Incremental stability of delayed neural fields: a unifying framework for endogenous and exogenous sources of pathological oscillations

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Introduction

We investigate two possible mechanisms of brain oscillations onset:

1. *Pacemaker effect*: feedback interaction between populations.
2. *Entrainment*: feed-forward propagation from a population to another.

We show that *incremental stability* constitutes a useful framework to investigate both mechanisms providing explicit conditions under which delayed neural fields are incrementally stable. We illustrate our findings with the study of a neural field model of parkinsonian STN-GPe network showing that the model can exhibit sustained oscillations, according to either the "pacemaker" or the "entrainment" mechanism, depending on the strength of the synaptic weights between STN and GPe populations.
The derived theoretical results thus seem to constitute a fertile ground for further investigations based on experimental data, to discriminate between hypotheses for Parkinsonian sustained oscillations in the STN-GPe network.

The Delayed Neural Field Model

We describe the spatiotemporal dynamics of our model network using the following coupled neural field equation (for a survey of neural fields see [5]):

$$\tau_i \frac{\partial u_i(r,t)}{\partial t} = -u_i(r,t) + \sum_{j=1}^{n} \int_{\Omega} w_{ij}(r,r') S_j(u_j(r', t - d_j(r,r')))dr' + I_i(r,t) \quad (1)$$

- Ω is a compact set
- $u_i(r,t)$: activity of population i at position $r \in \Omega$ and time $t \geq 0$
- $I_i(r,t)$: external input
- w_{ij}: synaptic strength functions
- d_j: axonal delays described by $d_j(r,r') = \frac{|r-r'|}{c_j}$, where c_i is the axonal conduction velocity of neurons
- S_i : Lipschitz continuous function with Lipschitz constant l_i

See [3, 4] for a more detailed mathematical description of delayed neural fields. A schematic representation of the model is depicted in **Figure 1**.

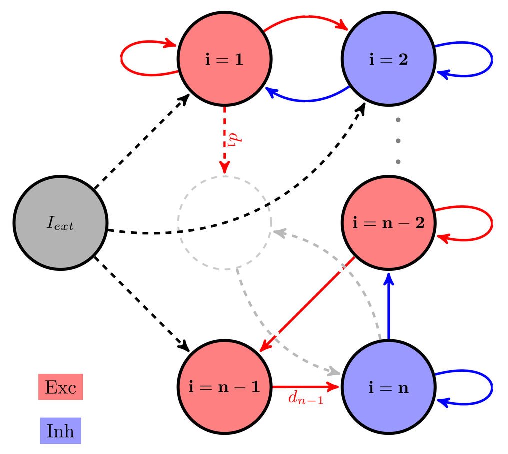

Figure 1

Incremental stability

$\mathcal{KL}$ function: positive definite increasing in first argument, decreasing to zero in second argument.

Definition 1. *[7] The delayed neural field* (1) *is incrementally stable if there exists $\beta \in \mathcal{KL}$ such that, for all admissible initial conditions u_0, v_0 and all input I,*

$$\|u(r,t) - v(r,t)\|_2 \leq \beta(sup_{t\in[-\bar{d};0]} \|u_0(r,t) - v_0(r,t)\|_2, t).$$

- Any two solutions converge to each other.
- If initial conditions are narrow, solutions are narrow at all times.

Two consequences:

- If the input I is constant: all solutions converge to a constant pattern (no pacemaker effect).
- If the input is T-periodic: all solutions tend to a T-periodic behavior (entrainment).

Theoretical Results

Theorem 1. *Let* $\Xi := \sum_{i,j=1}^{n} \int_{\Omega}\int_{\Omega} l_j^2 w_{ij}^2(r,r')dr'dr$, $\tau_{min} := \min_{i=1\ldots n}\{\tau_i\}$, *and* $\tau_{max} := \max_{i=1\ldots n}\{\tau_i\}$. *Assume that*

$$\Xi < \frac{2}{n}\frac{\tau_{min}^2}{\tau_{max}^2}. \quad (2)$$

Then the neural field model (1) *is incrementally stable.*

The proof relies on the Lyapunov function:

$$\mathcal{V}(t) = \frac{1}{2}\sum_{i=1}^{n}\int_{\Omega}(\tau_i v_i(r,t) - \tau_i \nu_i(r,t))^2 dr, \quad (3)$$

and on Lyapunov-Razumikhin conditions for incremental stability [8].

Numerical Experiments

Two hypotheses for sustained STN oscillations in Parkinson's disease:

1. STN-GPe pacemaker effect: in vitro [2] and in silico [9] evidence.
2. Entrainment by exogenous (striatal) structures [1].

We rely on the neural field model [6], which is tuned to experimental data from literature [9]. We run three different numerical cases in order to test our theoretical results and to investigate the conservativeness of our condition of Theorem 1.

Figure 2, left panel: STN oscillations amplitude as a function of the amplitude K_{21} of w_{21}: oscillations appear when $K_{21} > 25$. Right panel: the quantity Ξ appearing in condition (2) as a function of K_{21}. Redish dots indicate K_{21} for protocols **A** and **B**.

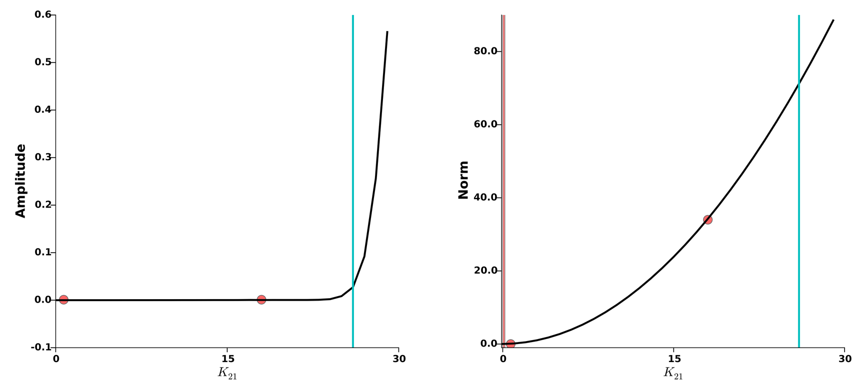

Figure 2

[A] Stability:

We first studied the model when the condition (2) of Theorem 1 is satisfied. In this case we choose the parameters of the synaptic strengths and the synaptic variance in order to get $\Xi = 0.04 < 0.18$. **Figure 3** panel **A** confirms the fact that the system is stable and no sustained oscillatory patterns take place when the external input is constant (no pacemaker effect).

[B] Stability (Non-satisfied cond.):

We choose the parameters of synaptic strengths in such a way that the condition (2) of Theorem 1 is violated ($\Xi = 18.0 > 0.18$). As one would expect the behavior of the system would be to build up oscillatory solution patterns. **Figure 3** panel **B** shows that no oscillations take place either: thus underlining the conservativeness of the proposed condition.

[C] Instability:

We further increase Ξ by increasing synaptic weights ($\Xi = 116 > 0.18$). **Figure 3** panel **C** show that oscillations take place (pacemaker effect).

[D] Bode Plot:

Since incrementally stable systems have T-periodic responses to any T-periodic inputs, we may draw its *Bode plot* in the same way we do for linear systems [7]. This plot indicates what frequencies the neural fields preferably amplify. **Figure 3** panel **D** presents the Bode plot for equation (1). It is apparent that frequencies that lie within the range of β-band are enhanced. This provides support to the striatal entrainment hypothesis: any β-oscillation originally generated in the striatum will propagate in the STN-GPe network.

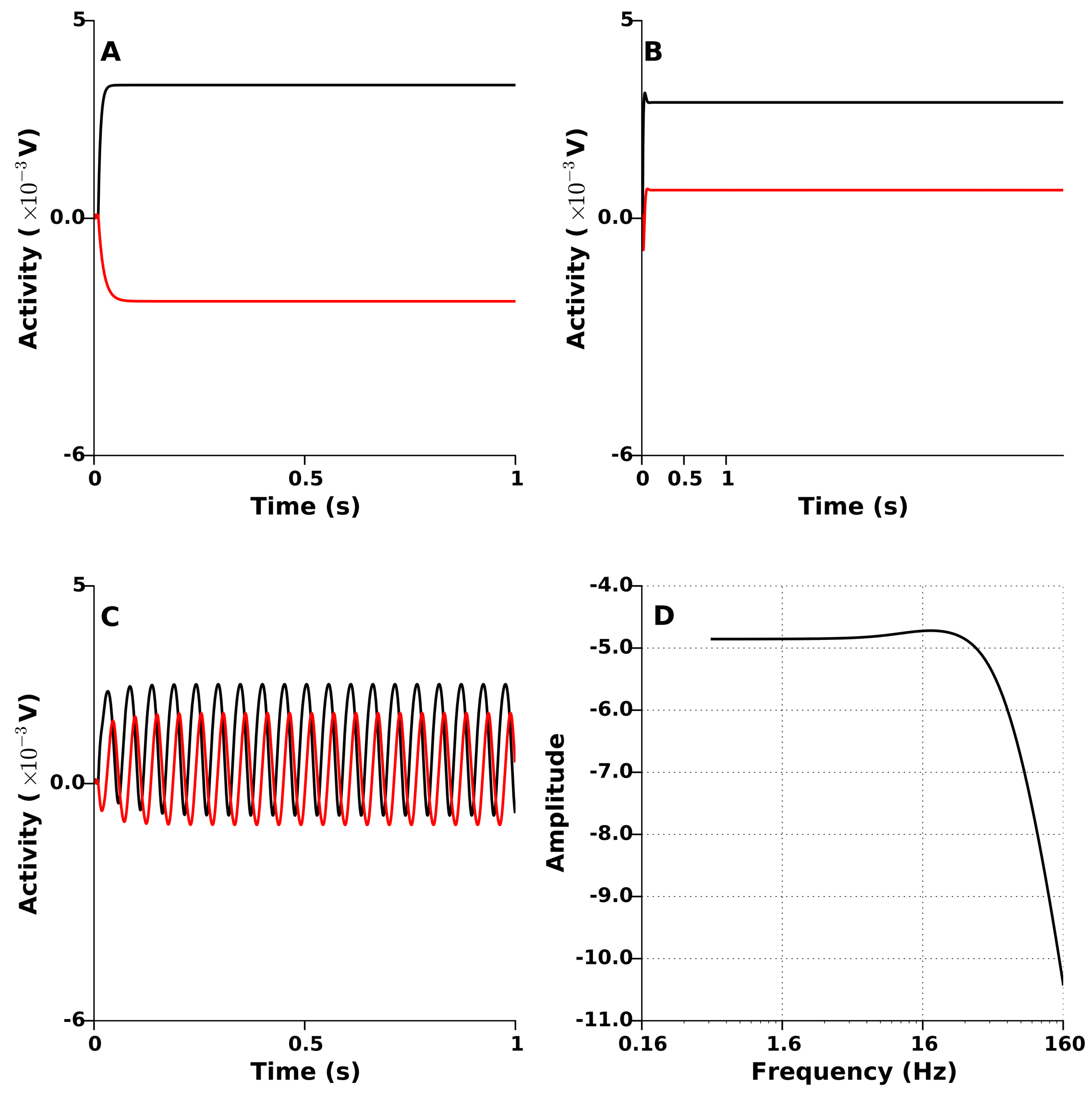

Figure 3

Conclusions

We provided a theoretical and numerical framework for studying incremental stability with delayed neural fields. Focusing on a parkinsonian STN-GPe network model, we showed that two main hypotheses for β-oscillations onset can be analyzed in a unified manner:

- Low STN-GPe synaptic weights: entrainability by external structures.
- High STN-GPe synaptic weights: pacemaker effect.

References

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