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# The non linear dynamics of retinal waves

Bruno Cessac

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# The non linear dynamics of retinal waves

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Sophia Antipolis, France

*Inria*

 UNIVERSITÉ  
CÔTE D'AZUR



NeuroMod

# Contributors

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S. Picaud, Institut de la Vision, Paris

B. Cessac, Biovision team, INRIA, Sophia Antipolis

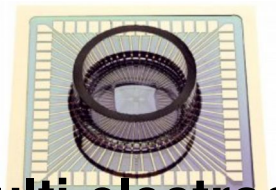
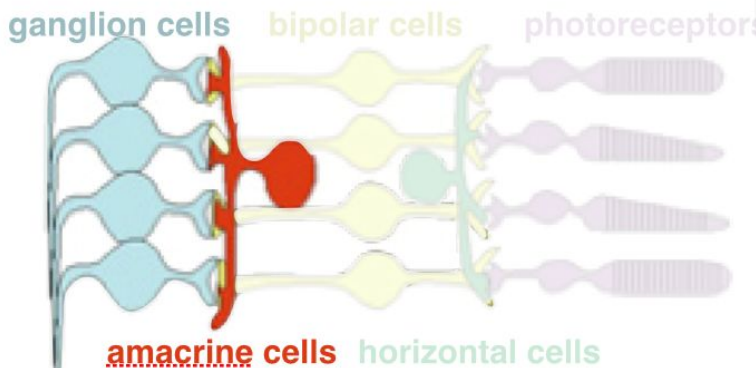
Acknowledgement: M. Hennig, E. Sernagor.

# Retinal waves

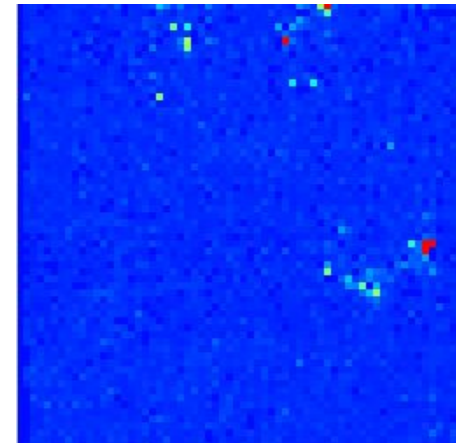
Spontaneous spatio-temporal waves during development

Disappear short after birth when vision is functional

Recordings from the retina



**Multi-electrode array (MEA)**



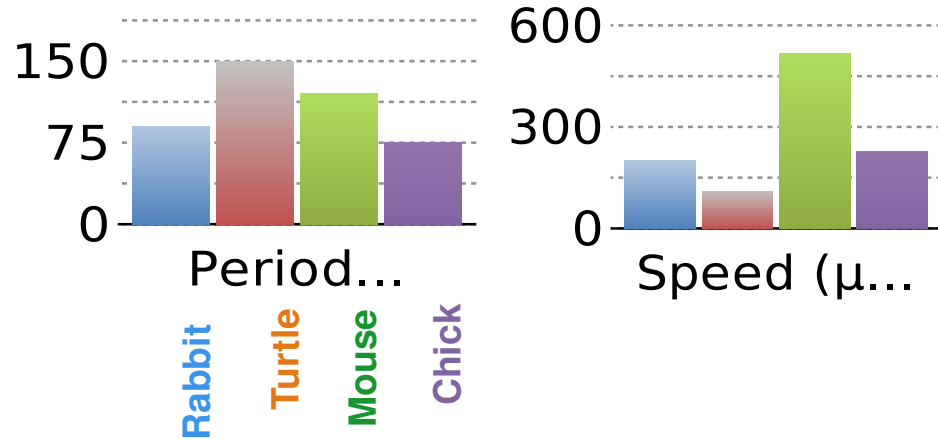
*MEA recording of the voltage from a mouse retina (courtesy E Sernagor).*



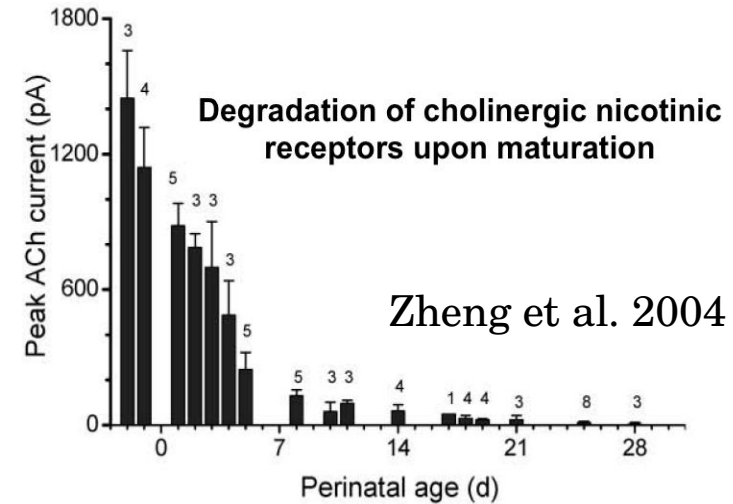
# Variability within retinal waves

## i) Across species

Godfrey et al. 2007

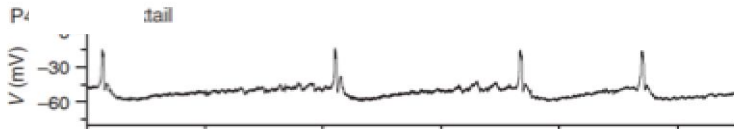


## ii) Development

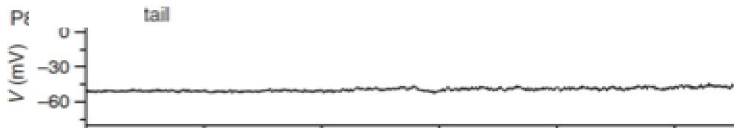


## iii) Pharmacology

P4

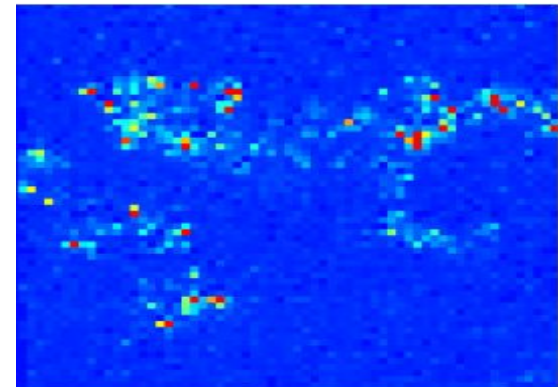


P8



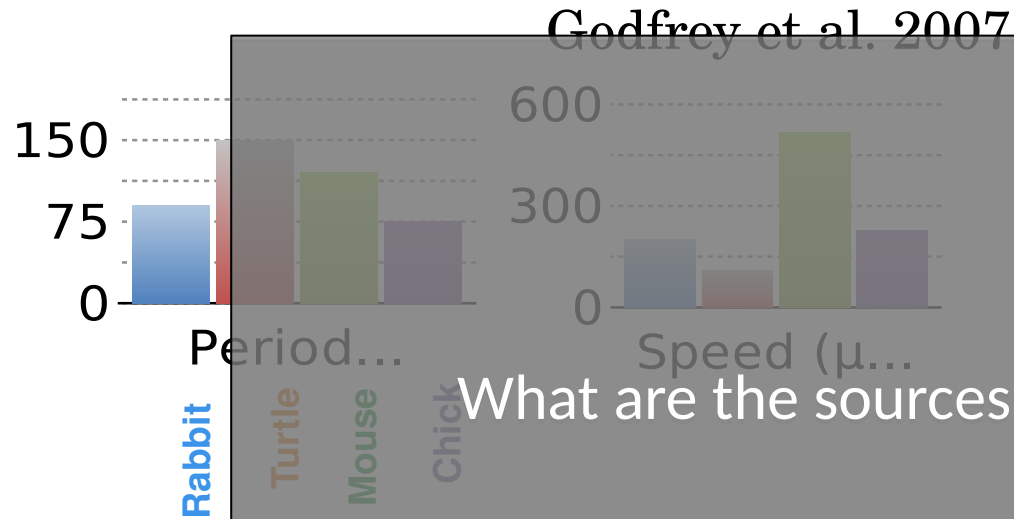
0 Time (sec) 50

## iv) Spatial Variability

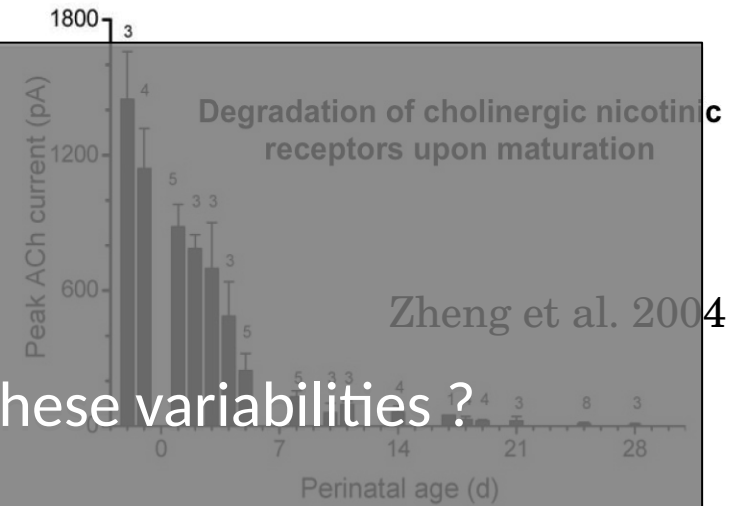


# Variability within retinal waves

## i) Across species



## ii) Development



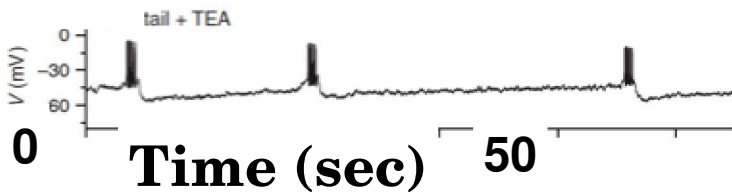
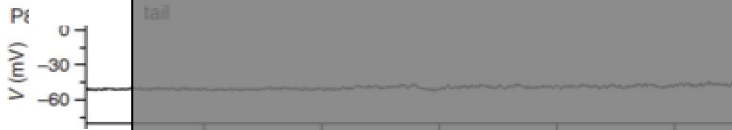
What are the sources of these variabilities?

## iii) Pharmacology

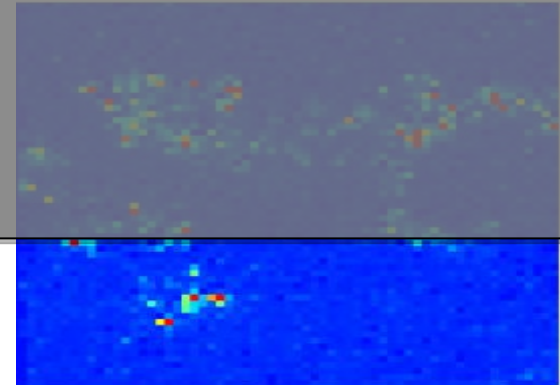
P4



P8



## iv) Spatial Variability

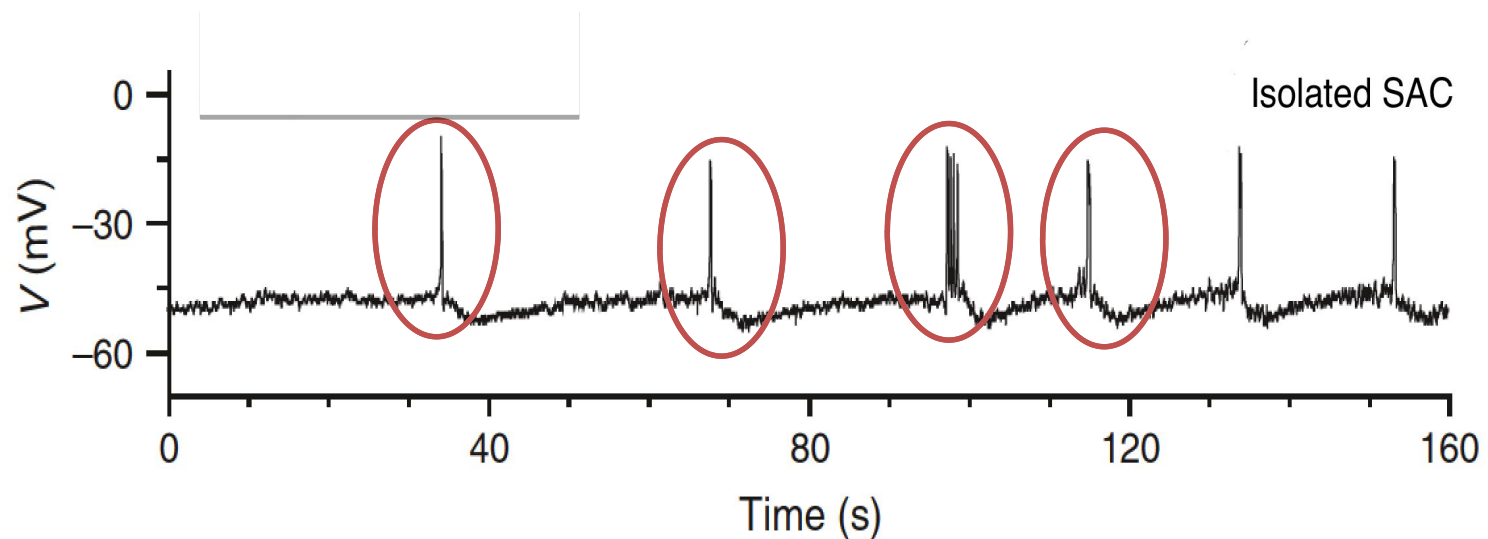


Is there a unique, global, mechanism explaining them?

# Experiments for the emergence of retinal waves

Retinal waves require three components:

- i) Spontaneous bursting activity

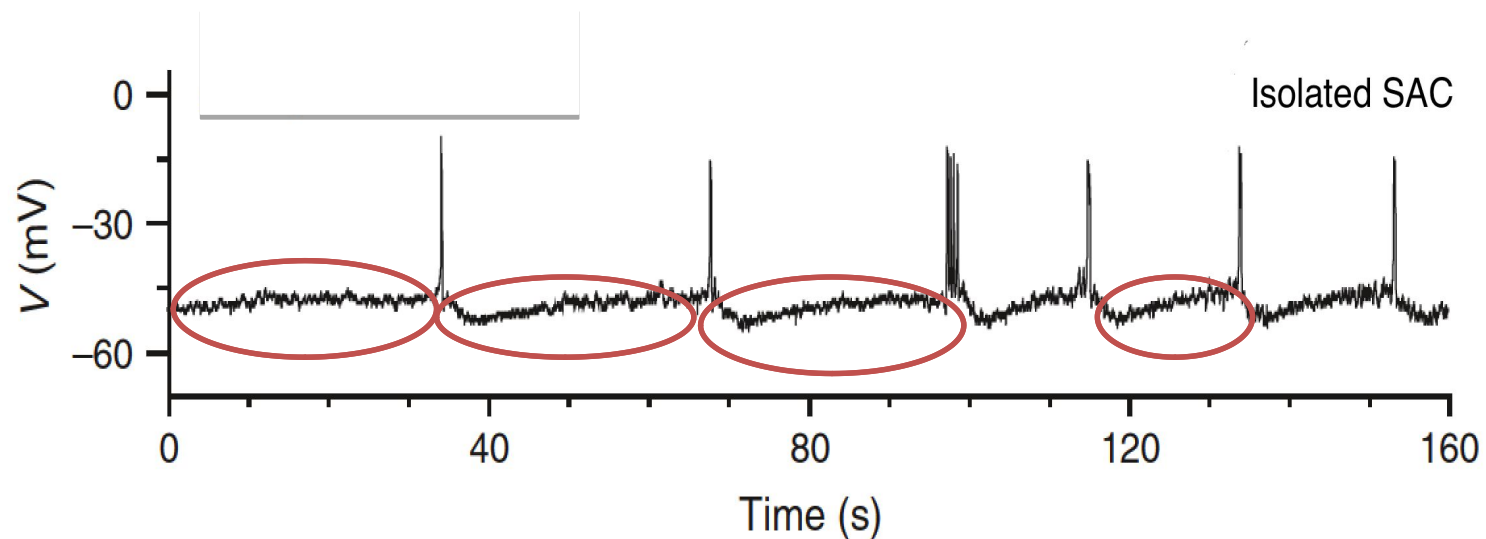


Experiment for isolated neurons,  
Zheng et al., 2006, Nature

# Experiments for the emergence of retinal waves

Retinal waves require three components:

- i) Spontaneous bursting activity
- ii) Refractory mechanism (slow After HyperPolarisation- sAHP)

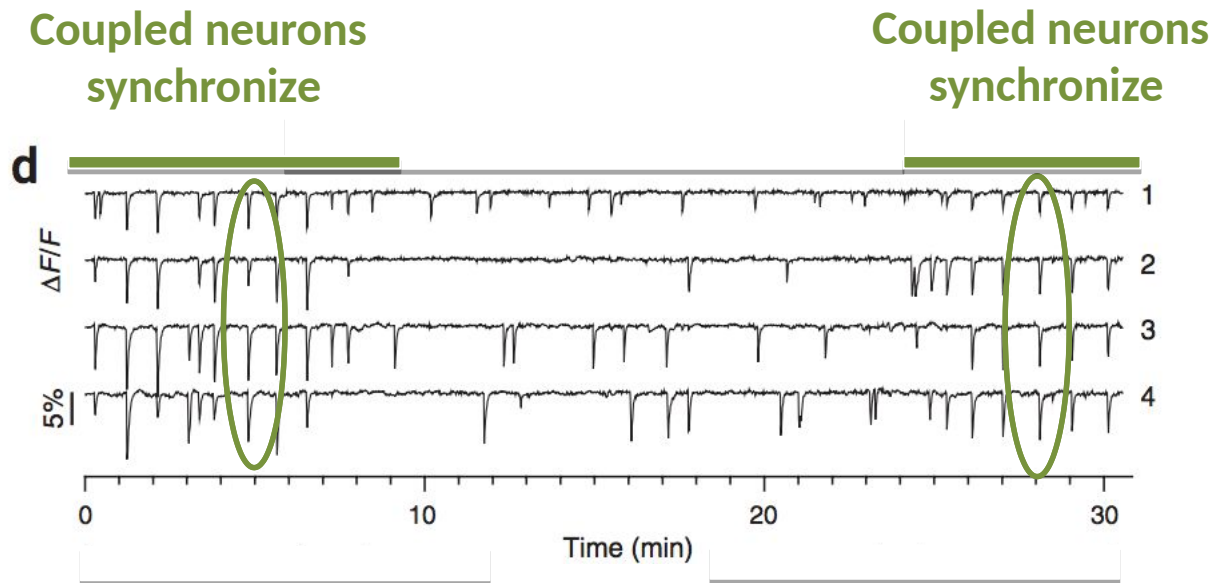


Experiment for isolated neurons,  
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# Experiments for the emergence of retinal waves

Retinal waves require three components:

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- iii) Coupling (through Acetylcholine neurotransmitter)

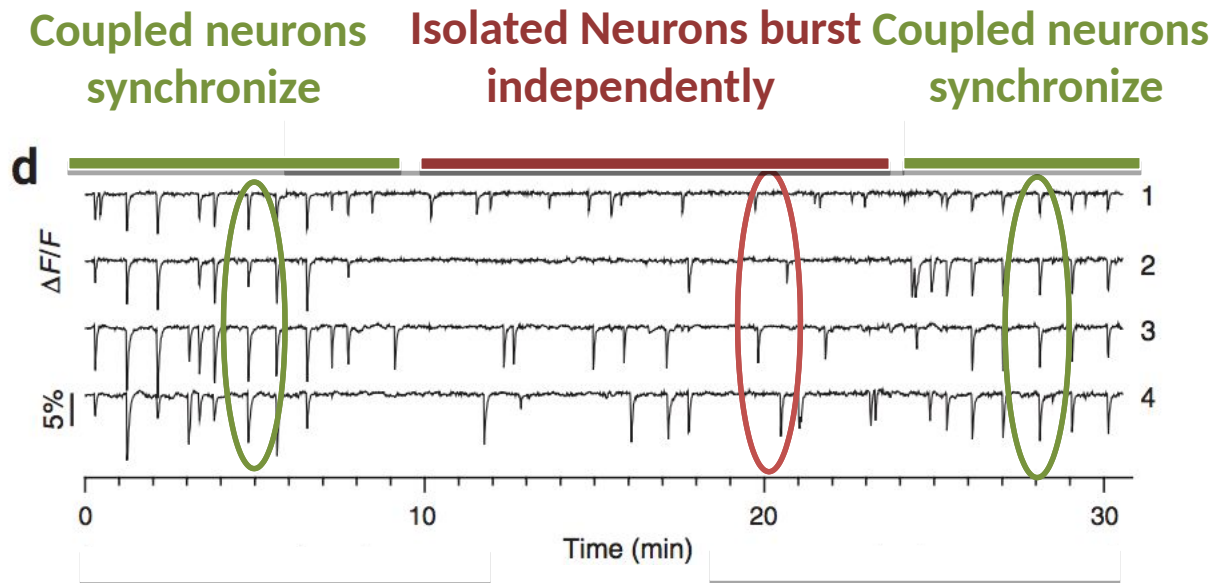


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Retinal waves require three components:

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Experiment for coupled and isolated neurons, Zheng et al., 2006, Nature

# Experiments for the emergence of retinal waves

Retinal waves require three components:

Several models of stage II retinal waves have been already proposed:

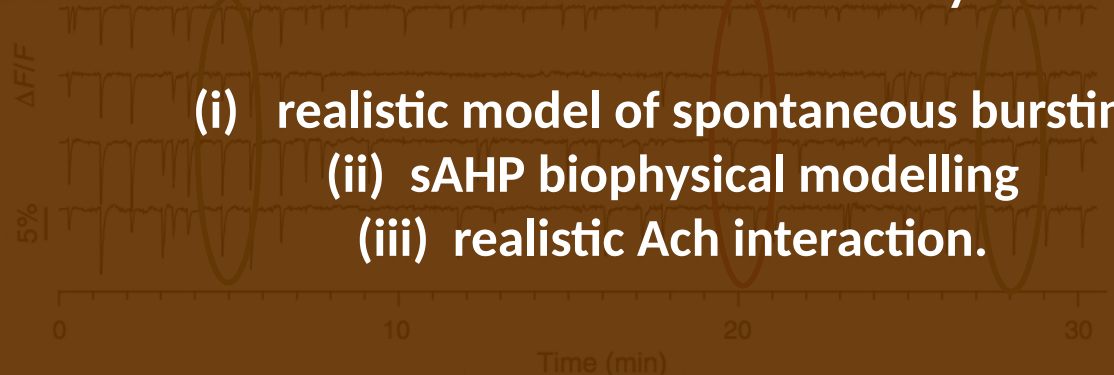
ii) Refractory mechanism (slow After HyperPolarisation- sAHP)

iii) Coupling (through Acetylcholine neurotransmitter)

- Feller et al Neuron, 1997.
- Eglon et al, J. Neuroscience 2000.
- Godfrey & Swindale. PLoS Comput Biol, 2007.
- Hennig et al, J. Neuroscience, 2009
- Lansdell et al, PLoS Computational Biology, 2014.

But none of them as studied simultaneously these 3 aspects:

- (i) realistic model of spontaneous bursting
- (ii) sAHP biophysical modelling
- (iii) realistic Ach interaction.



Experiment for coupled and isolated neurons, Zheng et al., 2006, Nature

# A network model for stage II retinal waves



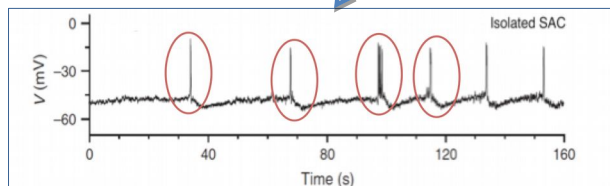
# A network model for stage II retinal waves

## Membrane potential dynamics

$$C_m \frac{\partial V}{\partial t} = -g_L^M (V - V_L) - g_{Ca}(V)(V - V_{Ca}) - g_K N (V - V_K)$$

$$\tau_N \frac{\partial N}{\partial t} = \Lambda(V)(N_\infty(V) - N)$$

**Morris-Lecar &  
Fast K<sup>+</sup> channels**



# A network model for stage II retinal waves

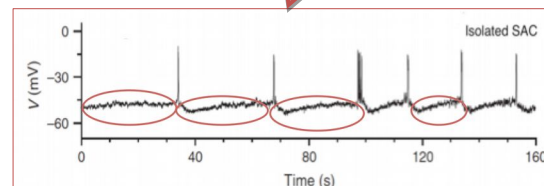
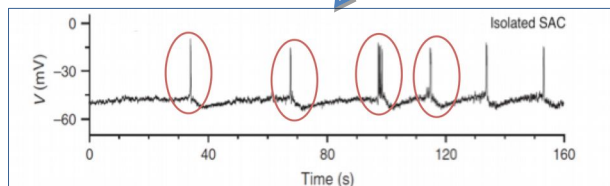
## Membrane potential dynamics

$$C_m \frac{\partial V_i}{\partial t} = -g_L^M (V_i - V_L) - g_{Ca}(V_i)(V_i - V_{Ca}) - g_K N_i (V_i - V_K) - g_{sAHP} R_i^4 (V_i - V_K) - \sum_j g_{j,Ach}(A_j)(V_i - V_{Ach})$$

$$\tau_N \frac{\partial N}{\partial t} = \Lambda(V)(N_\infty(V) - N)$$

**Morris-Lecar &  
Fast K<sup>+</sup> channels**

**sAHP current  
Refractory  
mechanism**



# A network model for stage II retinal waves

## Membrane potential dynamics

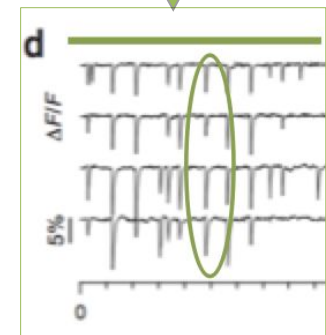
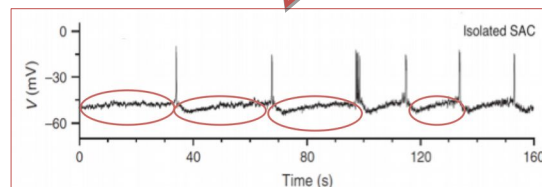
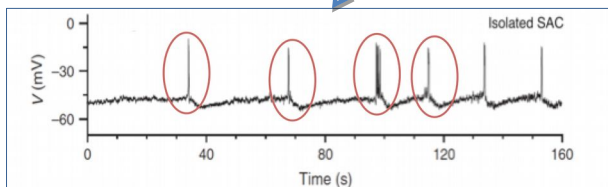
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**Morris-Lecar &  
Fast K<sup>+</sup> channels**

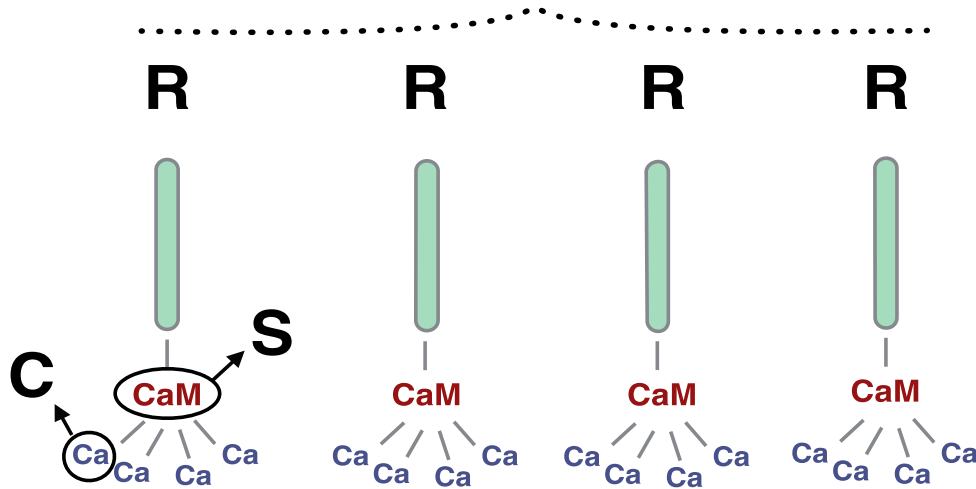
**sAHP current  
Refractory  
mechanism**

**Ach current  
Network  
effect**



# Set of equations for sAHP current

## Activated Ca-gated K<sup>+</sup> channel



## Gating mechanism

All 4 subunits R of the channel are bound to activate the channel



Saturated Calmodulin molecule (CaM) bind to each channel subunit



4 Calcium ions bound to each saturated Calmodulin complex S

# Set of equations for sAHP current

## Activated Ca-gated K<sup>+</sup> channel

$$\tau_R \frac{dR}{dt} = a_R S(1 - R) - R$$

**R** : Probability that one terminal of the channel is open

$$\tau_S \frac{dS}{dt} = -S + a_S C^4(1 - S)$$

**S** : Fraction of saturated calmodulin concentration (CaM)

$$\tau_{Ca} \frac{dC}{dt} = -a_R \frac{C}{H_x} + C_o + \delta_{Ca} I_{Ca}(V)$$

**C** : Intracellular Calcium concentration

# SACs Network

## Model synaptic interactions

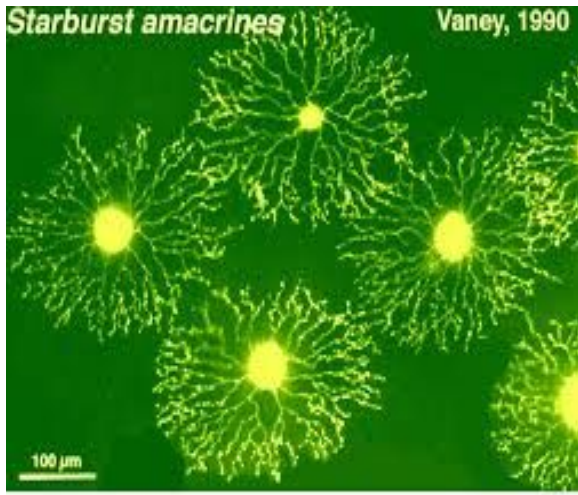
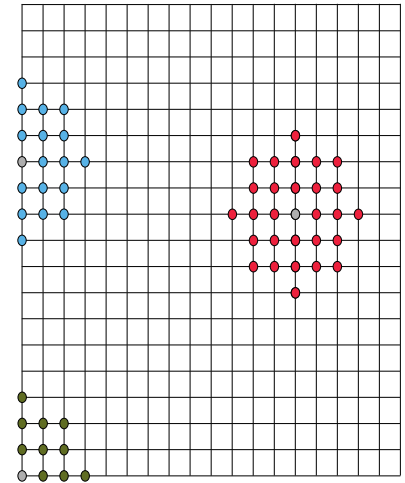
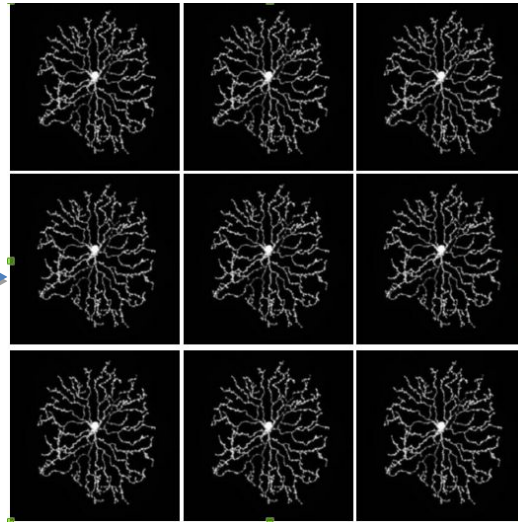


Fig 7. Starburst amacrine cells are stained with Lucifer yellow in whole-mount rabbit retina.



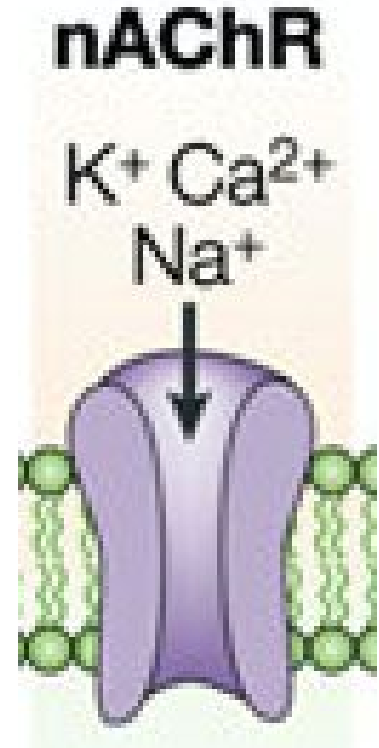
SACs realistic connections

SACs on a lattice

SACs become points on a lattice

# Acetylcholine current

- Molecular nicotinic receptors (nAChR)
- Two molecules of acetylcholine bind to open a nicotinic channel
- The nicotinic conductance depends on the second power of the acetylcholine neurotransmitter concentration A



$$g_{ACh} = g_{ACh}^m \frac{A^2}{K_d^2 + A^2}$$

# A network model for stage II retinal waves

## Membrane potential dynamics

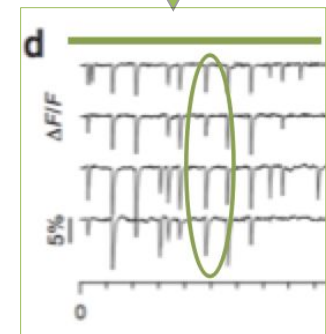
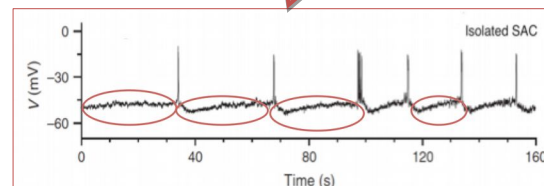
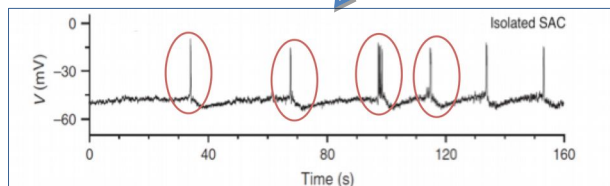
$$C_m \frac{\partial V_i}{\partial t} = -g_L^M (V_i - V_L) - g_{Ca}(V_i)(V_i - V_{Ca}) - g_K N_i (V_i - V_K) - g_{sAHP} R_i^4 (V_i - V_K) - \sum_j g_{j,Ach}(A_j)(V_i - V_{Ach})$$

$$\tau_N \frac{\partial N}{\partial t} = \Lambda(V)(N_\infty(V) - N)$$

**Morris-Lecar &  
Fast K<sup>+</sup> channels**

**sAHP current  
Refractory  
mechanism**

**Ach current  
Network  
effect**





# A network model for stage II retinal waves

$$\left\{ \begin{array}{l} C_m \frac{dV_i}{dt} = -g_L(V_i - V_L) - g_C M_\infty(V_i)(V_i - V_C) - g_K N_i(V_i - V_K) \\ \quad - G_{sAHP}(R_i)(V_i - V_K) - G_A(\{A_k\}_{k \in \mathcal{B}_i})(V_i - V_A) \\ \tau_N \frac{dN_i}{dt} = \Lambda(V_i)(N_\infty(V_i) - N_i) \\ \tau_C \frac{dC_i}{dt} = -\frac{\alpha_C}{H_X} C_i + C_0 - \delta_C g_C M_\infty(V_i)(V_i - V_C) \\ \tau_S \frac{dS_i}{dt} = \alpha_S(1 - S_i)C_i^4 - S_i \\ \tau_R \frac{dR_i}{dt} = \alpha_R S_i(1 - R_i) - R_i \\ \frac{dA_i}{dt} = -\mu A_i + \beta_A T_A(V_i), \end{array} \right.$$

# A network model for stage II retinal waves

N neurons, 6 N equations

$$\left\{ \begin{array}{l}
 C_m \frac{dV_i}{dt} = -g_L(V_i - V_L) - g_C M_\infty(V_i)(V_i - V_C) - g_K N_i(V_i - V_K) \\
 \quad - G_{sAHP}(R_i)(V_i - V_K) - G_A(\{A_k\}_{k=1, \dots, n})(V_i - V_A) \\
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~ 20 parameters per neuron  
 (But neurons are assumed identical)

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~ 20 parameters per neuron  
(But neurons are assumed identical)

Time scale separation

**Fast**  $V, N$ .  $\tau_L = 11$  ms,  $\tau_N = 5$  ms.

**Medium**  $C, A$ .  $\tau_C = 2$  s,  $\tau_A = 1.86$  s.

**Slow**  $S, R$ .  $\tau_R = \tau_S = 44$  s.

# Fast time scale

On the fastest time scale, the sAHP conductance  $G_{S_i} = g_S R_i^4$

and the Acetylcholine conductance  $G_{A_i} = g_A \sum_{j \in \mathcal{B}_i} \frac{A_j^2}{\gamma_A + A_j^2}$

are, as slow variables, essentially constant =>

control parameters of the fast dynamics.

$$\begin{cases} C_m \frac{dV}{dt} = -g_L(V - V_L) - g_C M_\infty(V)(V - V_C) - g_K N(V - V_K) - G_S(V - V_K) - G_A(V - V_A) \\ \tau_N \frac{dN}{dt} = \Lambda(V)(N_\infty(V) - N) \end{cases}$$

# Fast time scale

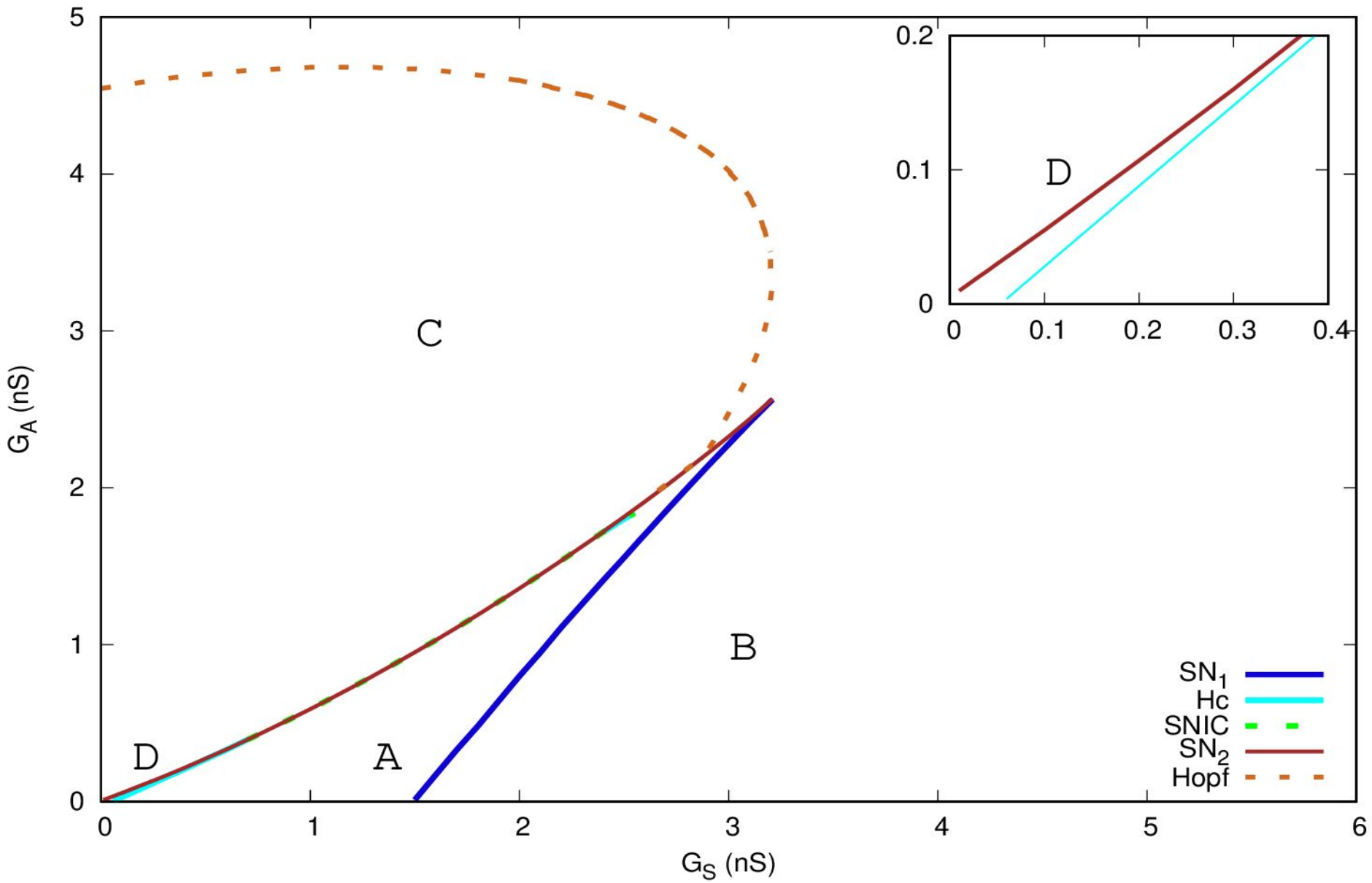
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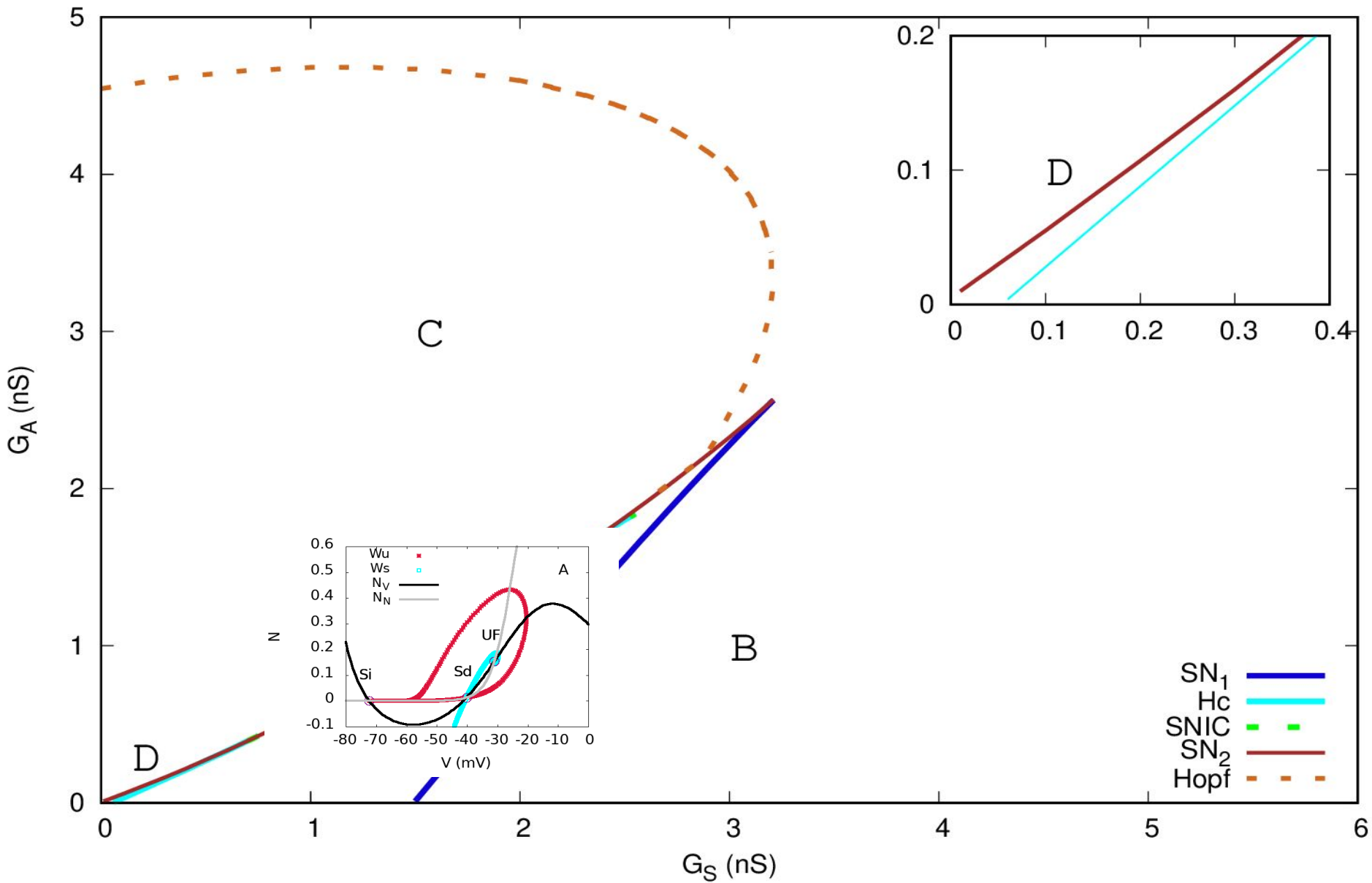
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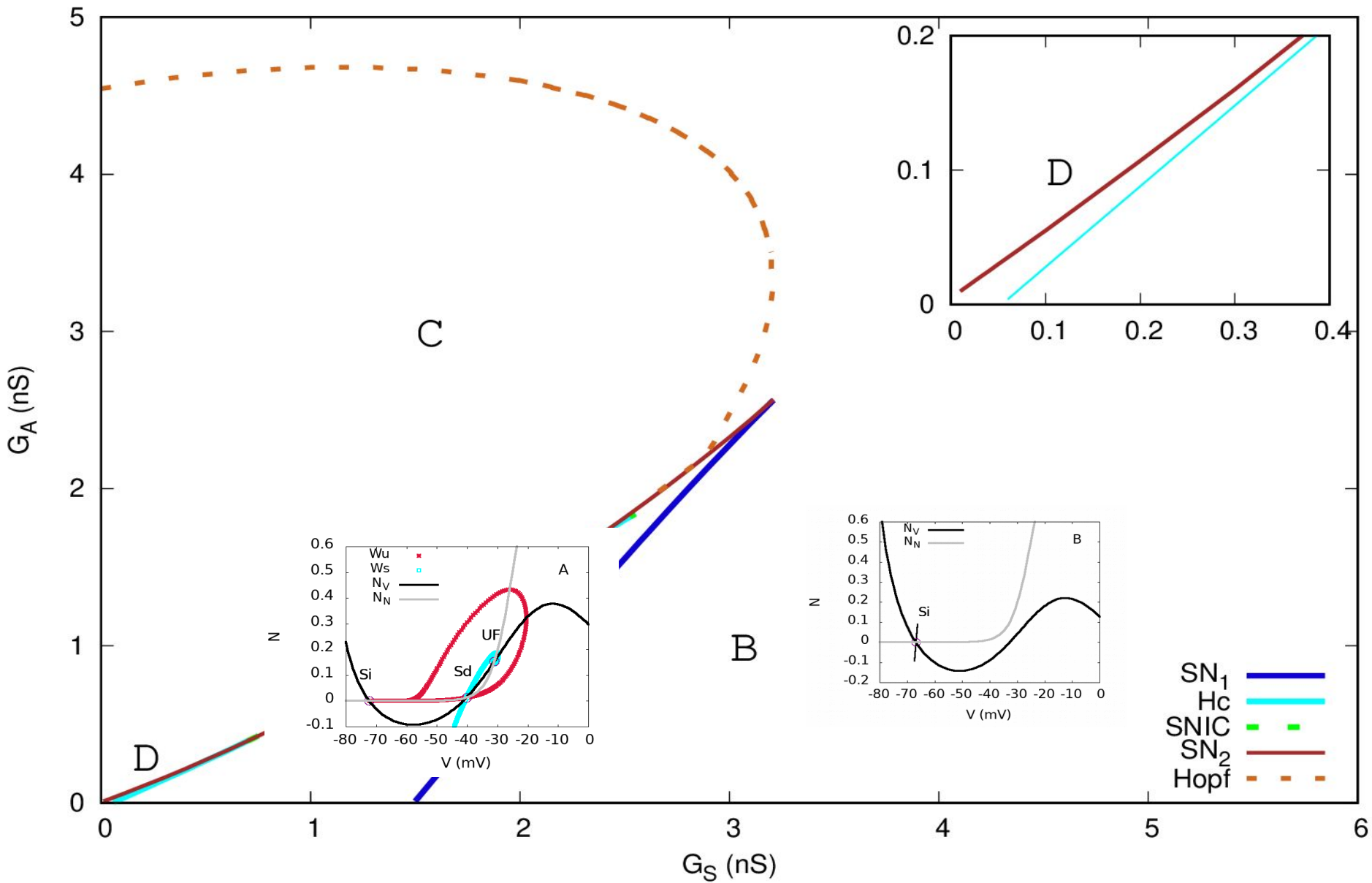
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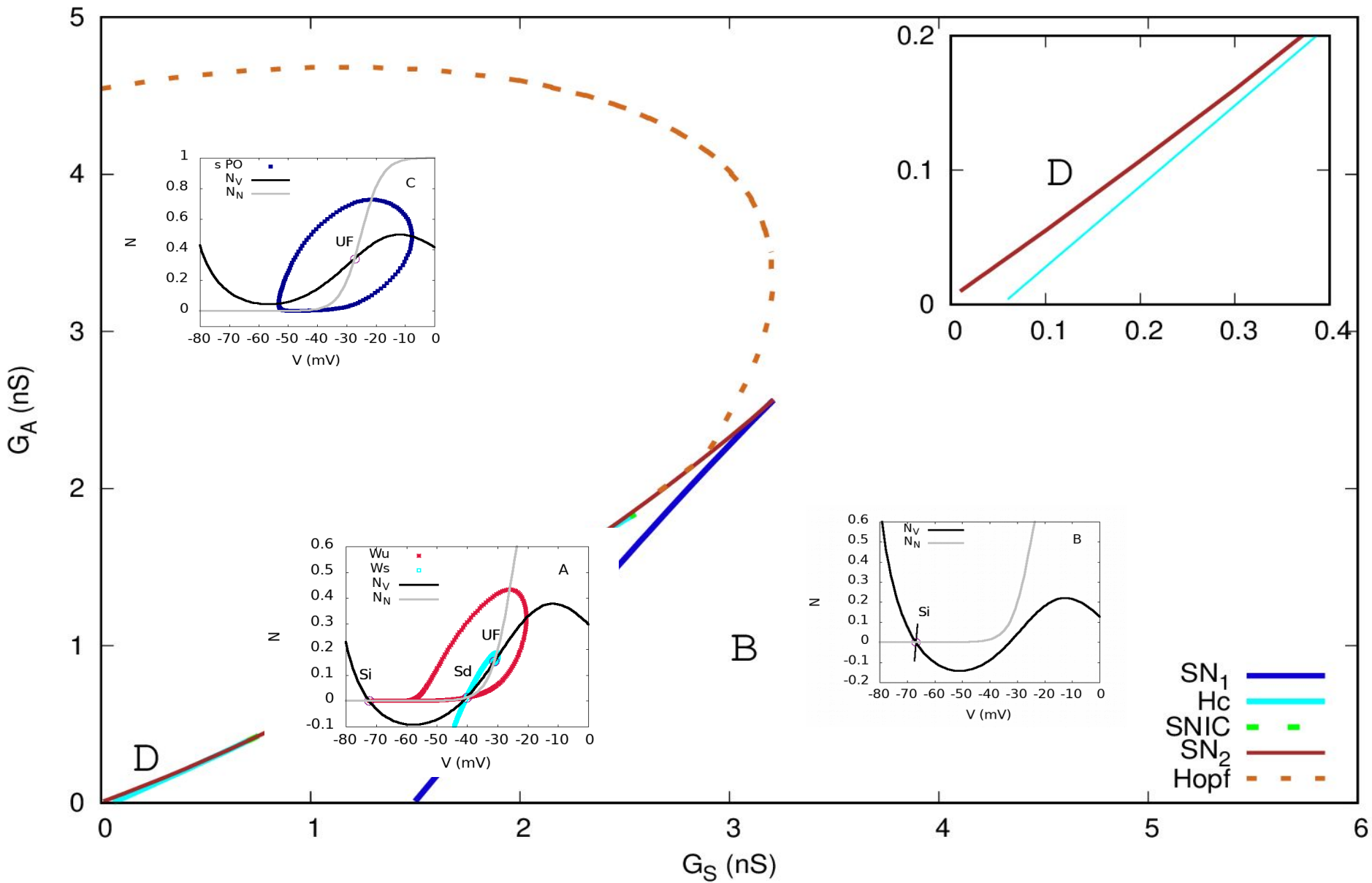
$$\begin{cases} C_m \frac{dV}{dt} = -g_L(V - V_L) - g_C M_\infty(V)(V - V_C) - g_K N(V - V_K) - G_S(V - V_K) - G_A(V - V_A) \\ \tau_N \frac{dN}{dt} = \Lambda(V)(N_\infty(V) - N) \end{cases}$$

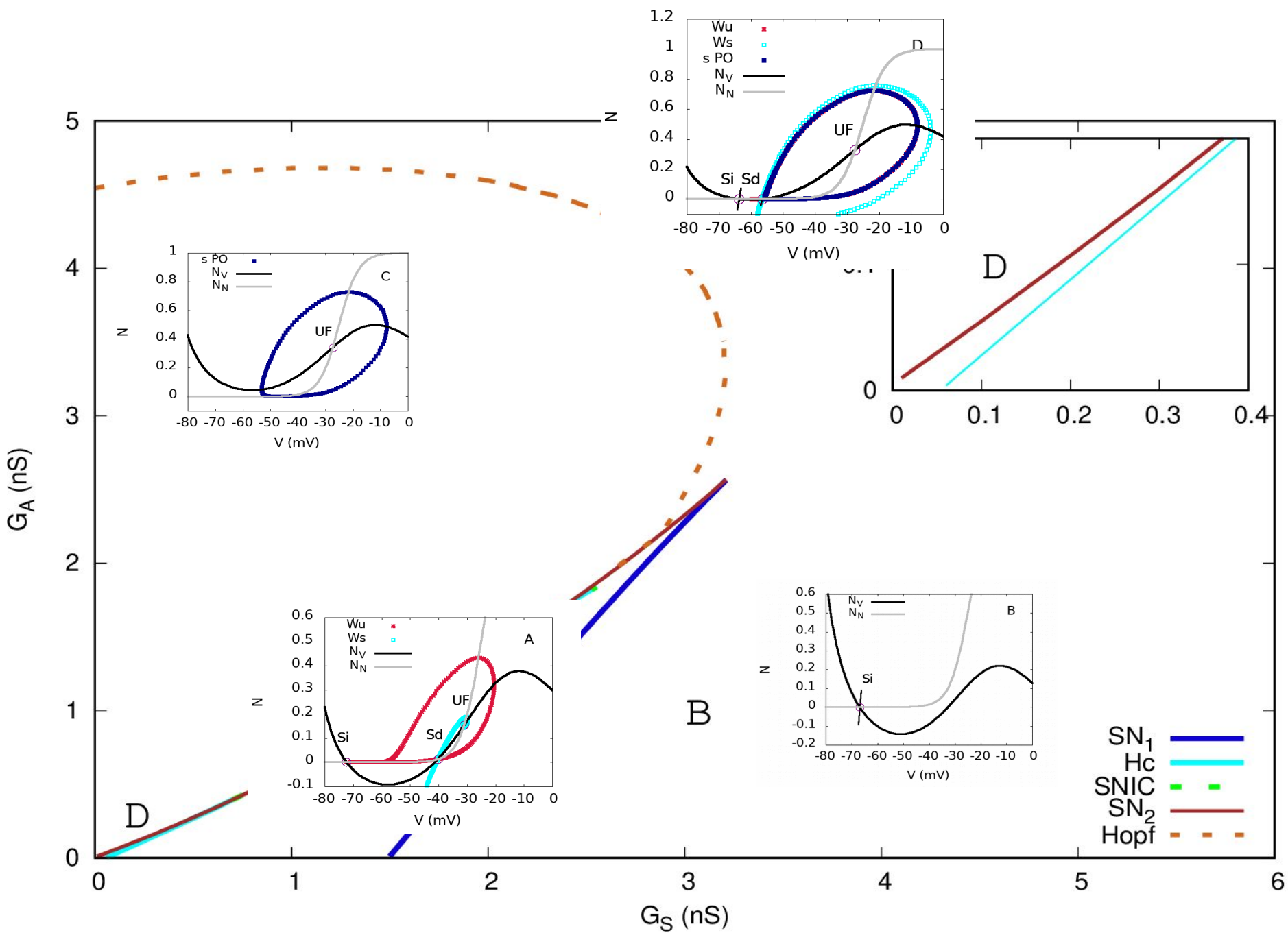




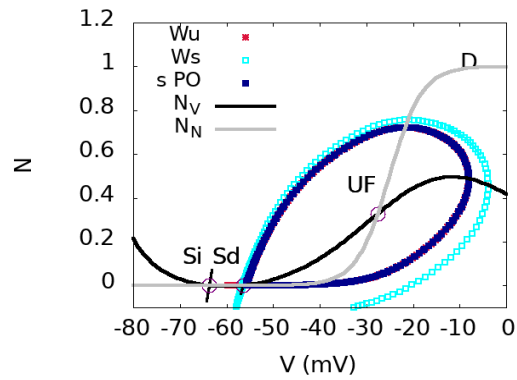




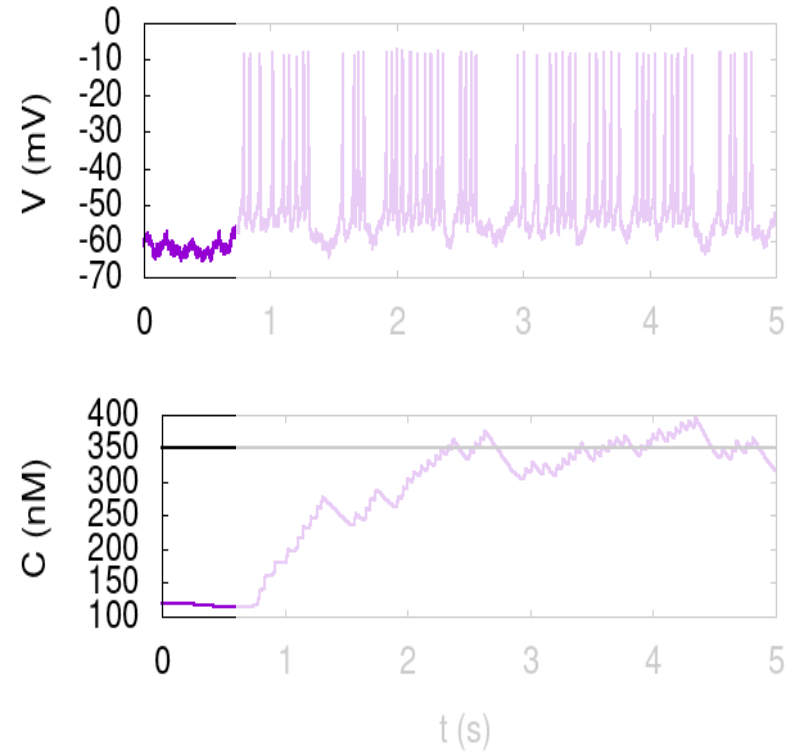
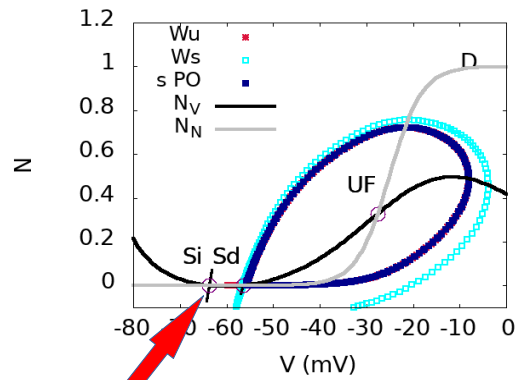




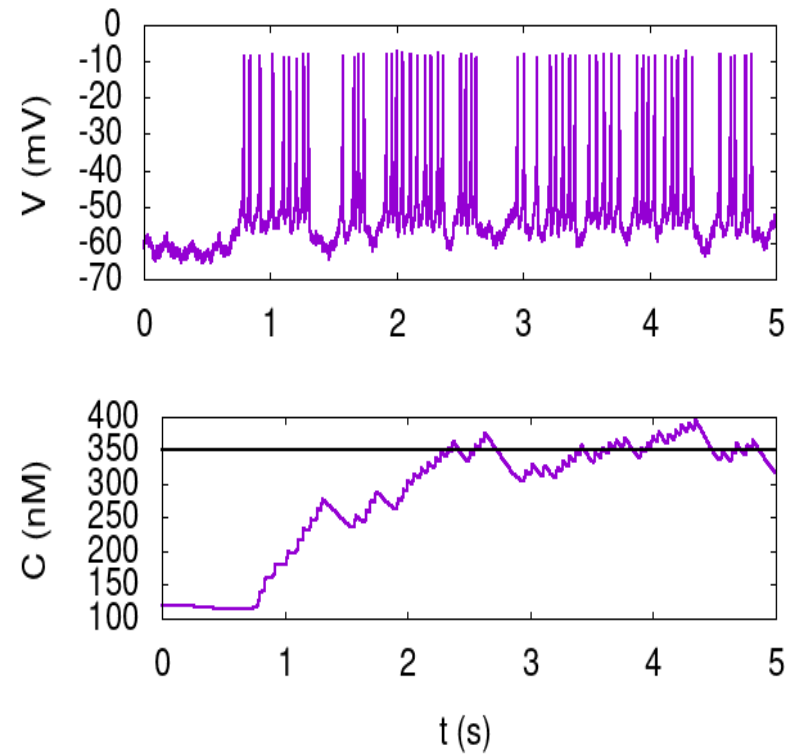
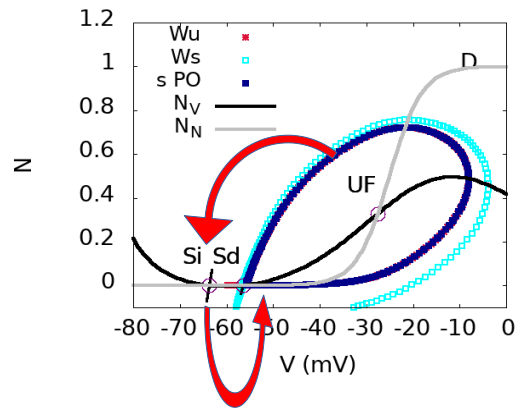
# Noise induced bursting



# Noise induced bursting

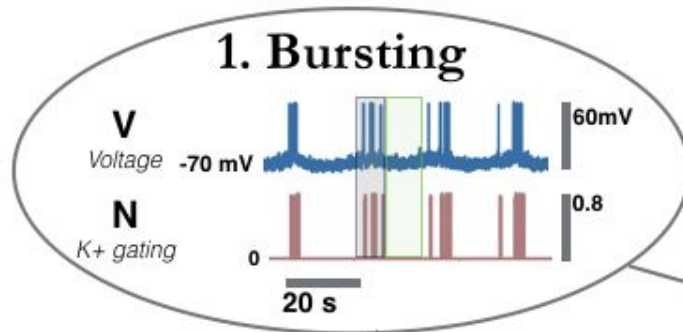


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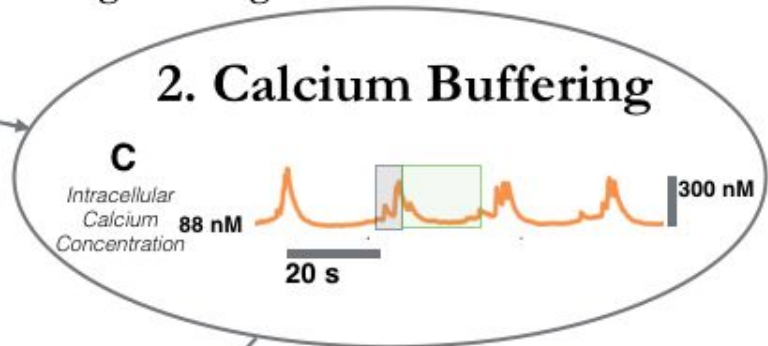


# Spontaneous Bursting Mechanism of SACs

■ Bursting  
■ sAHP

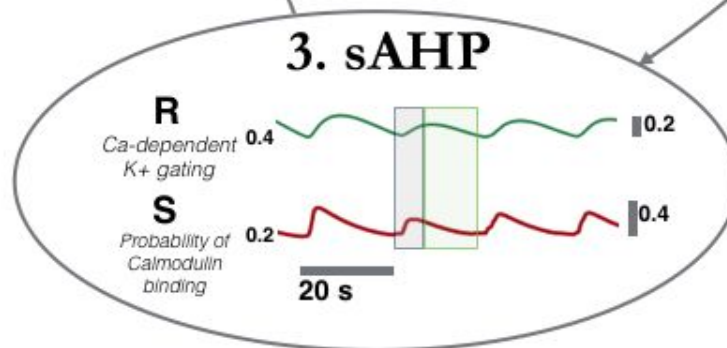


Increase of Calcium load during bursting

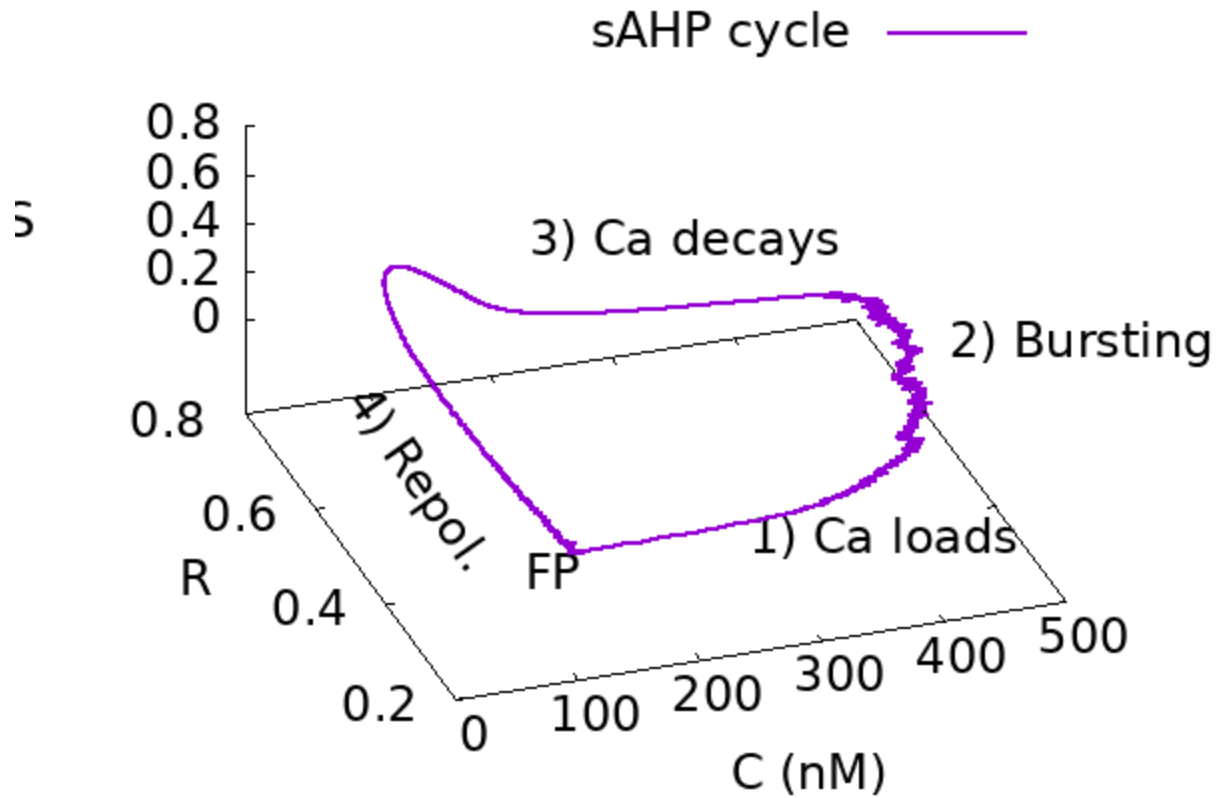


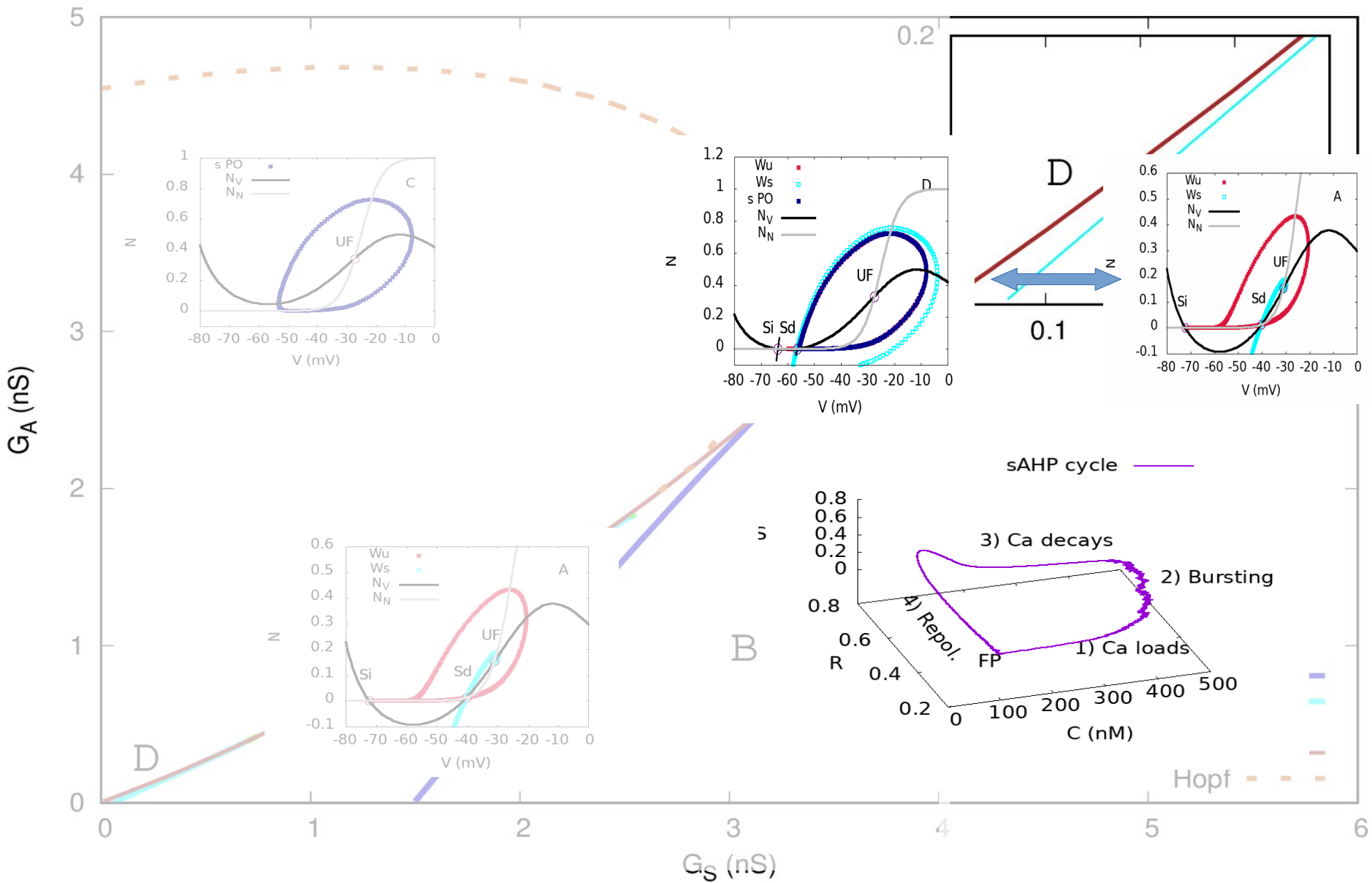
Calcium controls the sAHP phase. Voltage is low and Calcium starts offloading

Bursting starts again when the Calcium load is low enough



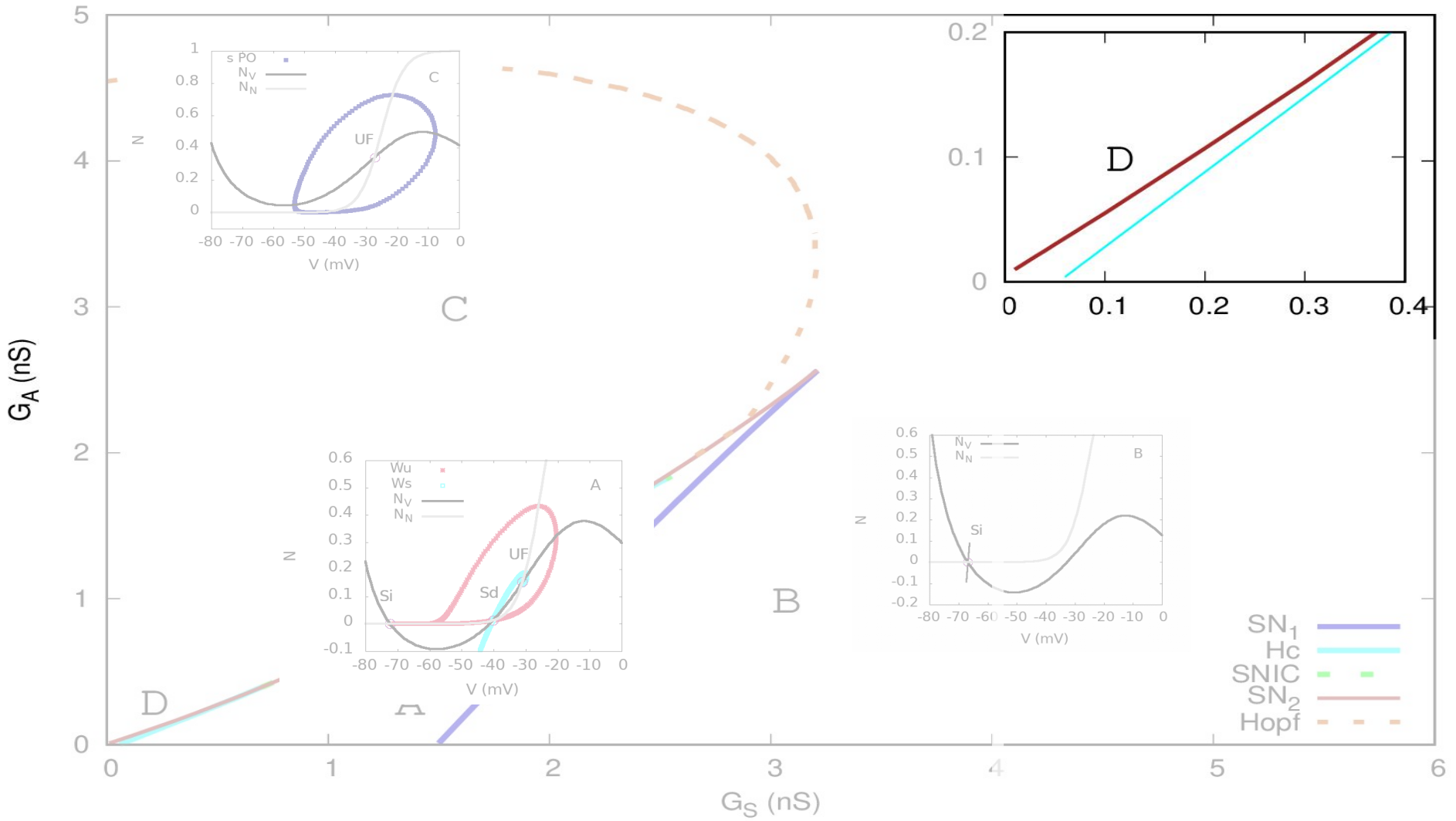
# The sAHP cycle



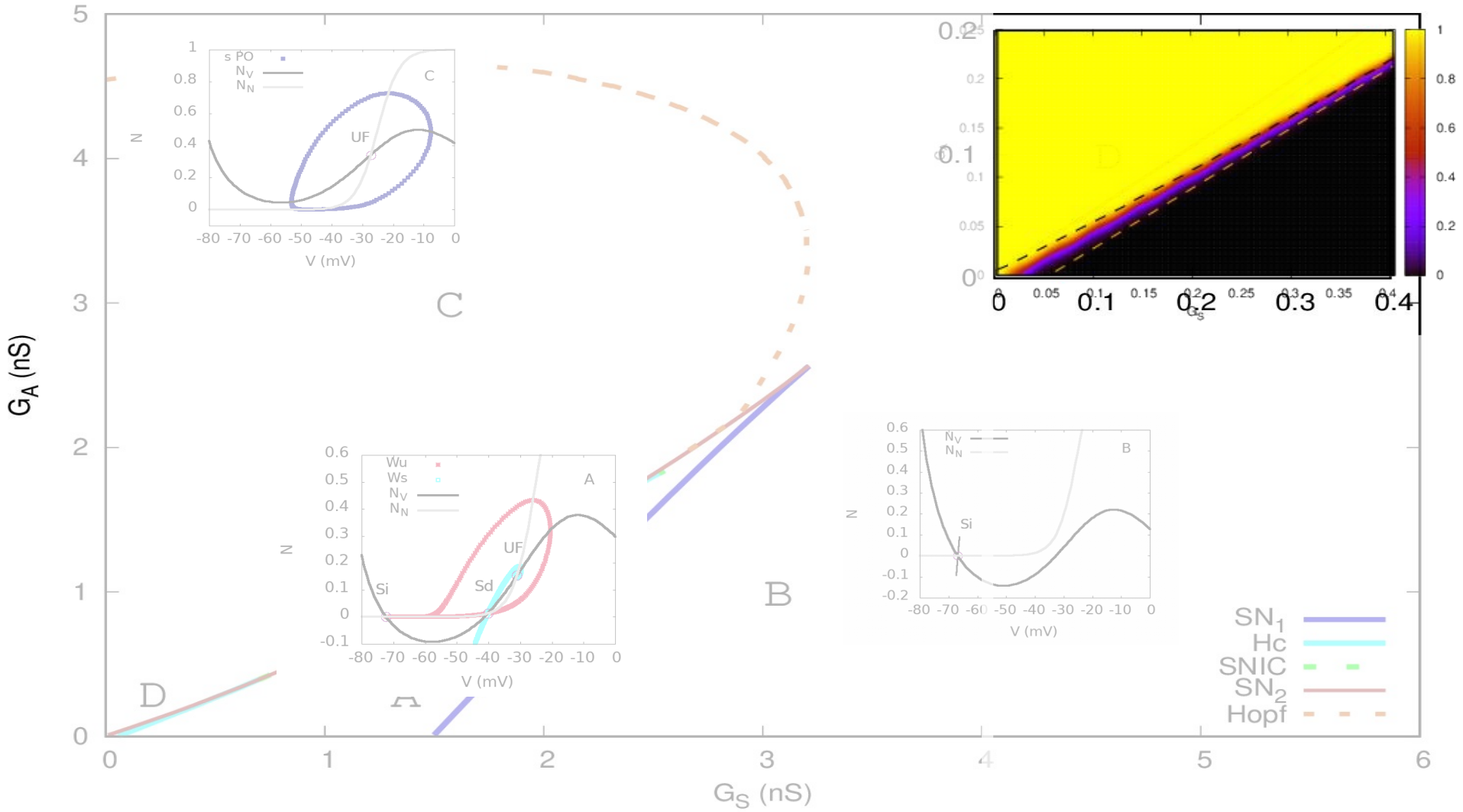




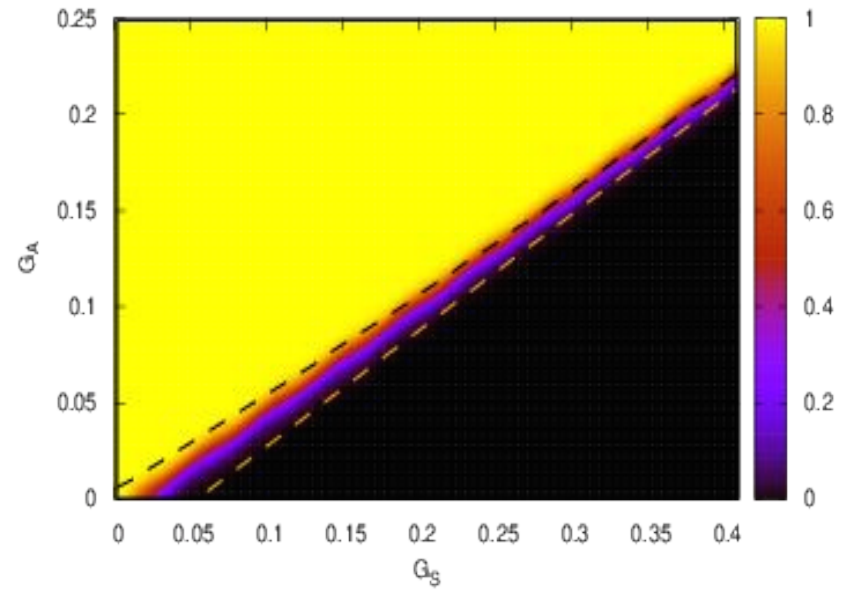
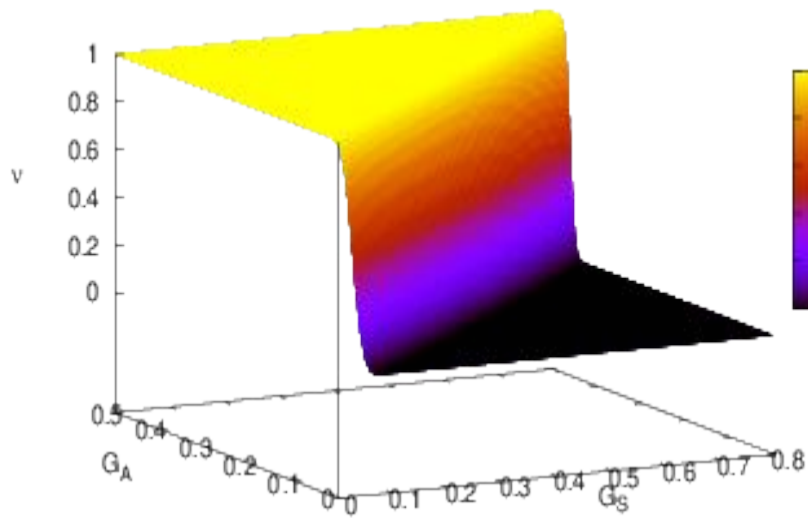
# Probability to trigger a burst



# Probability to trigger a burst



# Probability to trigger a burst

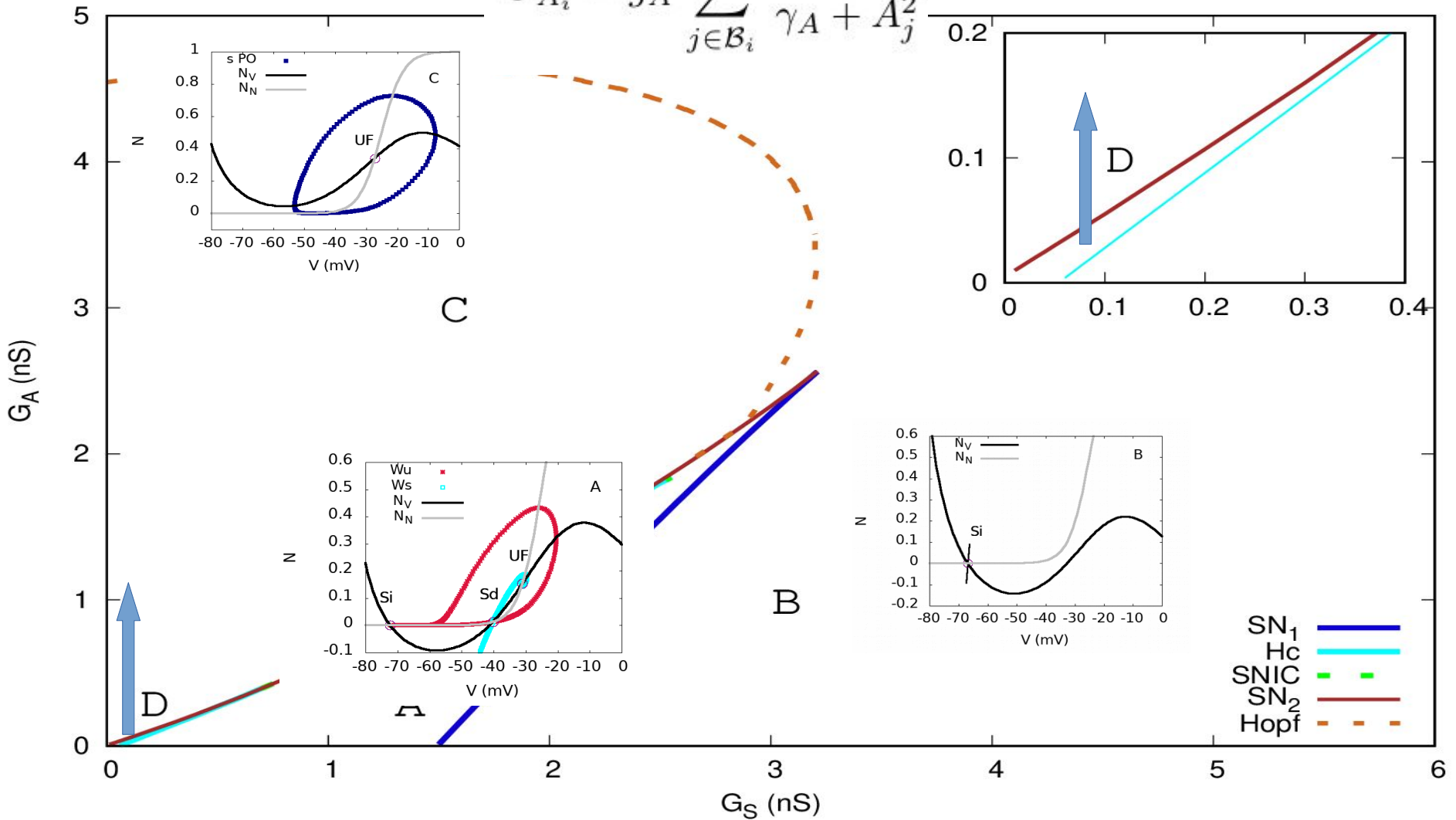


Bursting cells produce Ach

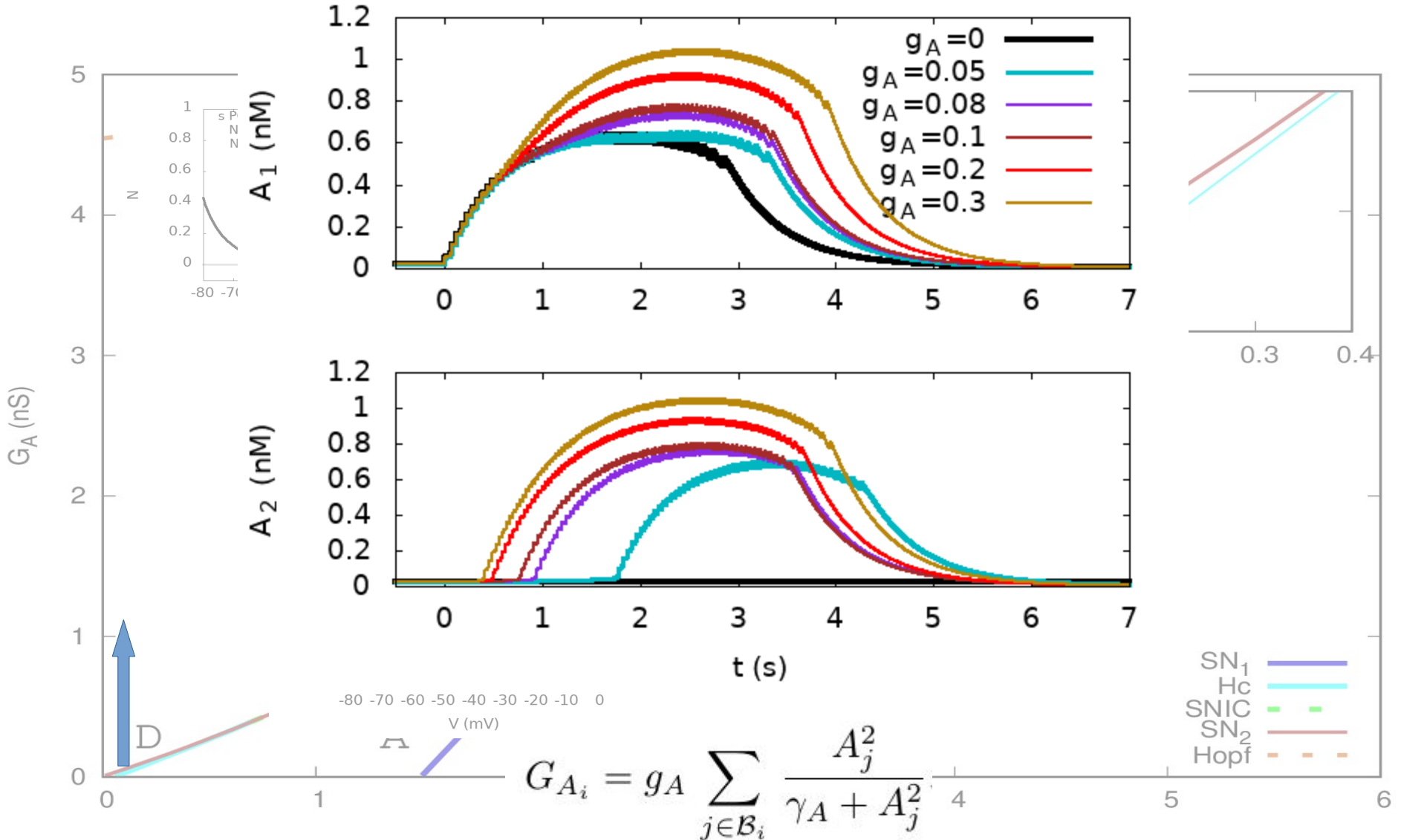
$$G_{A_i} = g_A \sum_{j \in \mathcal{B}_i} \frac{A_j^2}{\gamma_A + A_j^2}$$

# Bursting cells produce Ach

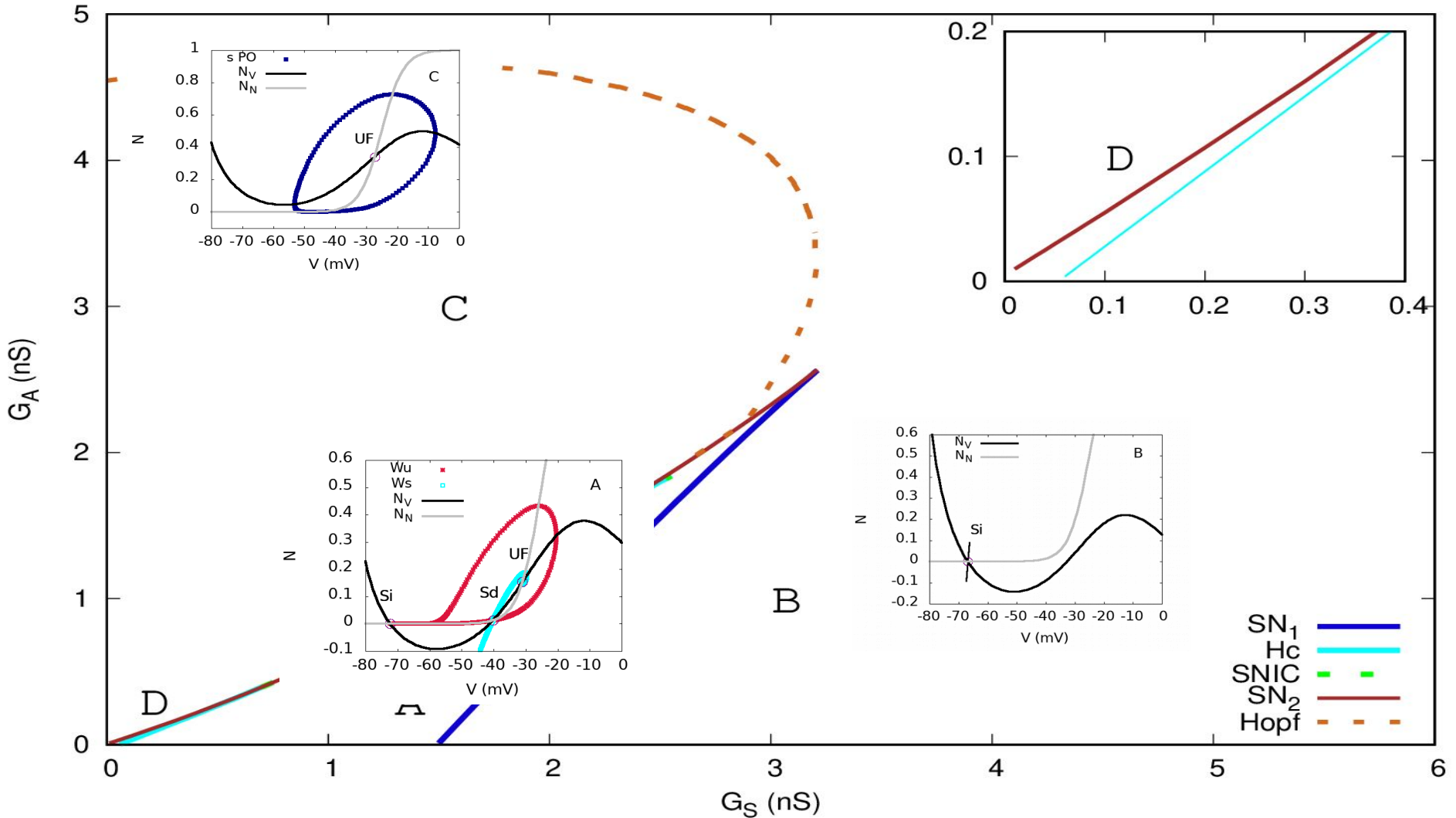
$$G_{A_i} = g_A \sum_{j \in \mathcal{B}_i} \frac{A_j^2}{\gamma_A + A_j^2}$$



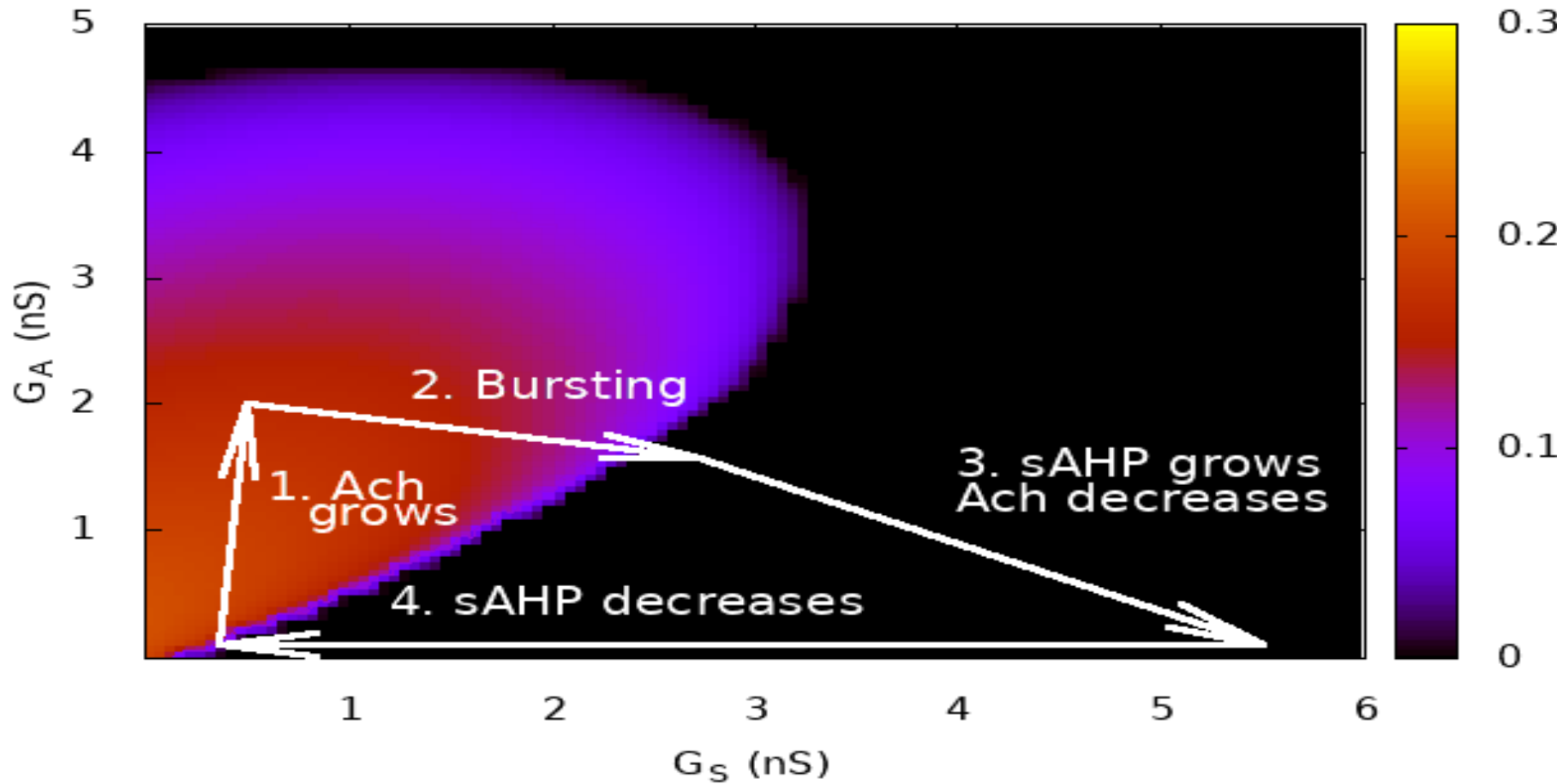
# Bursting cells produce Ach



# The mechanism of retinal waves

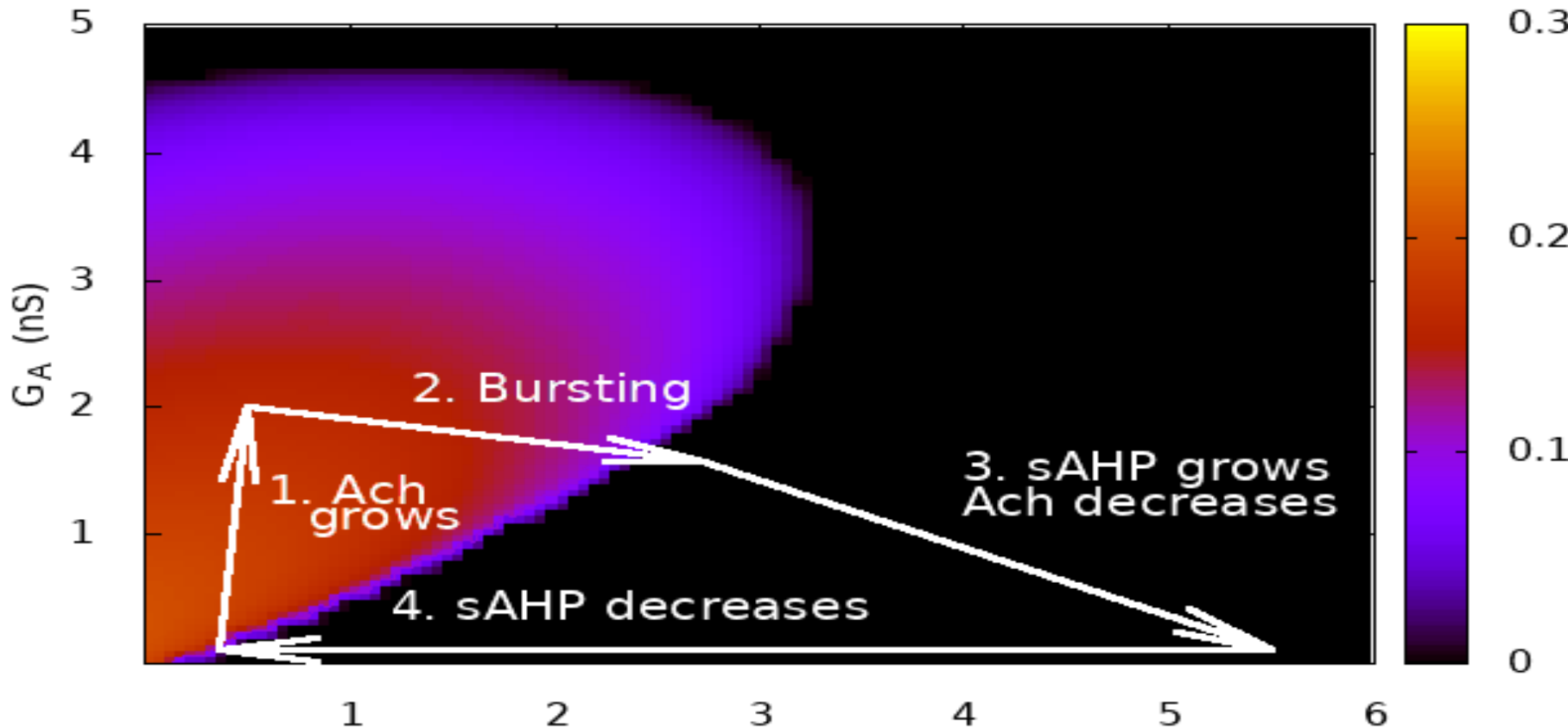


# The mechanism of retinal waves





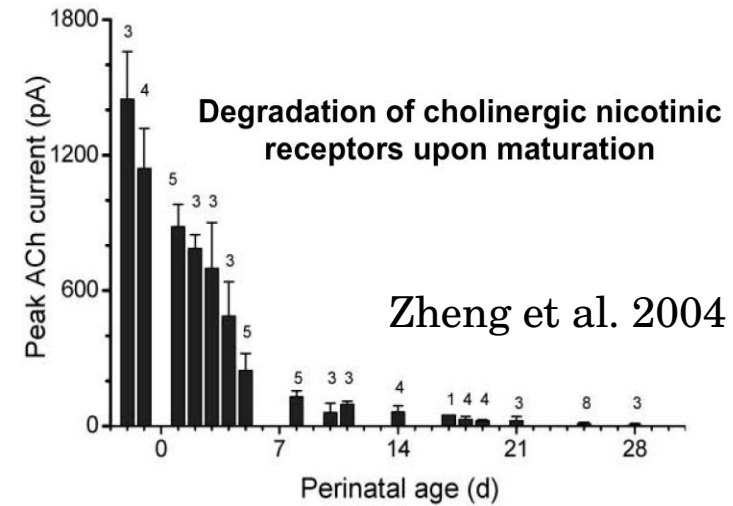
# The mechanism of retinal waves



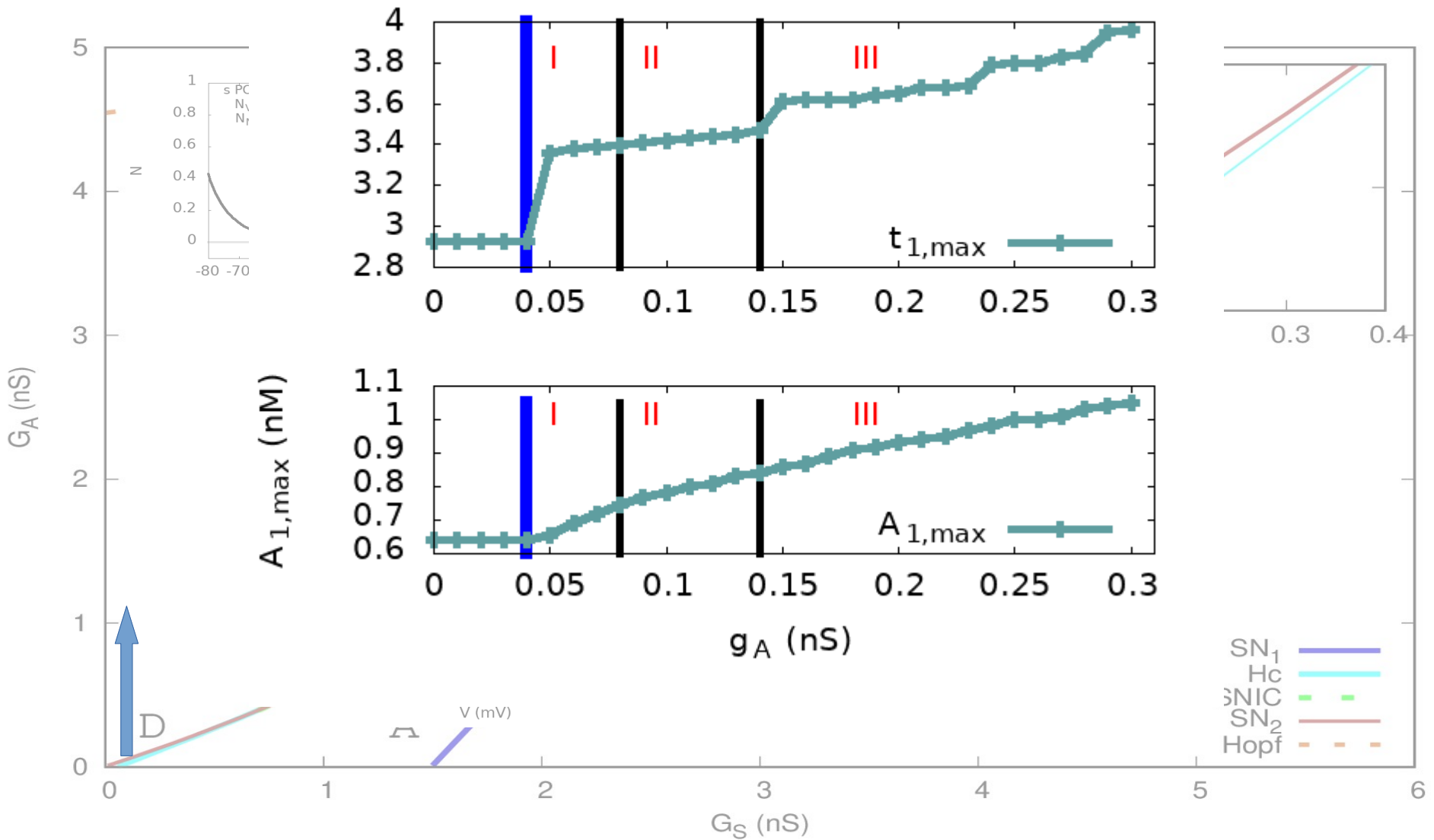
Region D is tiny, but recurrent.

# Variability within retinal waves

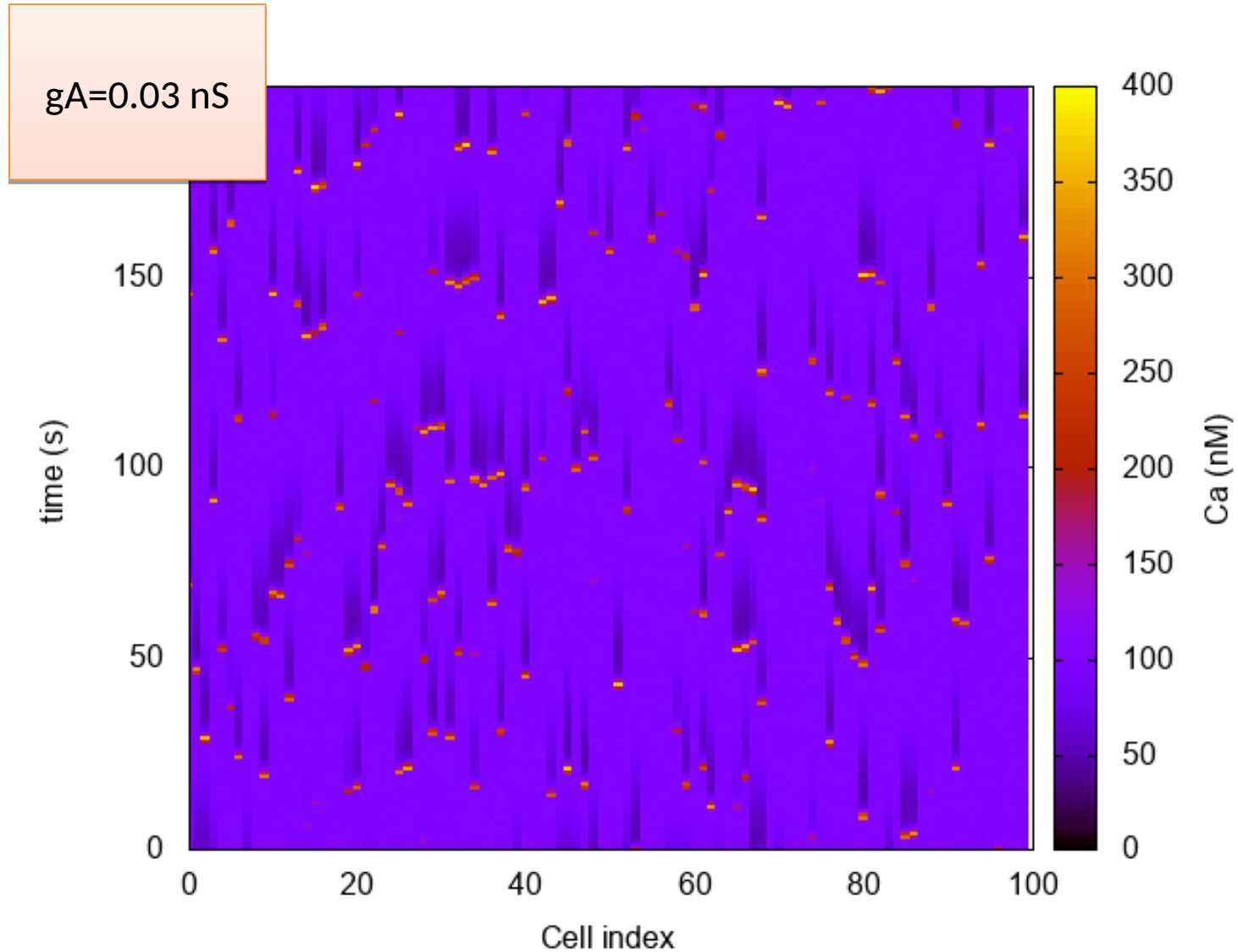
## ii) Development



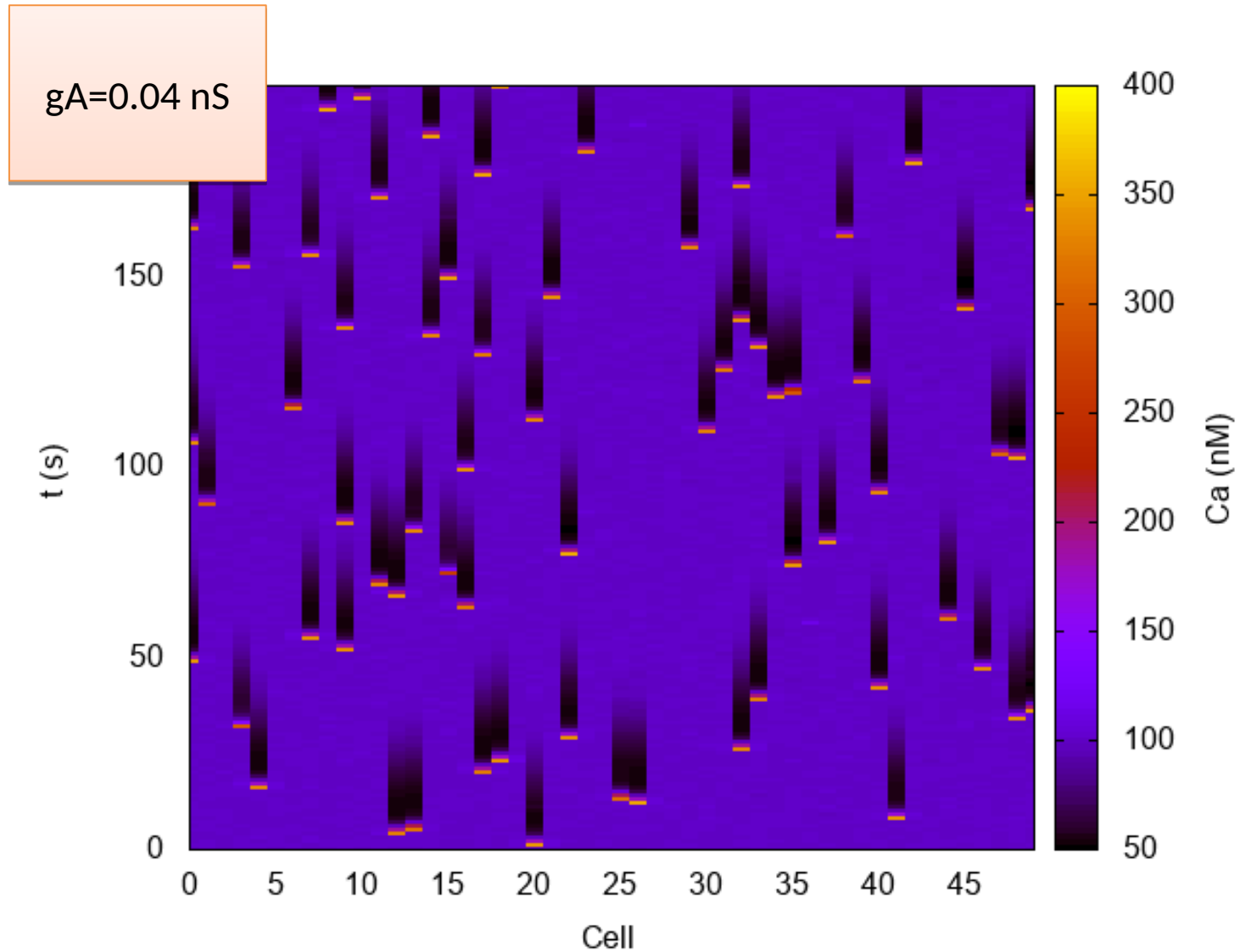
# Non linear coupling and feedback



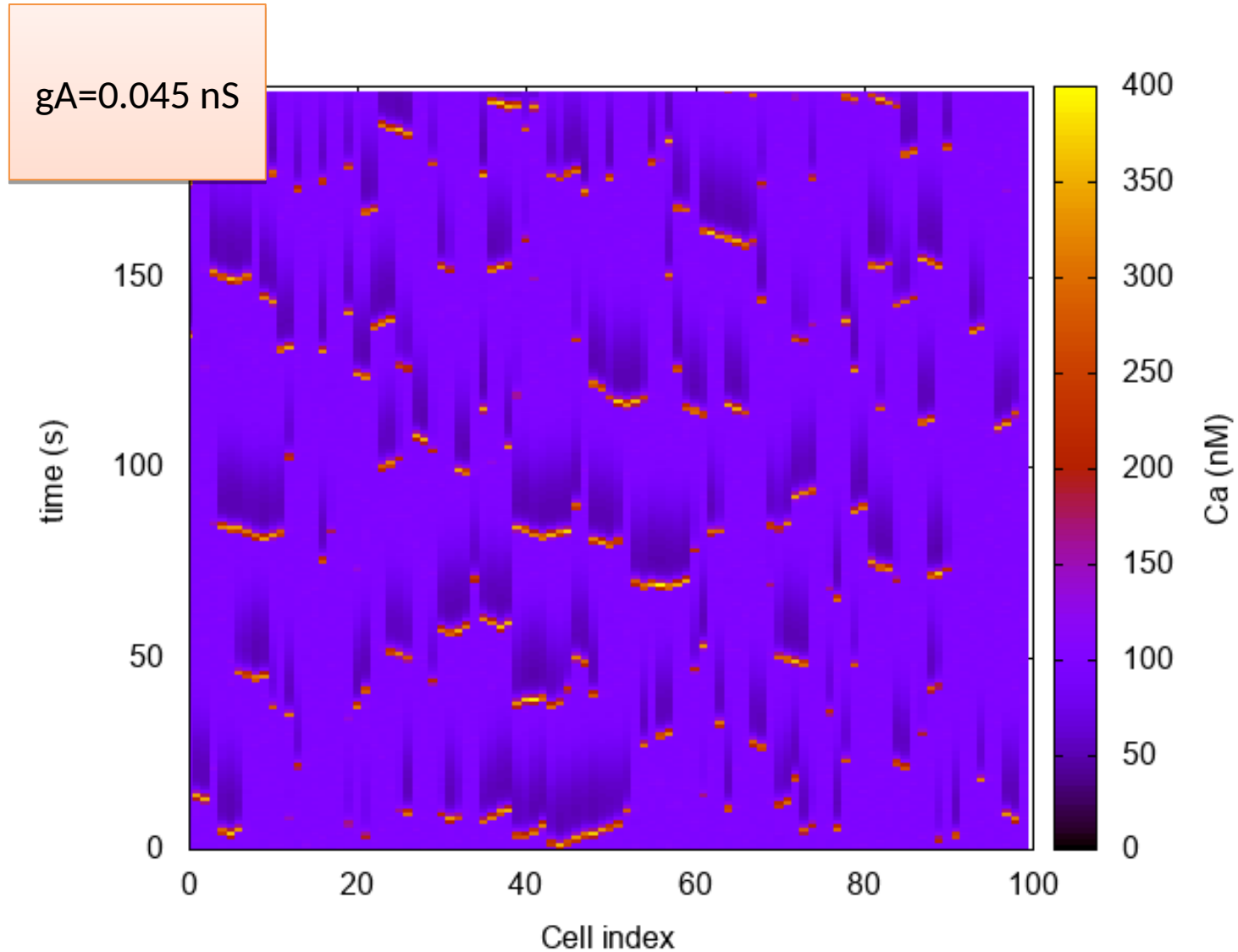
# Waves propagation



# Waves propagation

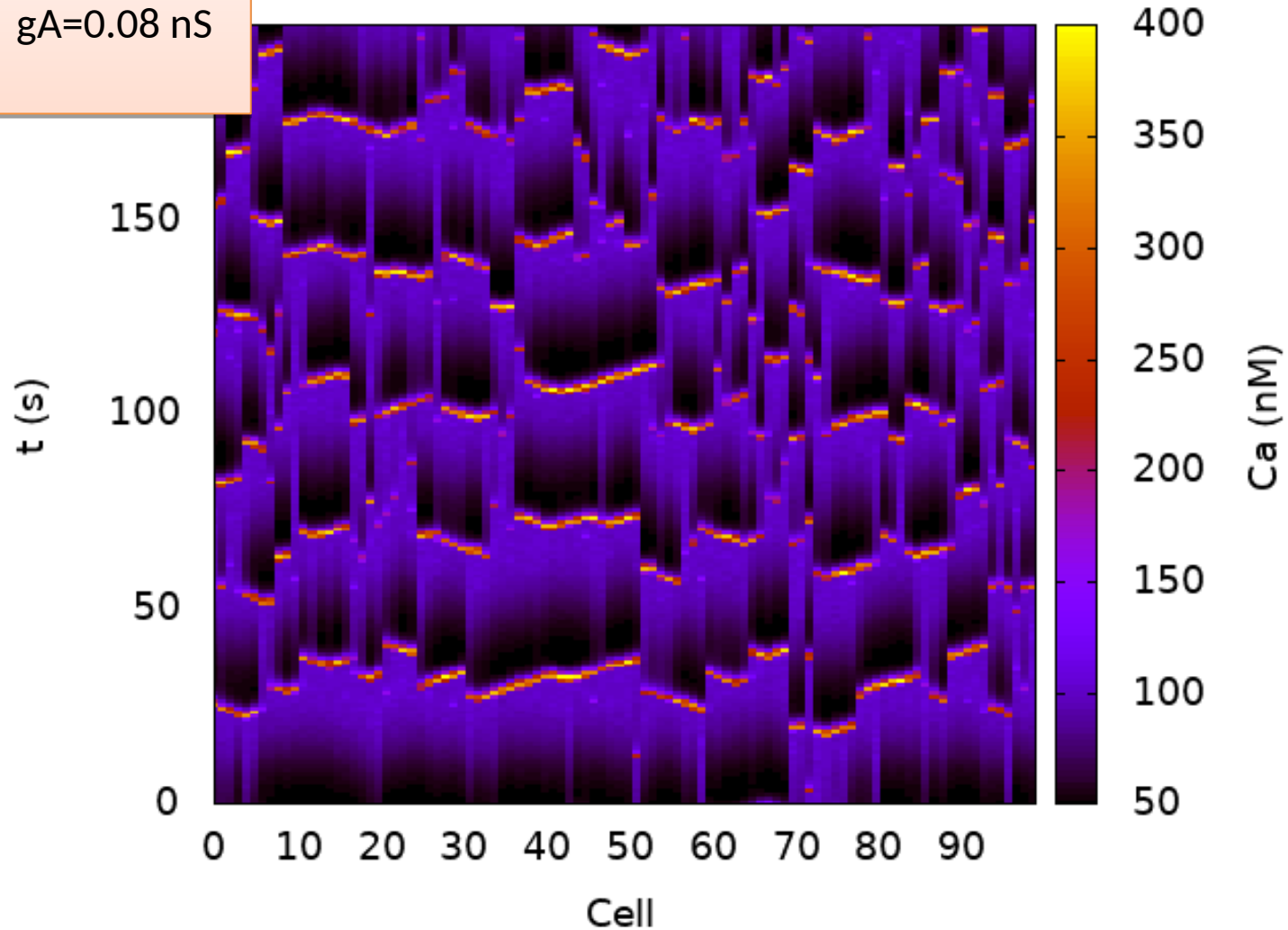


# Waves propagation



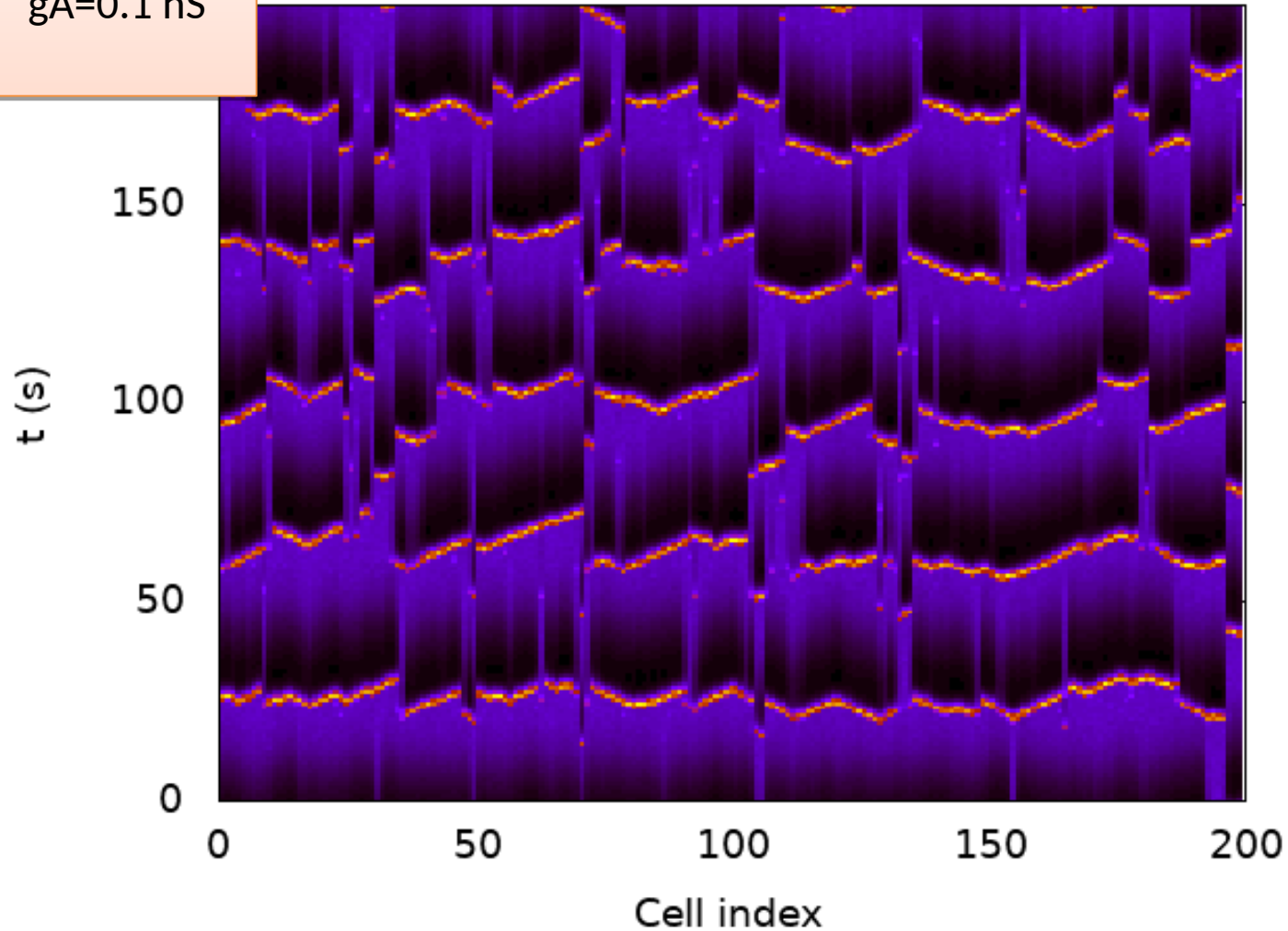
# Waves propagation

$g_A = 0.08 \text{ nS}$



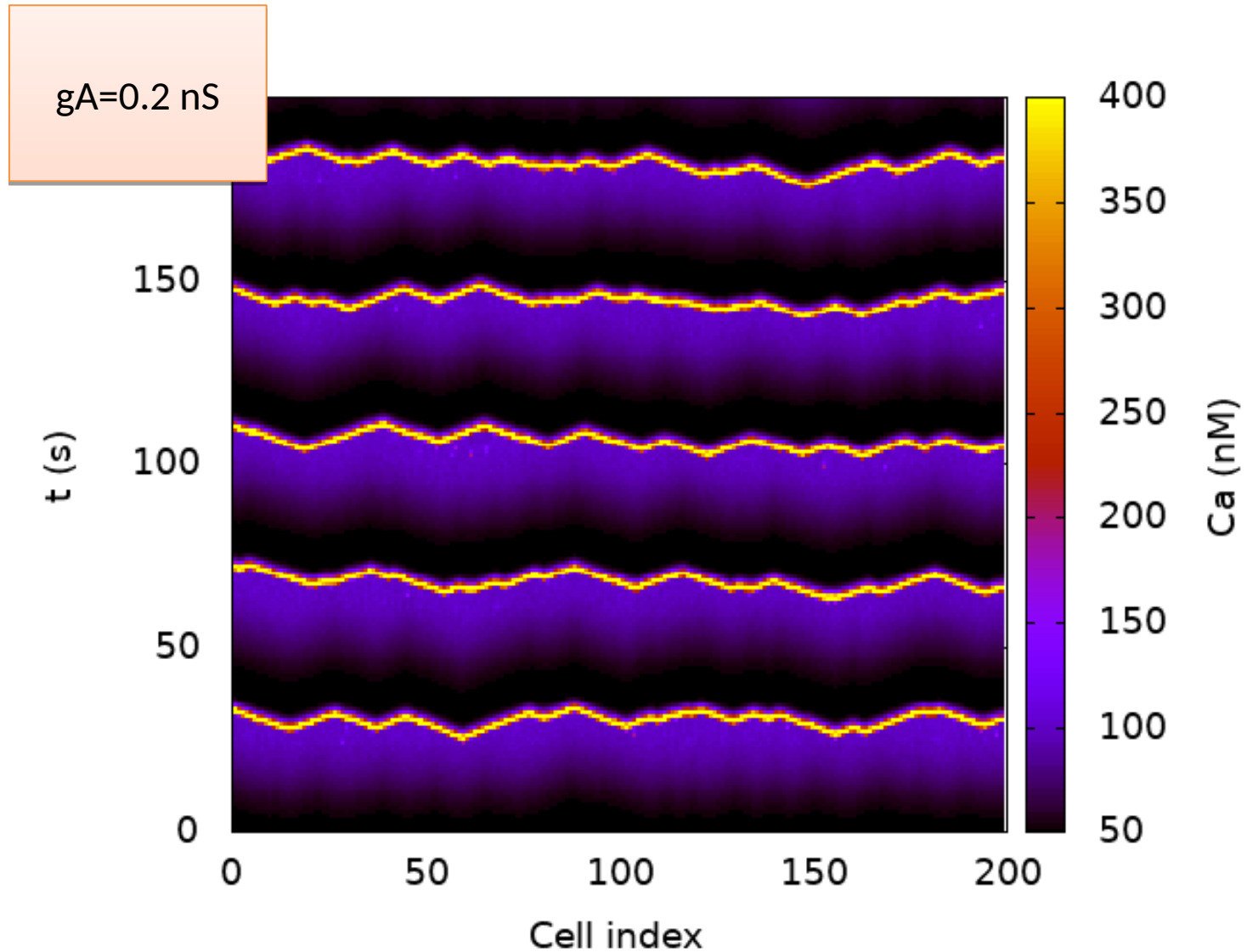
# Waves propagation

$gA=0.1 \text{ nS}$

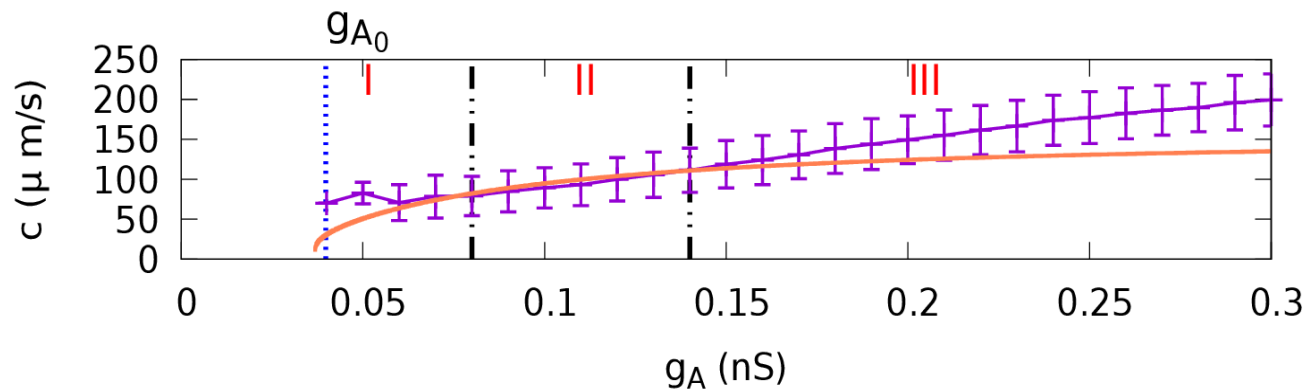
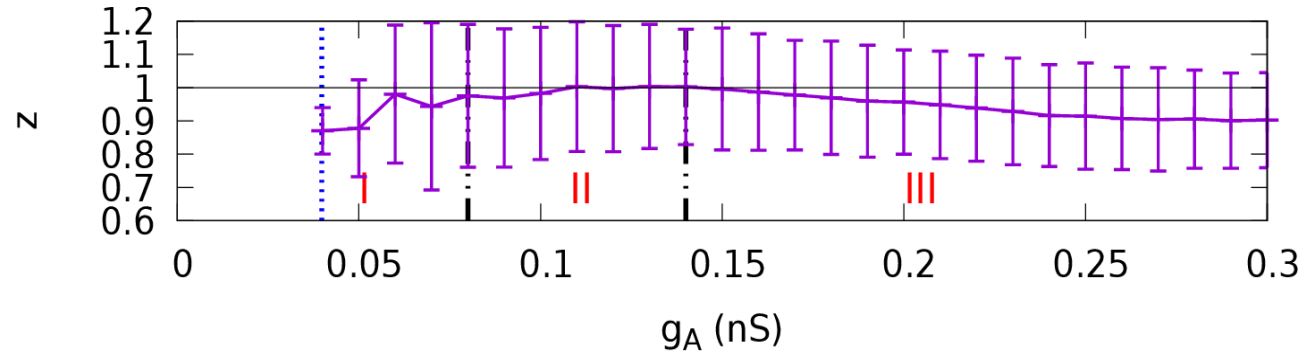




# Waves propagation

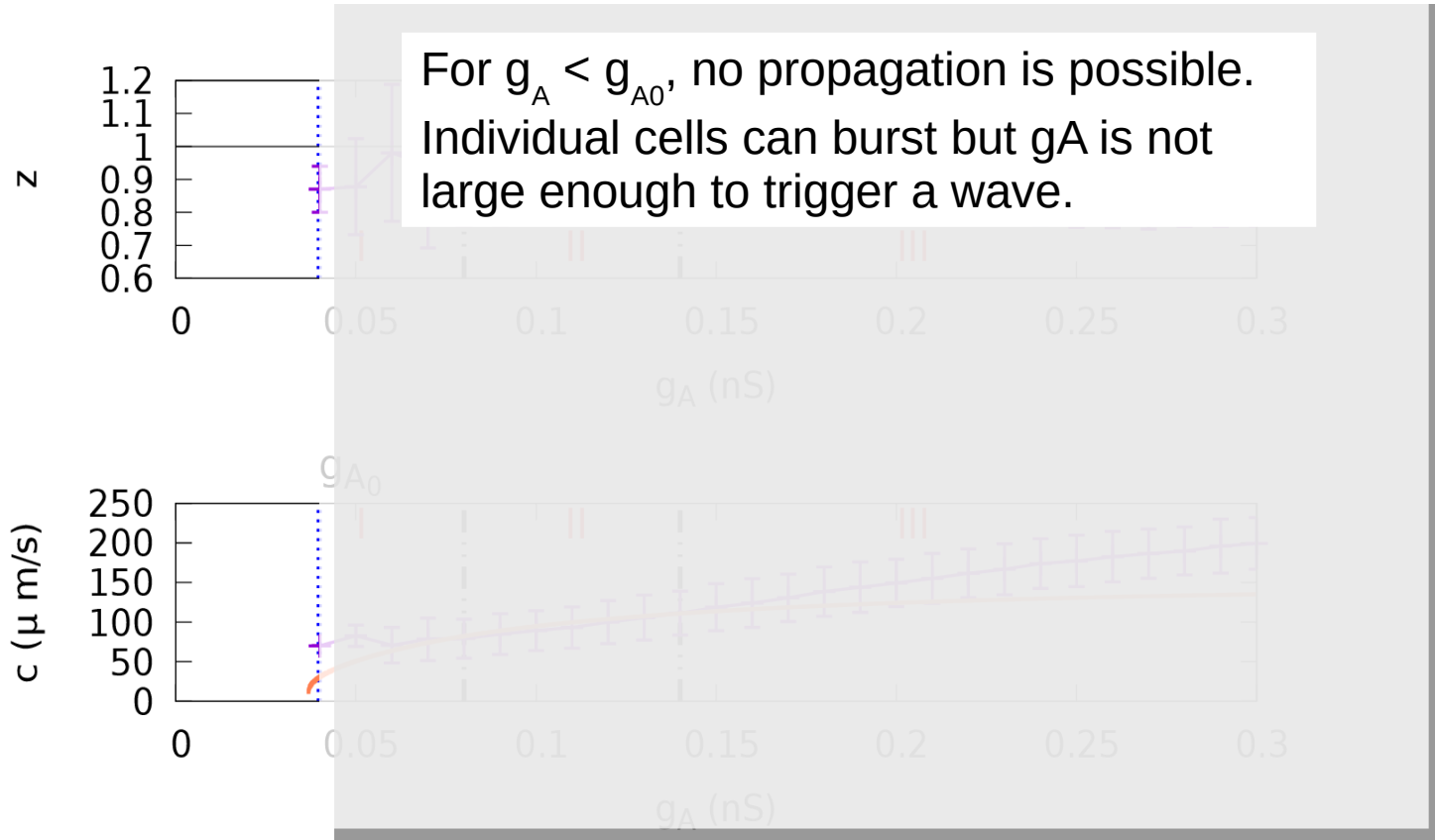


# Regimes



$$r(t) = c.t^z$$

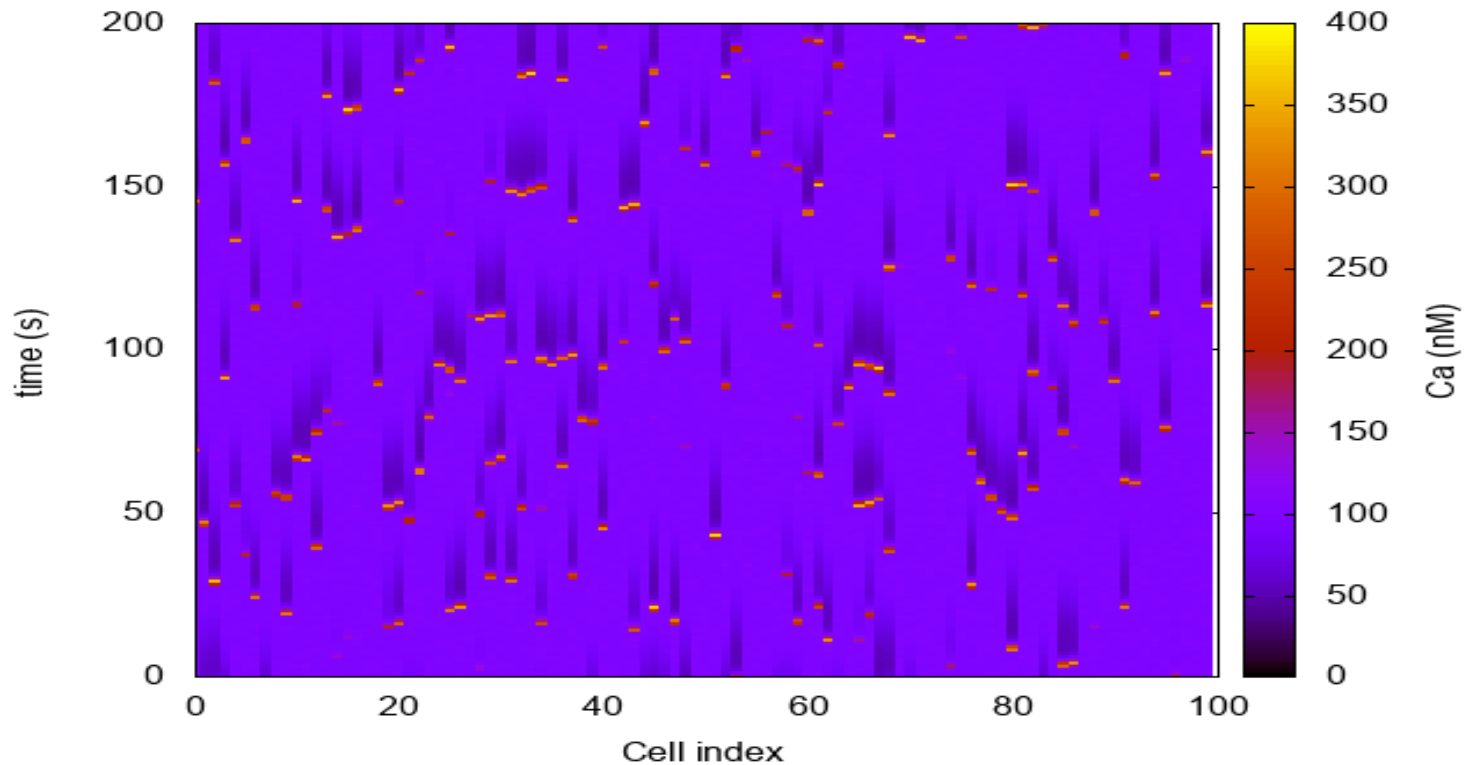
# Regimes



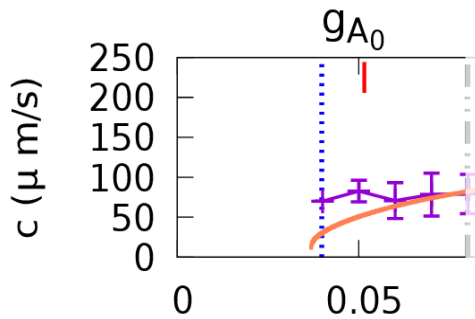
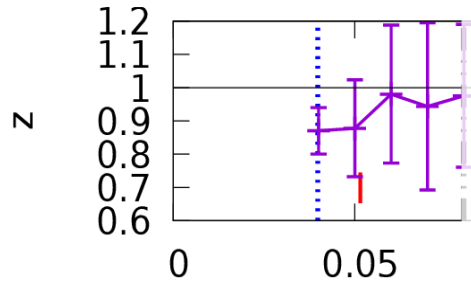
# Regimes

z  
1.2  
1.1  
1.0  
0.9  
0.8  
0.7  
0.6

For  $g_A < g_{A0}$ , no propagation is possible.  
Individual cells can burst but  $g_A$  is not large enough to trigger a wave.

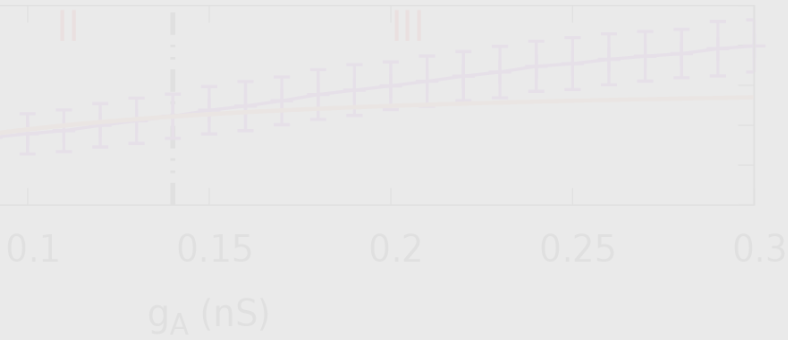


# Regimes

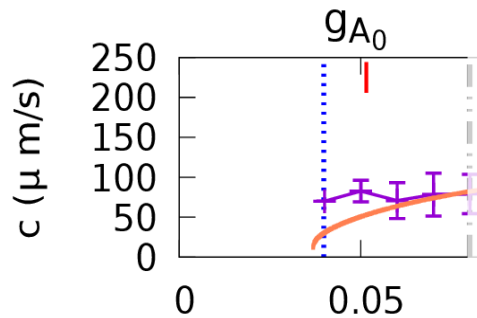
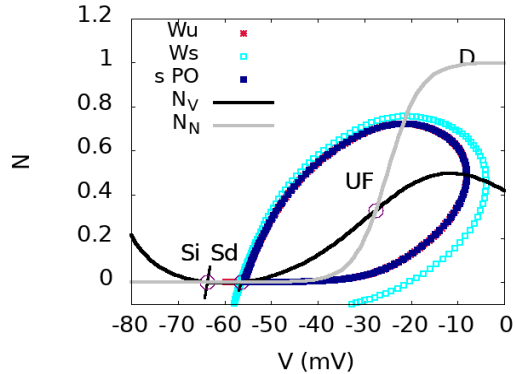


For  $g_A < g_{A0}$ , no propagation is possible. Individual cells can burst but  $g_A$  is not large enough to trigger a wave.

I. A bursting cell can induce bursting of a neighbour, but transition is driven by noise. Transport is sub-ballistic.

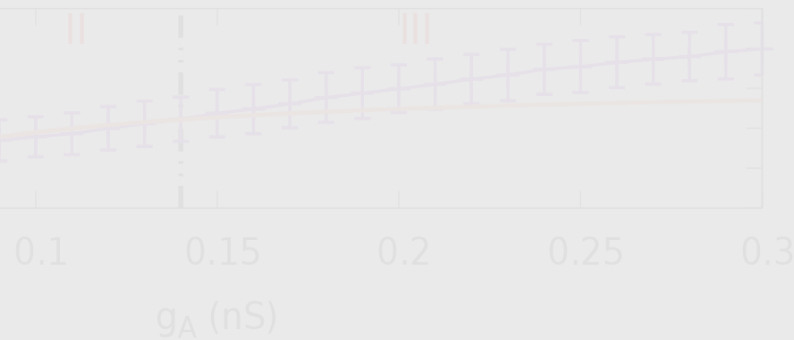


# Regimes

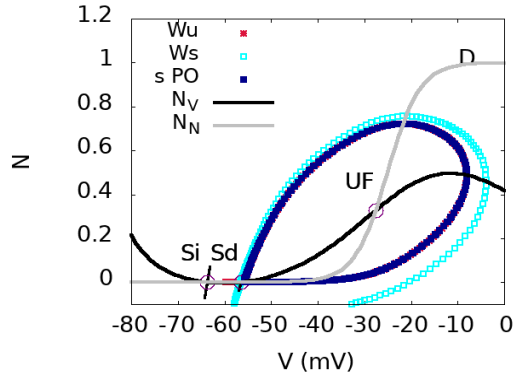


For  $g_A < g_{A_0}$ , no propagation is possible. Individual cells can burst but  $g_A$  is not large enough to trigger a wave.

I. A bursting cell can induce bursting of a neighbour, but transition is driven by noise. Transport is sub-ballistic.

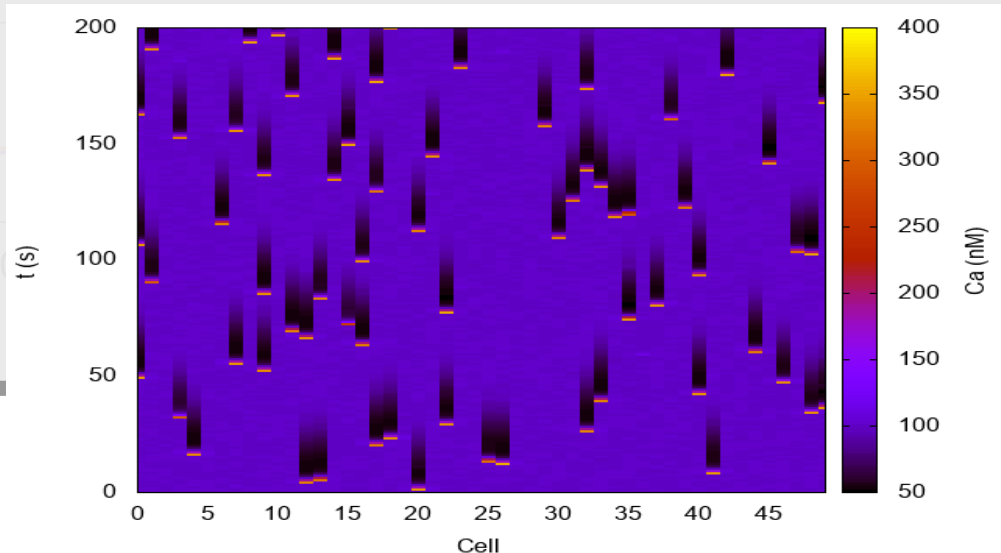
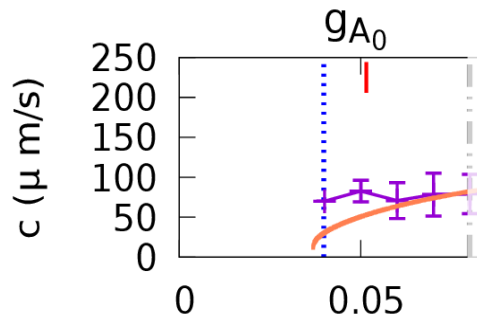


# Regimes

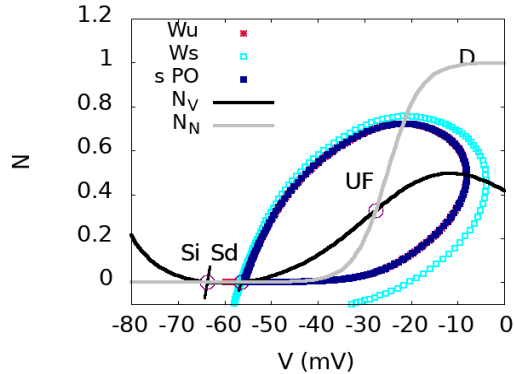


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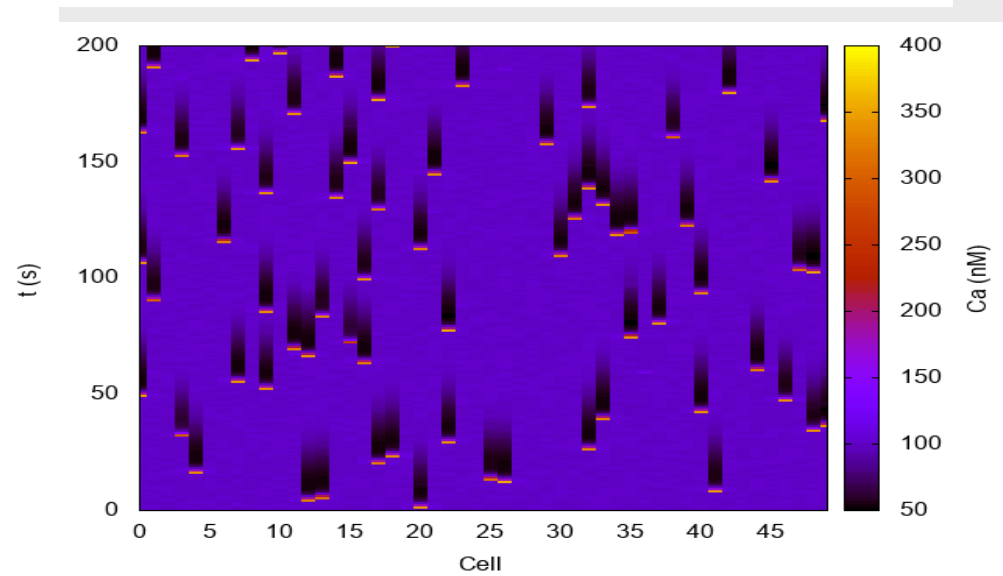
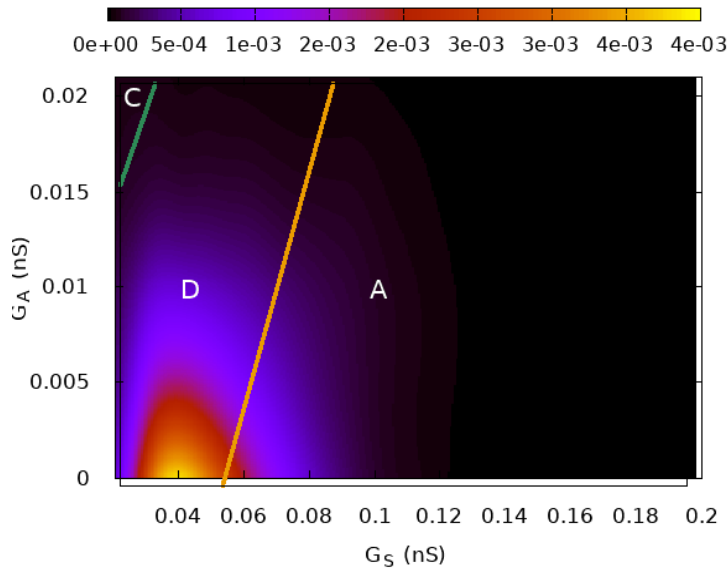


# Regimes



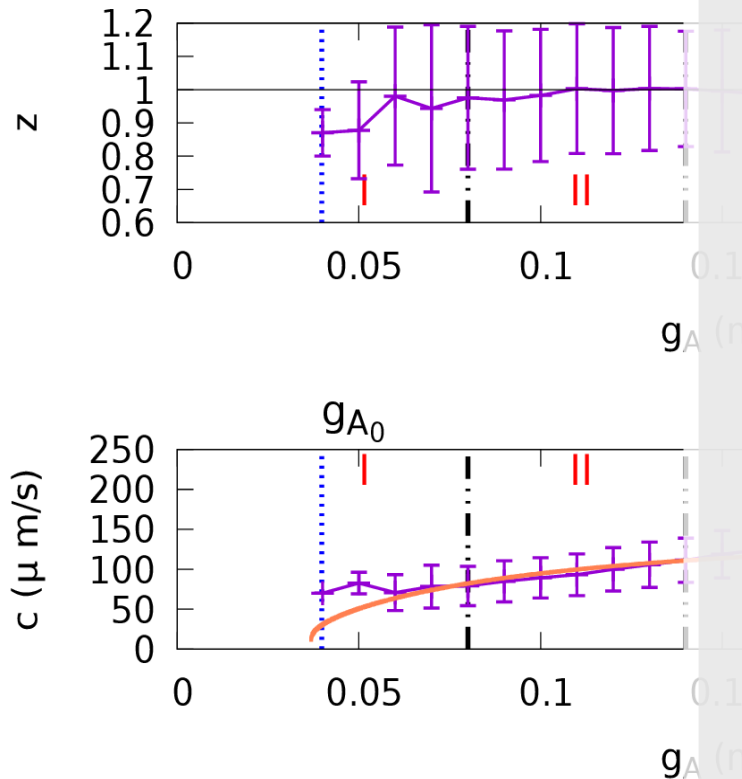
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I. A bursting cell can induce bursting of a neighbour, but transition is driven by noise. Transport is sub-ballistic.





# Regimes

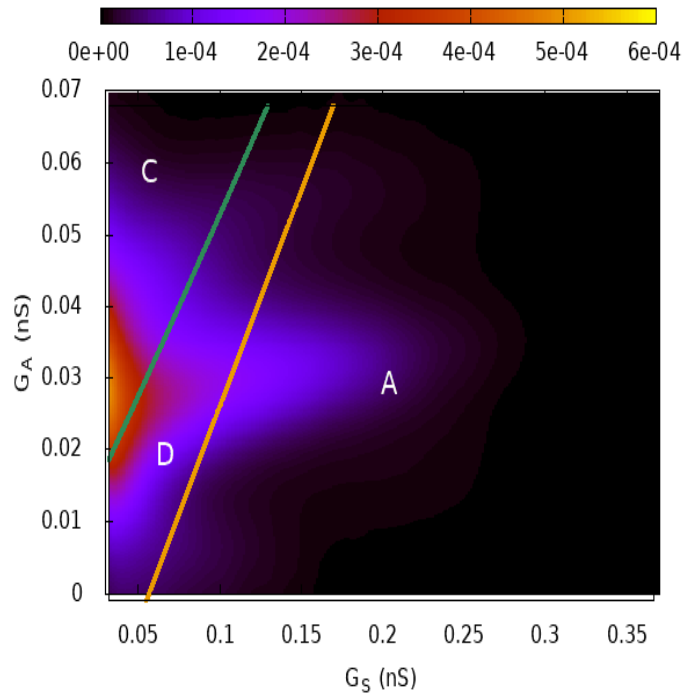


For  $g_A < g_{A_0}$ , no propagation is possible. Individual cells can burst but  $g_A$  is not large enough to trigger a wave.

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II. Bursting cells can make their neighbours burst. Transport is ballistic. Waves collide at sAHP barriers.

# Regimes



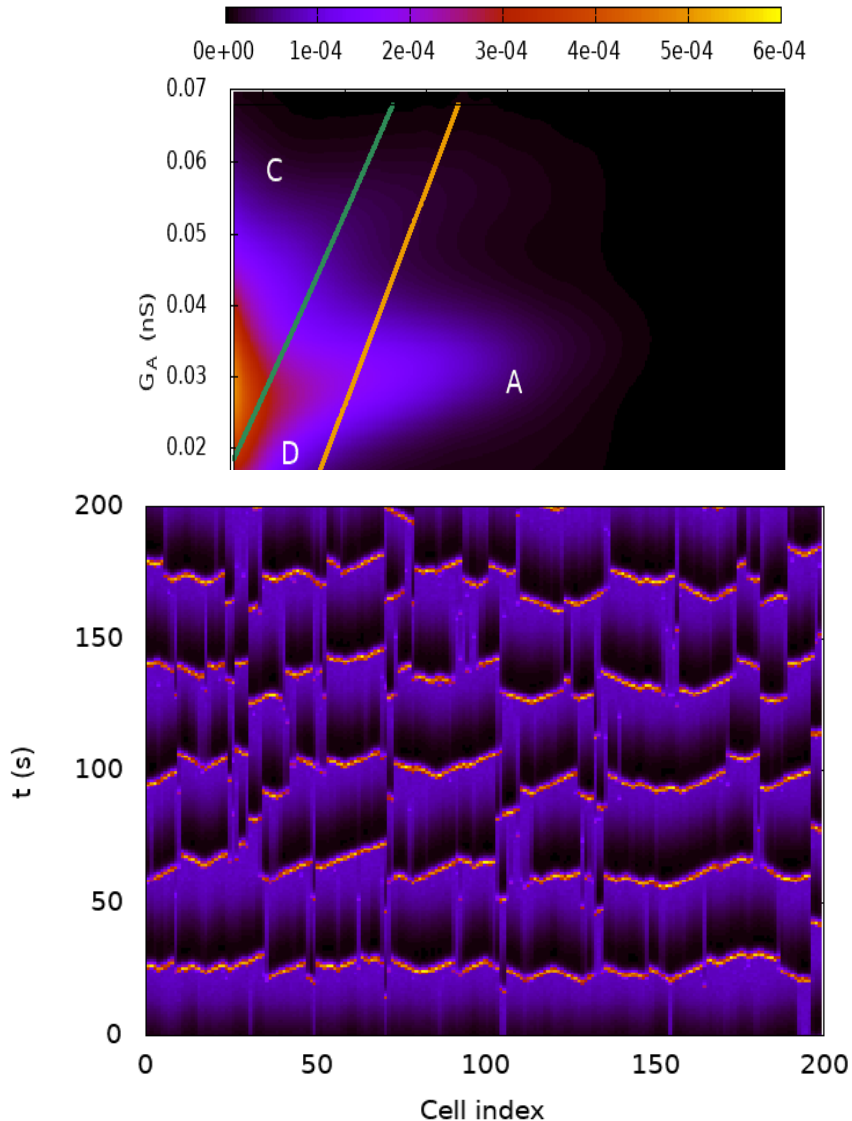
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$g_A$  (nS)

# Regimes



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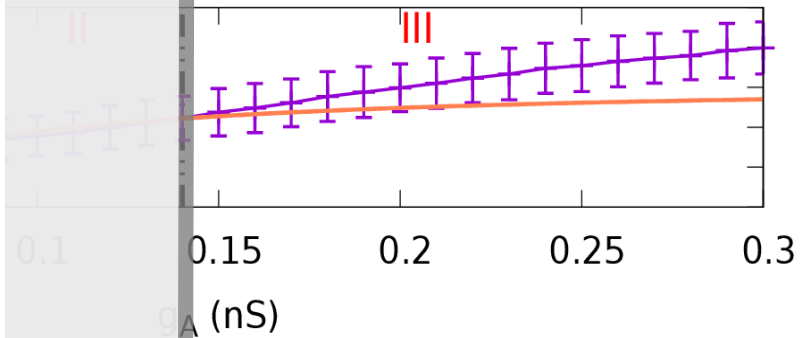
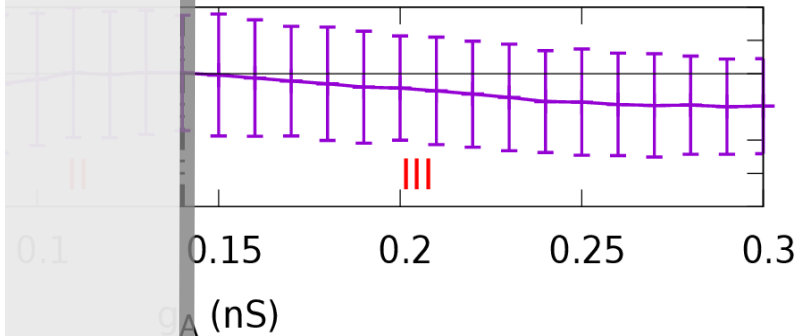
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III. Bursting cells can make their neighbours burst. Transport is ballistic. Two interacting waves cooperate.



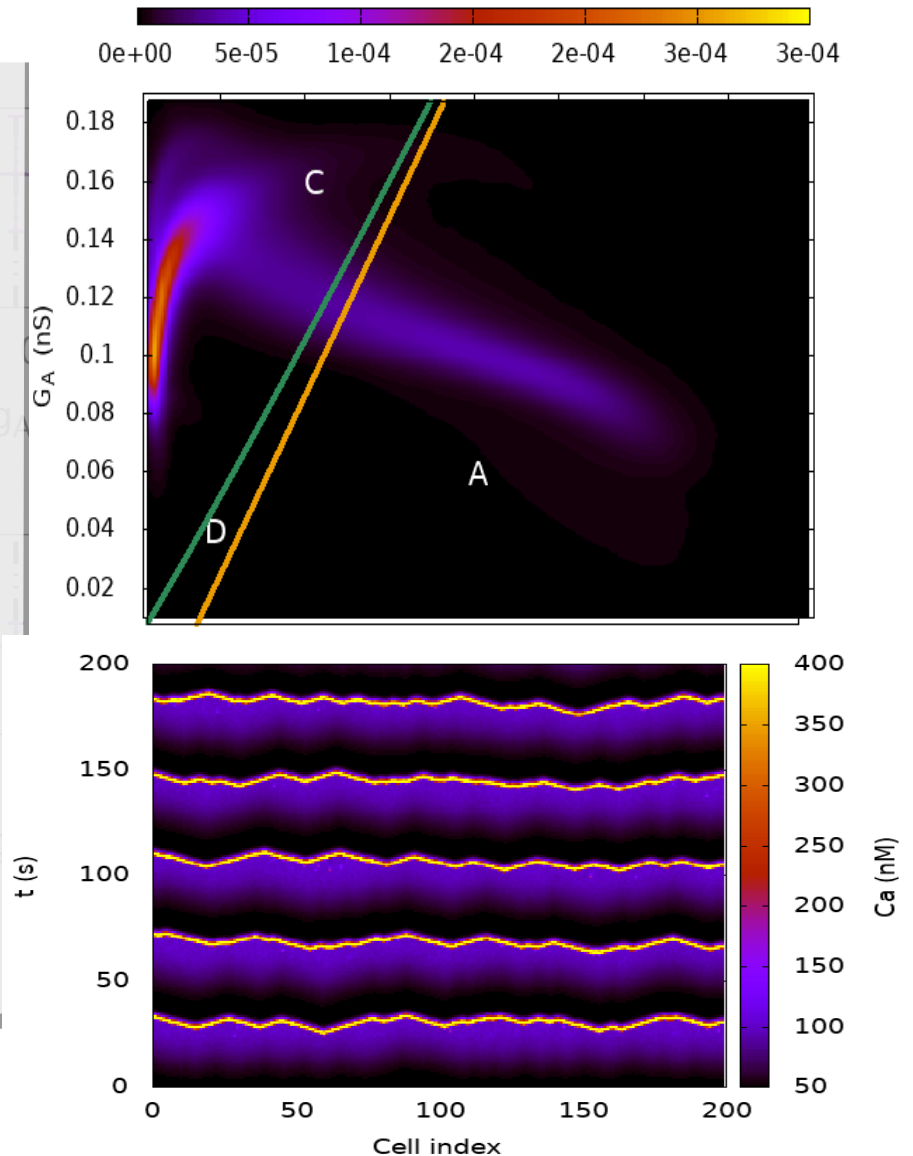
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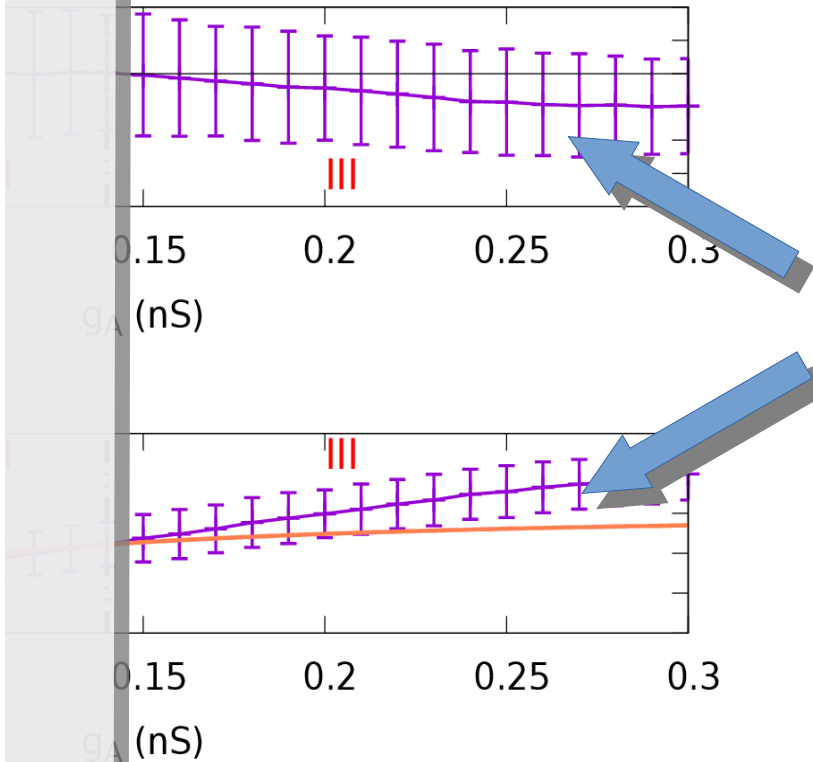
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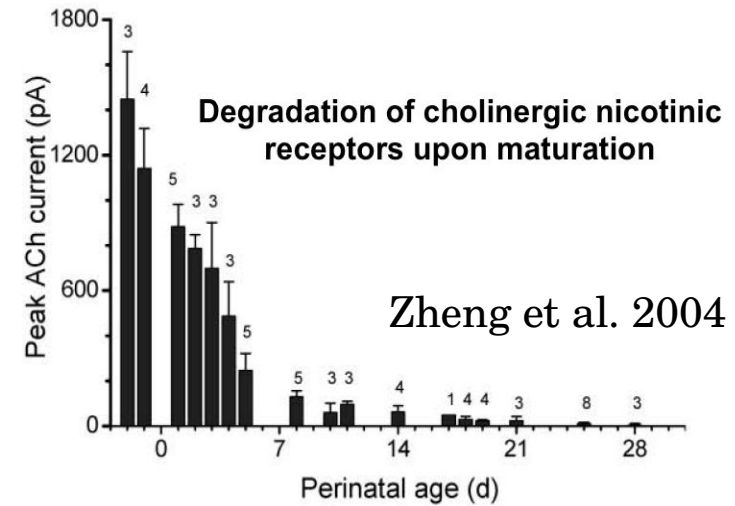
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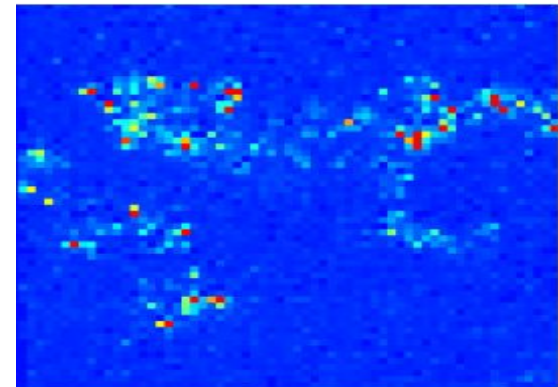


# Variability within retinal waves

## ii) Development

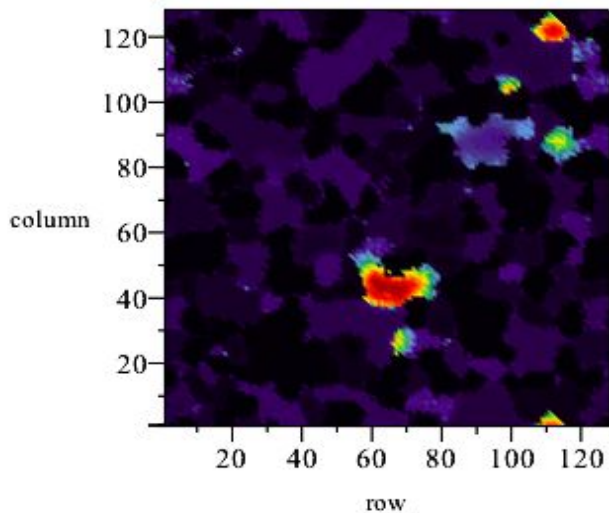


## iv) Spatial Variability



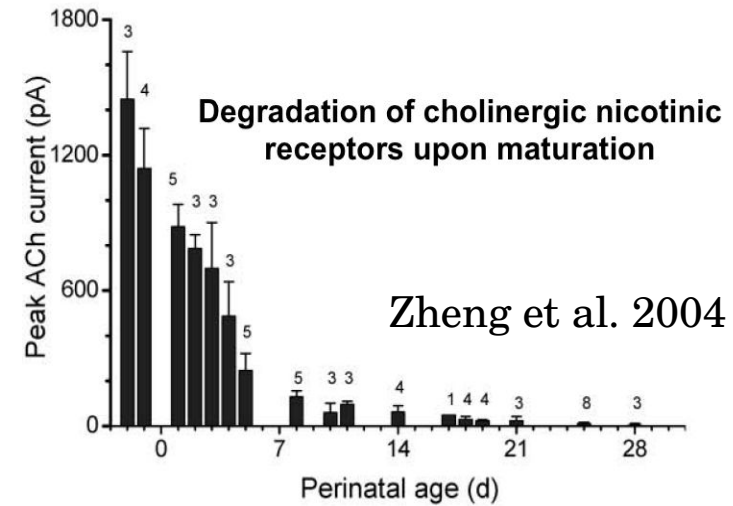
# Variability within retinal waves

Simulations made by L. Gil

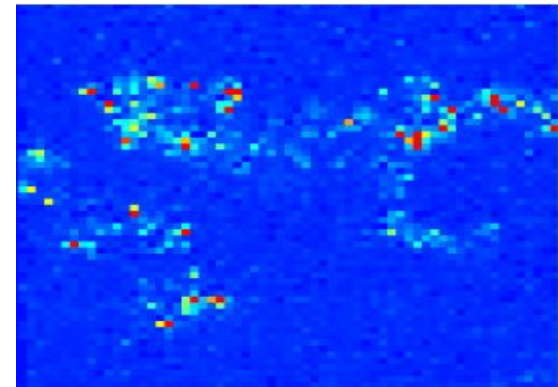


<https://www.youtube.com/watch?v=shMR3NMCBDE>

## ii) Development



## iv) Spatial Variability





# A transport theory of retinal waves

1. The leading parameters are the Ach conductance  $G_A$  and the sAHP conductance  $G_c$ .

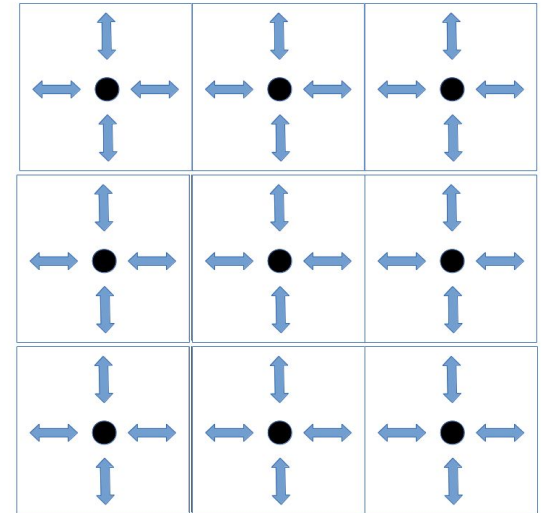
# A transport theory of retinal waves

1. The leading parameters are the Ach conductance  $G_A$  and the sAHP conductance  $G_S$ .
  2. Waves history generates transient refractory zones of sAHP which constrain the forthcoming waves propagation.
-

# A transport theory of retinal waves

## Assume:

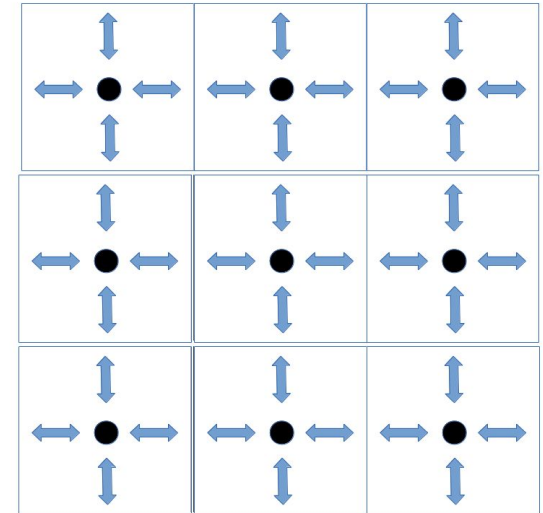
- Nearest neighbours interaction;
- Continuous space limit ;
- GS, GA are considered as spatio-temporal fields ;
- Mean-field approximation to Ach production



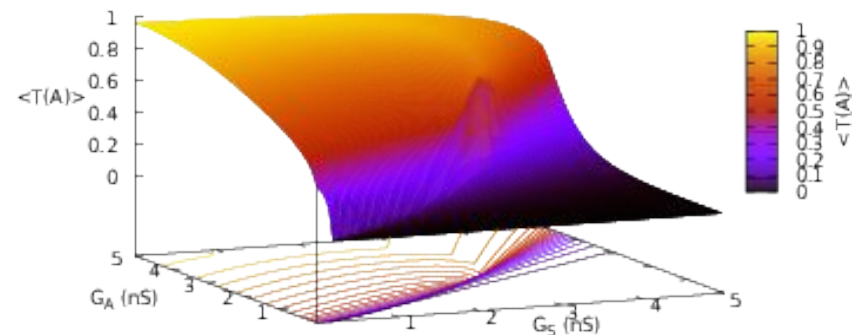
# A transport theory of retinal waves

## Assume:

- Nearest neighbours interaction;
- Continuous space limit ;
- $G_S$ ,  $G_A$  are considered as spatio-temporal fields ;
- Mean-field approximation to Ach production



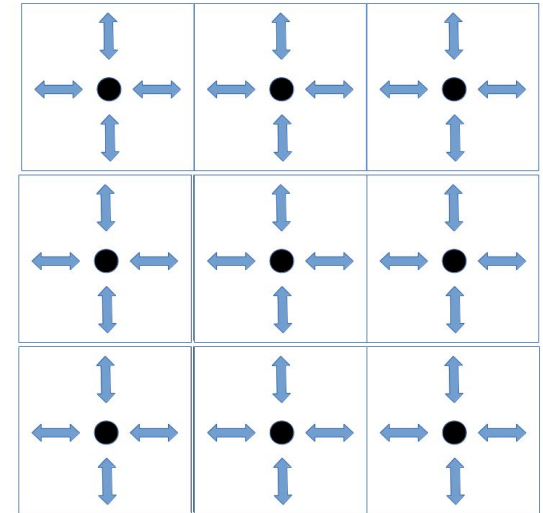
$$\langle T_A(V) \rangle \equiv \Omega(G_S, G_A)$$



# A transport theory of retinal waves

## Assume:

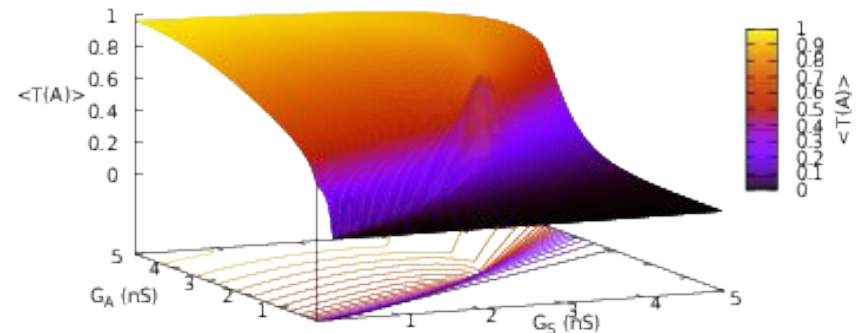
- Nearest neighbours interaction;
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- Mean-field approximation to Ach production



$$\langle T_A(V) \rangle \equiv \Omega(G_S, G_A)$$

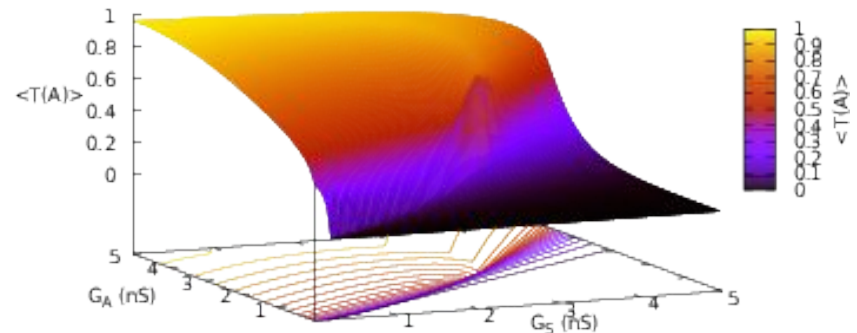
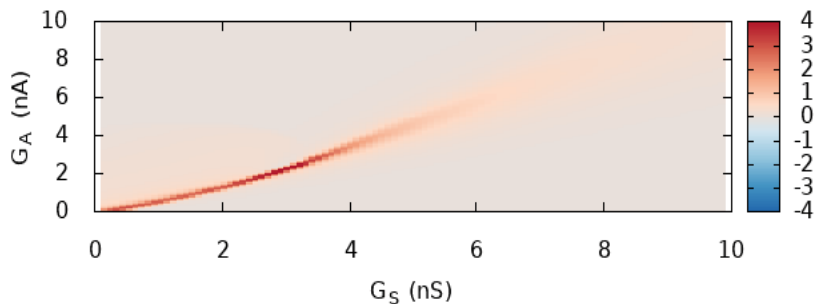
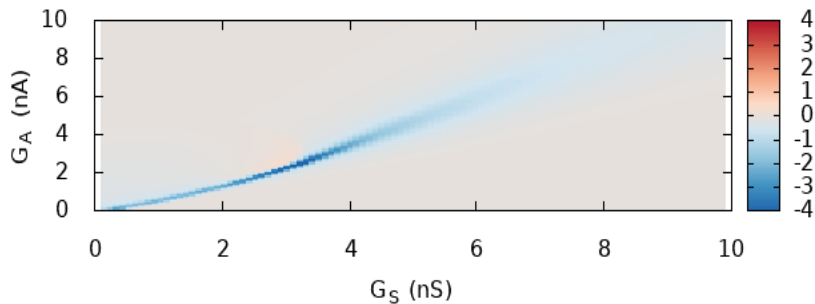
$$\frac{\partial G_A}{\partial t} = g_A D_A \Delta \Omega + S.$$

$$S = -\mu_A G_A + g_A \frac{d\beta_A}{\sqrt{\gamma_A}} \Omega$$



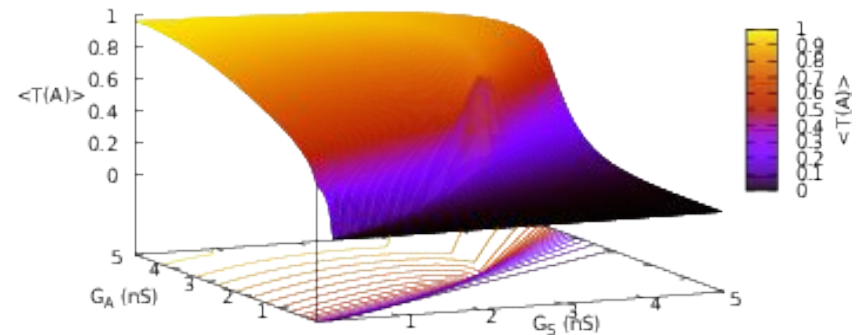
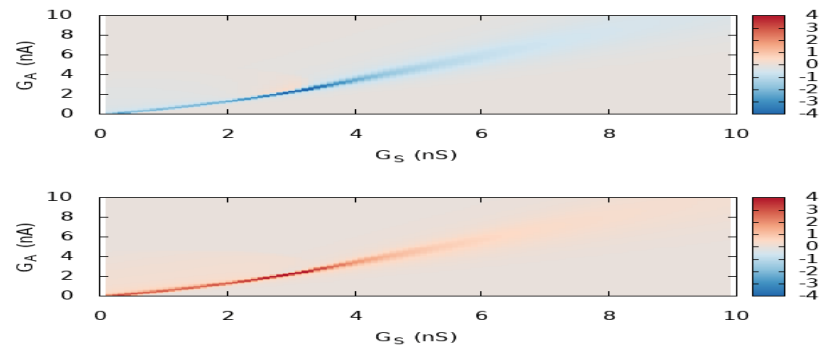
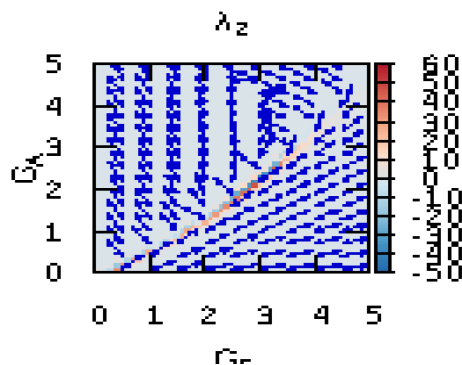
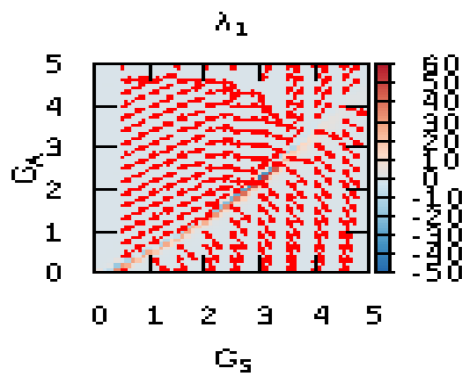
# A transport theory of retinal waves

$$\frac{\partial G_A}{\partial t} = g_A D_A \left[ (\nabla G_S, \nabla G_A) H_\Omega \begin{pmatrix} \nabla G_S \\ \nabla G_A \end{pmatrix} + \frac{\partial \Omega}{\partial G_S} \Delta G_S + \frac{\partial \Omega}{\partial G_A} \Delta G_A \right] + S$$

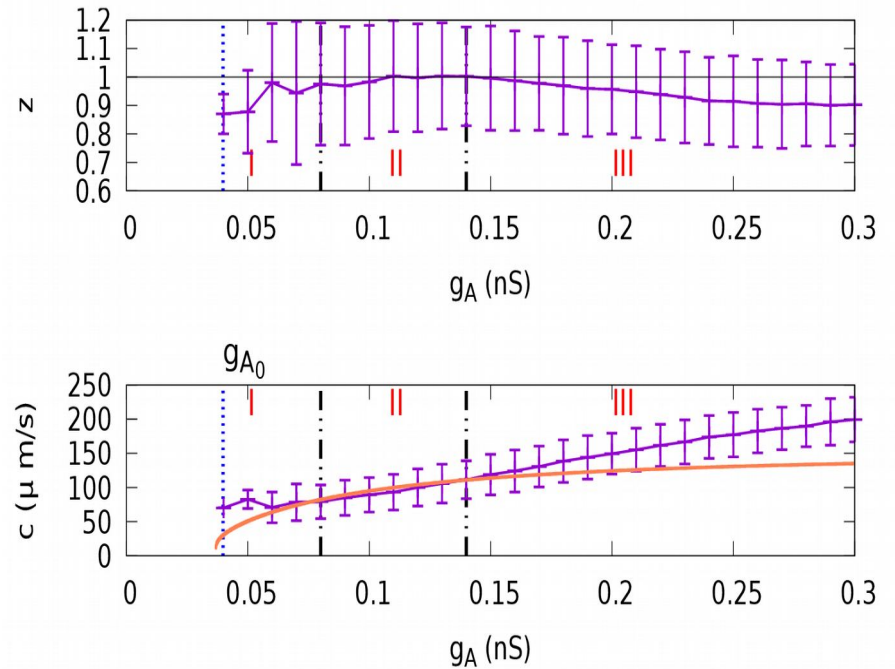


# A transport theory of retinal waves

$$\frac{\partial G_A}{\partial t} = g_A D_A \left[ (\nabla G_S, \nabla G_A) H_\Omega \begin{pmatrix} \nabla G_S \\ \nabla G_A \end{pmatrix} + \frac{\partial \Omega}{\partial G_S} \Delta G_S + \frac{\partial \Omega}{\partial G_A} \Delta G_A \right] + S$$



# A transport theory of retinal waves

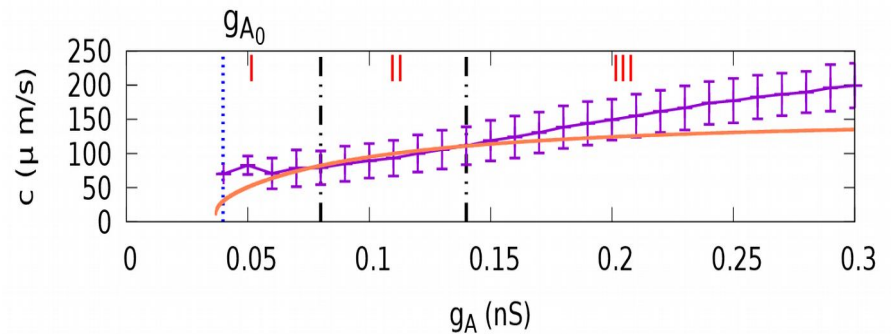
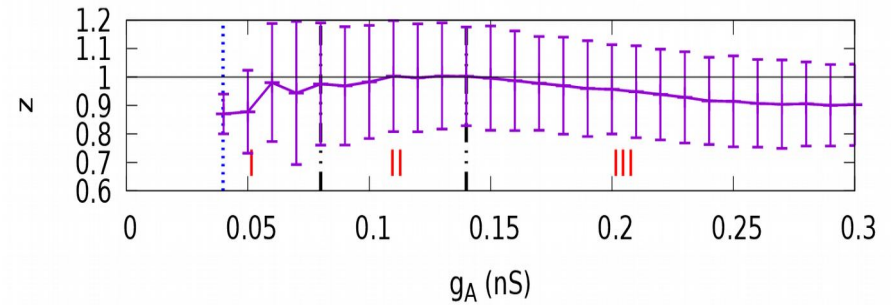




# A transport theory of retinal waves

1) Minimal  $g_A$  value for wave propagation

$$g_{A_0} \equiv \frac{2\mu_A \sqrt{\gamma_A}}{\beta_A \Omega} \frac{I_{SN} + G_{S_2} (V_{Si_2} - V_K)}{V_A - V_{Si_2}}$$



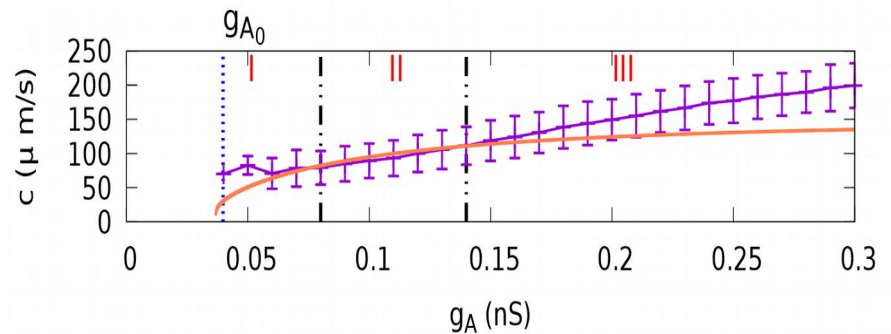
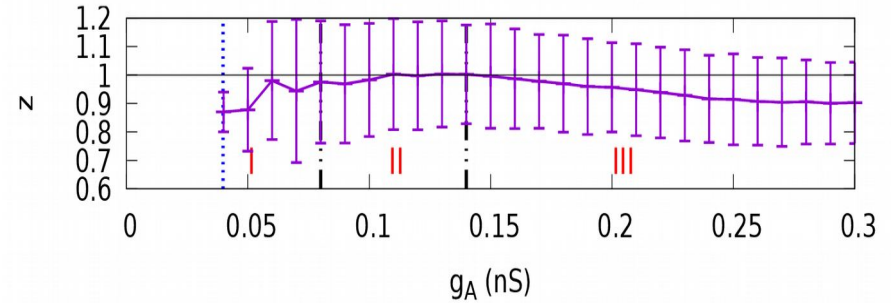
# A transport theory of retinal waves

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2) Wave speed in the ballistic range

$$c = \frac{a}{t_C - \frac{1}{\mu_A} \log \left[ 1 + \frac{1}{g_A} \frac{2\mu_A \sqrt{\gamma_A}}{n\beta_A \Omega} \frac{I_{SN} + G_{S_2} (V_{Si_2} - V_K)}{V_{Si_2} - V_A} \right]}$$



# A transport theory of retinal waves

1) Minimal  $g_A$  value for wave propagation

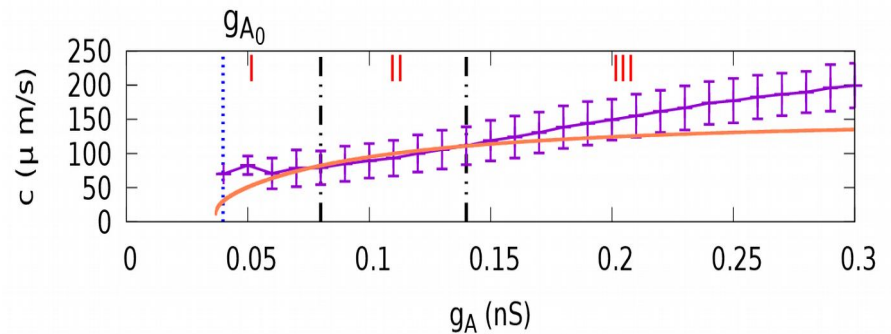
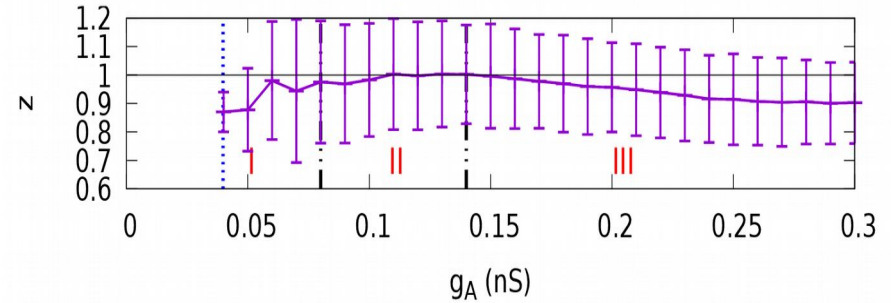
$$g_{A_0} \equiv \frac{2\mu_A \sqrt{\gamma_A}}{\beta_A \Omega} \frac{I_{SN} + G_{S_2} (V_{Si_2} - V_K)}{V_A - V_{Si_2}}$$

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3)  $g_A$  value to enter in regime III

$$g_A = \frac{g_{A_0}}{1 - \frac{1}{2d}}$$



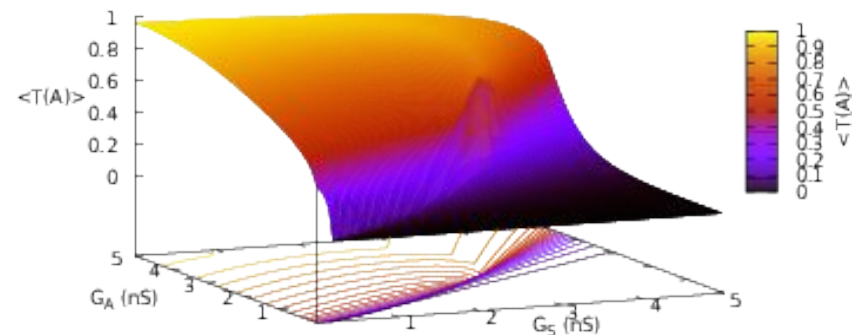
# A transport theory of retinal waves

$$\frac{\partial G_A}{\partial t} = g_A D_A \left[ (\nabla G_S, \nabla G_A) H_\Omega \begin{pmatrix} \nabla G_S \\ \nabla G_A \end{pmatrix} + \frac{\partial \Omega}{\partial G_S} \Delta G_S + \frac{\partial \Omega}{\partial G_A} \Delta G_A \right] + S$$

Links with :

Kardhar, Parisi, Zhang

Self-Organised criticality



Thanks ◀◀