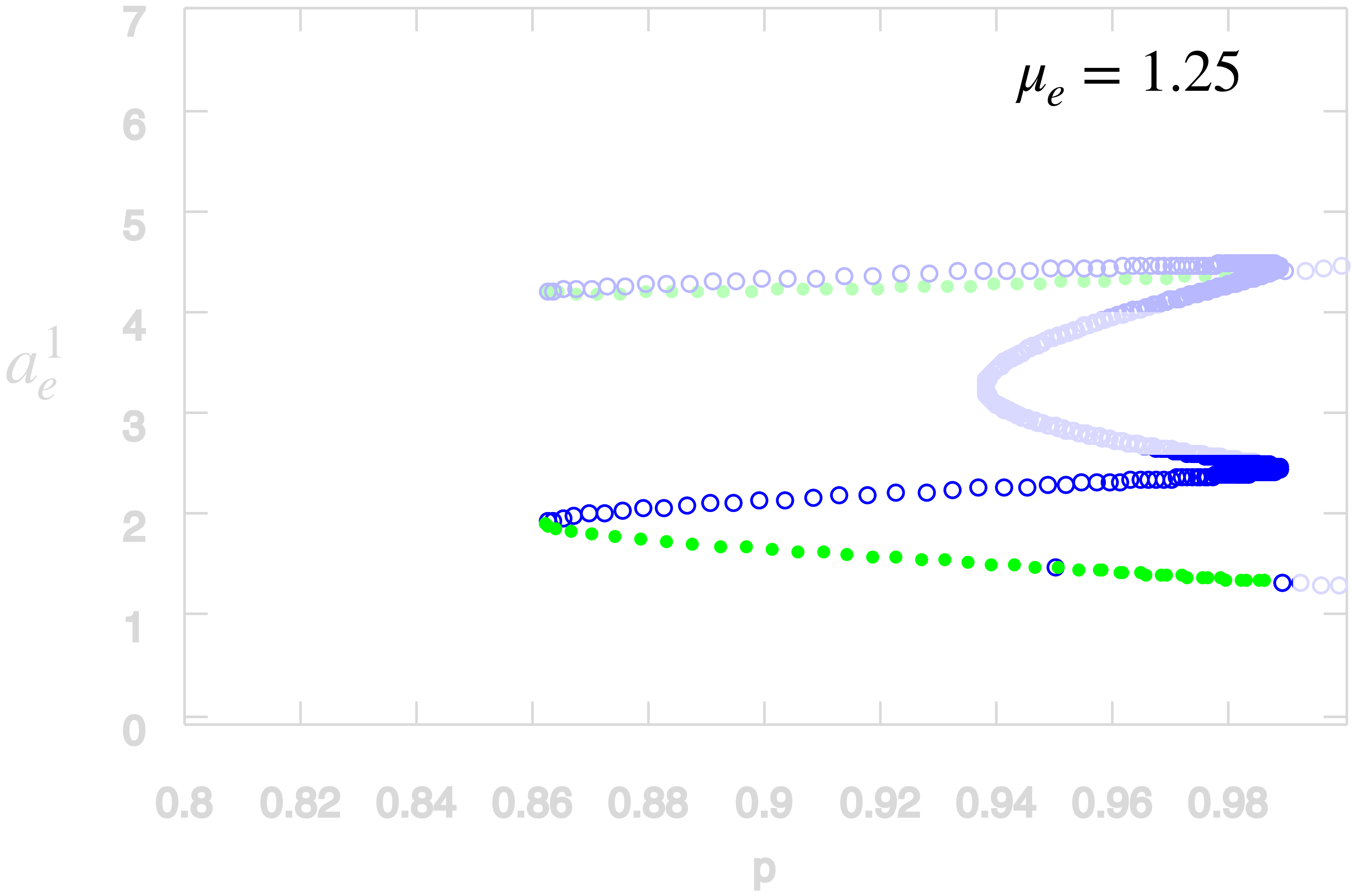


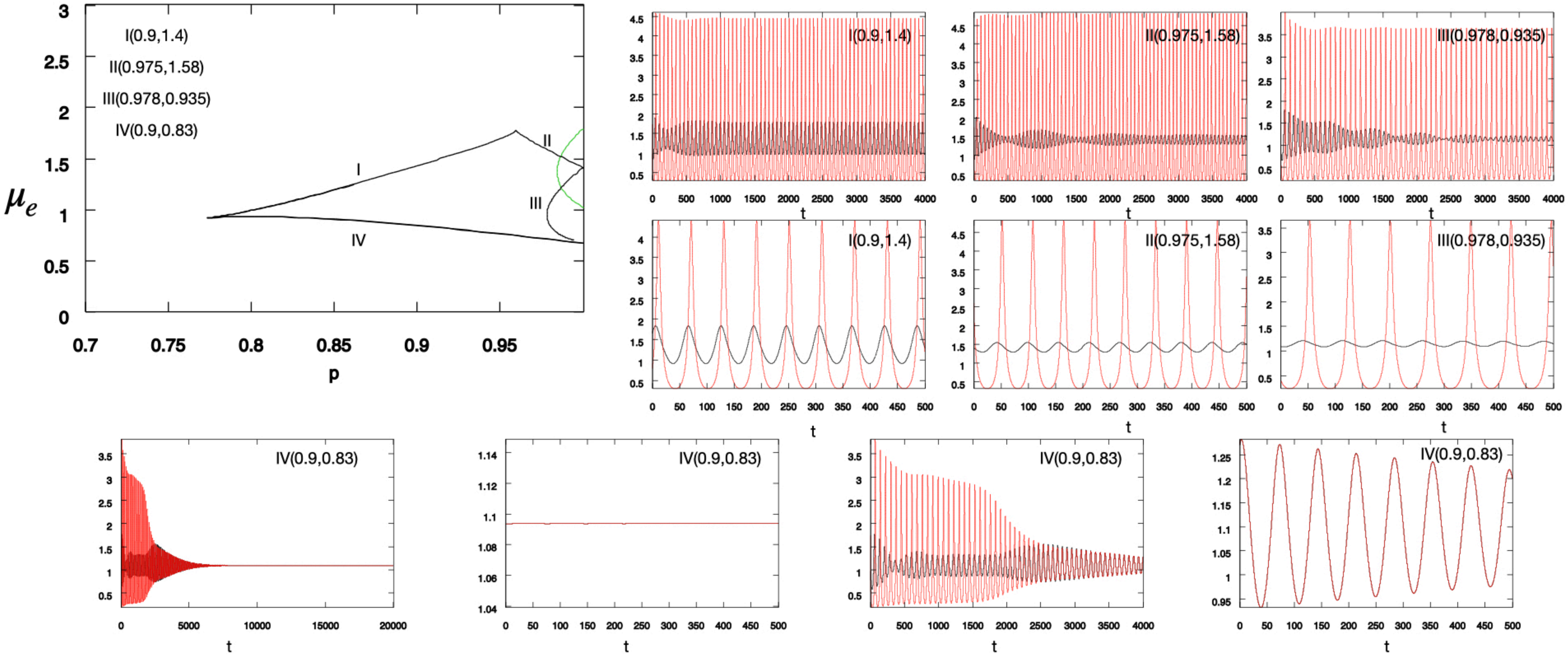
$\mu_e = 1.25$
a_e^1
p
0
1
2
3
4
5
6
7
0.8
0.82
0.84
0.86
0.88
0.9
0.92
0.94
0.96
0.98
Two oscillation
doesn't
coexist.
Either big one takes
over the small one or
the other way around

This is the region where a_e^1 is big and a_e^2 is small

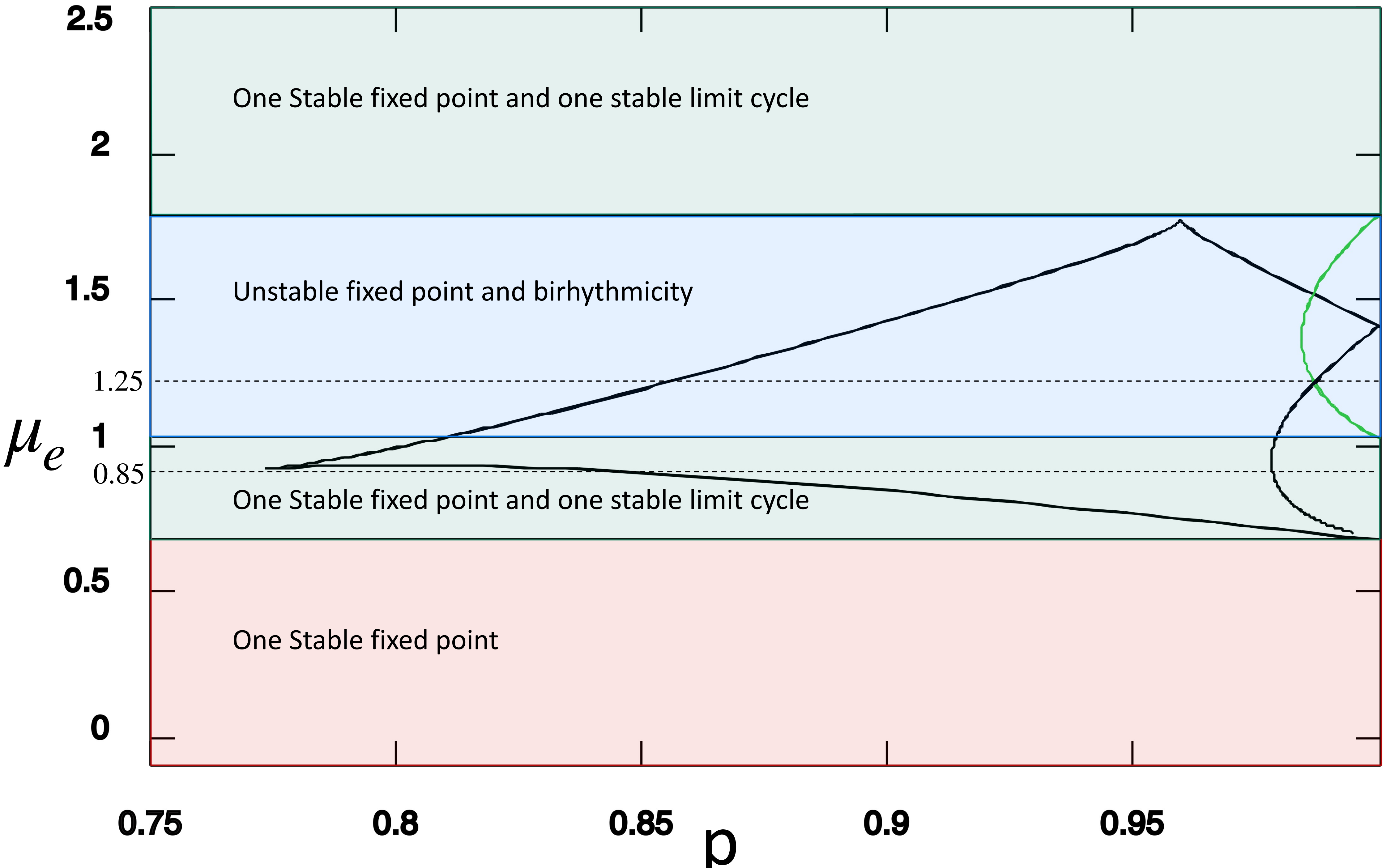


This is the region where a_e^1 is small and a_e^2 is big





a_e^1 : *Black* a_e^2 : *Red*



2.5
2
1.5
1.25
1
0.85
0.5
0
μ_e
One Stable fixed point and one stable limit cycle
Unstable fixed point and birhythmicity
One Stable fixed point and one stable limit cycle
One Stable fixed point
0.75
0.8
0.85
0.9
0.95
p

Conclusion

- We have started with globally coupled QIF to come to a 9D model to search parameters for multi rhythmicity.
- Tried to think about what we get with and without Somatostatin and with or without effect of pervalbumin.
- Simulate mean field model and spiking model
- Tried to replicate analytical phenomenon in 3D.
- Mixing population to start some in bigger limit cycle and some in smaller limit cycle

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Future work

- Investigate mixing populations in 3D to understand convergence dynamics between larger and smaller limit cycle groups.
- Explore trirhythmicity in 3D systems and characterize the parameter regions supporting it.
- Producing the jump from smaller limit cycle to the bigger one in 18D model.
- Study wavefront propagation under spatial all-to-all coupling to reveal emergent collective behaviors.

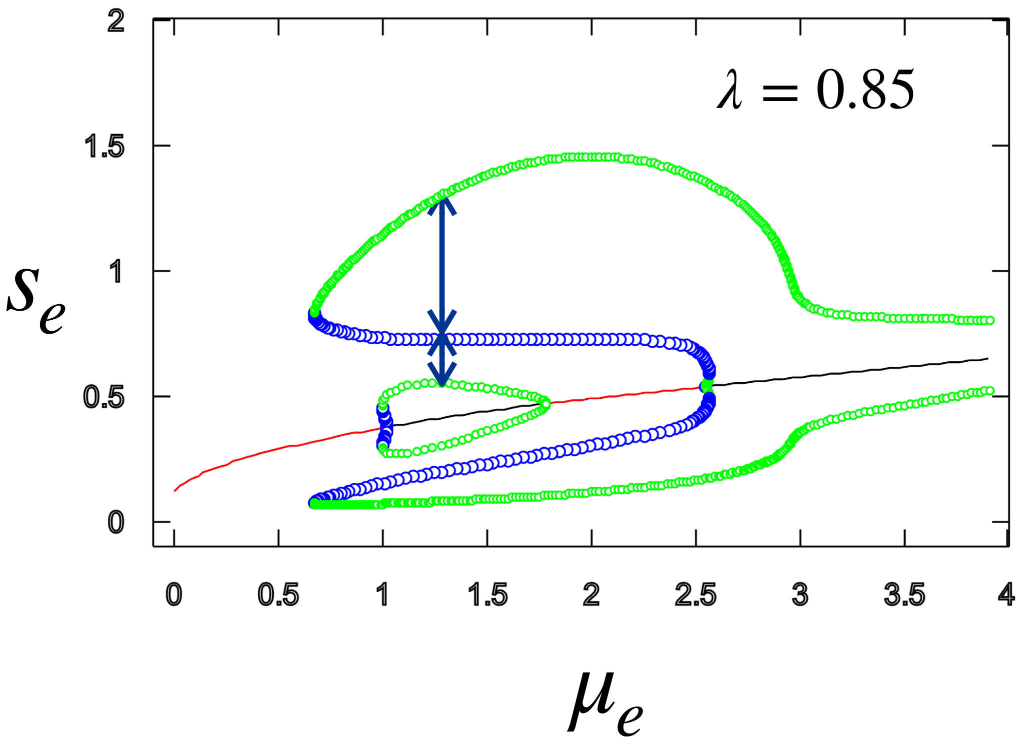
Mixing population in 3D

Certain limiting cases allow analytical reduction of the 9D system to 3D, where unexplored regions may exhibit trirhythmicity.

We have not yet investigated the interaction of two populations in 3D, with one on a large limit cycle and the other on a small one; mixing them and analyzing the resulting bifurcation structure remains an open direction.

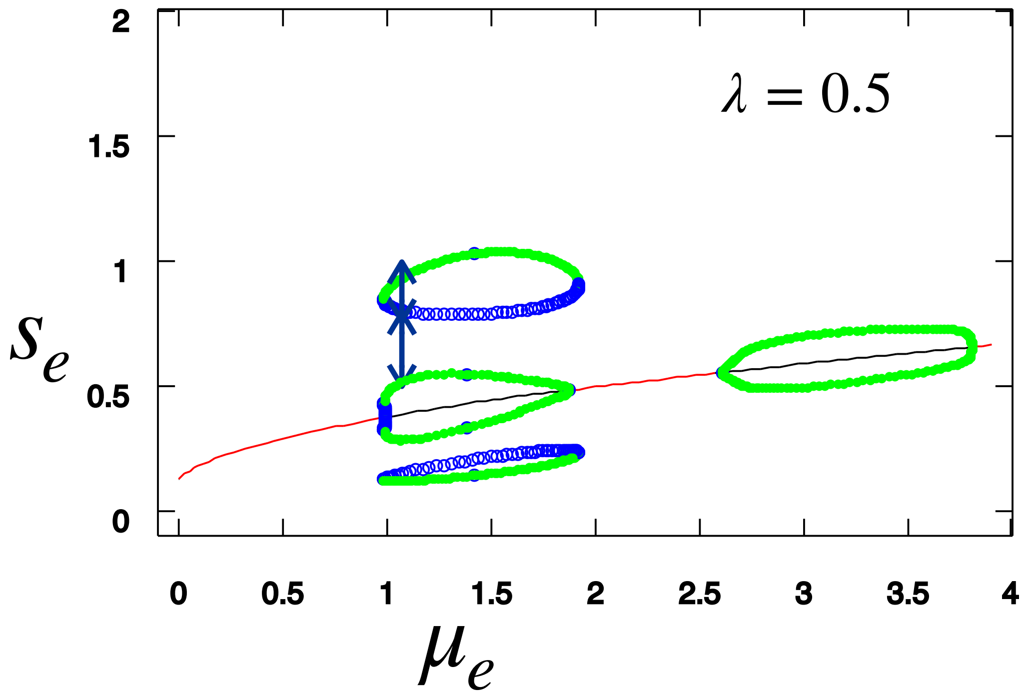
Mixing population in 9D: big to small

In mixing of population we have seen the small limit cycle going to the big limit cycle but not the other way around. The main reason might be:



Mixing population in 9D: big to small

So, one of the better option can be to choose:



Spatial all to all coupling

We consider an all-to-all coupling architecture where each neuron interacts with its immediate neighbors (preceding and following neurons). Our goal is to study how varying the coupling strength influences the system's collective dynamics.

$$a_l[j]' = \frac{1}{t_{m_l}}(2a_l[j]b_l[j] + \Delta_l)$$

$$b_l[j]' = \frac{1}{t_{m_l}}(b_l[j]^2 - a_l[j]^2 + g_{el}((1-2q_e)s_e[j] + q_e(s_e[j+1] + s_e[j-1])) - g_{il}((1-2q_i)s_i[j] + q_i(s_i[j+1] + s_i[j-1])) - \lambda g_{se}((1-2q_s)s_s[j] + q_s(s_s[j+1] + s_s[j-1])) + \mu_l)$$

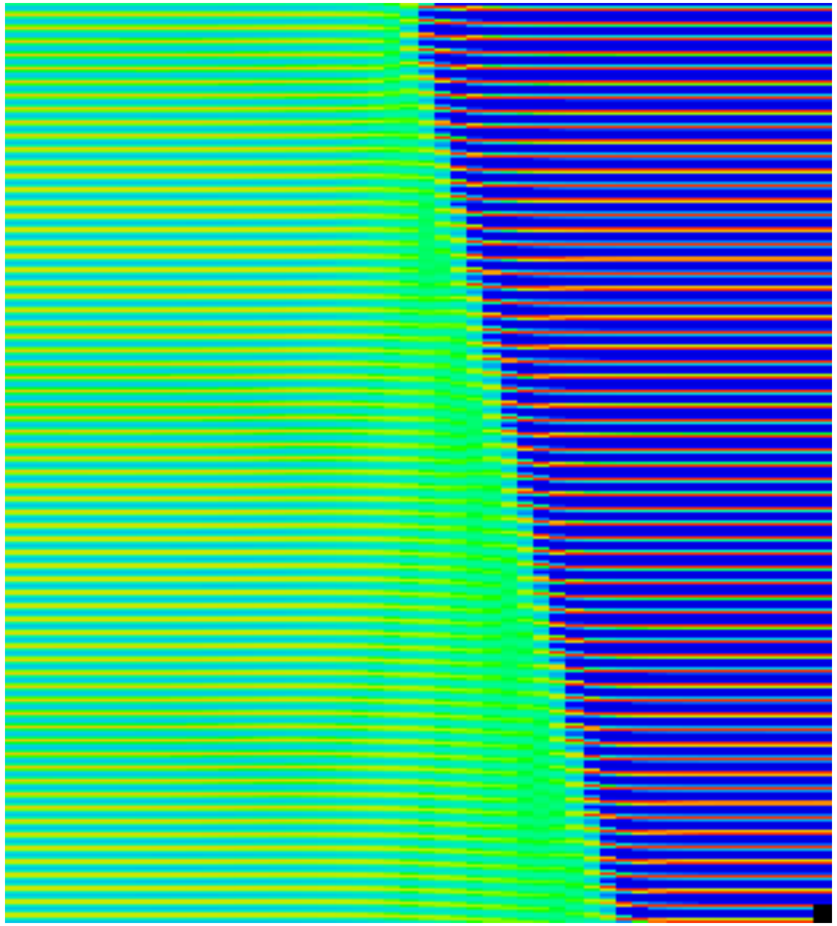
$$s_l[j]' = \frac{1}{\tau_l}(-s_l[j] + a_l[j]/\pi)$$

where $k, l \in \{e, i, s\},\ j = 1, \cdots, 50$

Spatial all to all coupling

time(t)

$0 < t < 4000$



$a_e[1]\ a_e[2]\cdots a_e[49]\ a_e[50]$

$q_e = 0.2,\ q_i = 0,\ q_s = 0.3$

We observe the emergence of a wavefront in the coupled system. As part of our future work, we plan to analyze this wavefront behavior in detail, focusing on the underlying model dynamics and how coupling influences its propagation.

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Timeline:

End of summer: Submit the paper and working with 3D model, jump from smaller limit cycle to the bigger one in 18D model.

Next Fall: Thesis and spatial network

Next January: Defense

Kenneth P.Dietrich School of Arts & Sciences; Department of Mathematics

Thank you

ard129@pitt.edu