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Modelling Activity-Based, Delayed Neuronal Networks

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Introduction

A neuronal network, in its simplest form, can be understood as two populations of neurones (U and V) that have axonal connections with each other. These connections could be inhibitory or excitatory, and each population might also display recurrent activity.

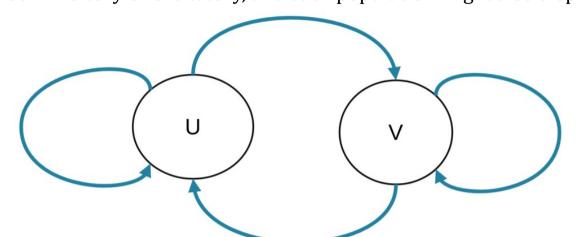


Figure 1. Network diagram of two interacting neuronal populations U and V

Blue arrows indicate axon connections, in this case. either recurrent or interpopulation connectivity

Interacting neurone populations are common in the brain, one example is the interplay between hippocampal and prefrontal regions, implicated in memory processes (see [2])

To accurately model these networks, we should consider time delays in the system equations. These delays reflect the time taken for the generation of an action potential and its propagation from one population to the next - meaning that the activity of one neuronal population depends on the past behaviour of itself/the other population. This poster demonstrates how we can analyse and understand the dynamics of a neuronal network that has these delays.

The Model

Conceptualised by Coombes and Laing [1] as a delayed version of the Wilson and Cowan model [2], it captures the dynamics between two coupled neurone populations:

$$egin{aligned} \dot{u} &= -u + f(heta_u + au(t- au_1) + bv(t- au_2)) \ rac{1}{lpha}\dot{v} &= -v + f(heta_v + cu(t- au_2) + dv(t- au_1)) \end{aligned}$$

Here U and V represent the synaptic activity of both populations, with α^{-1} setting the relative time scale for the response. The change in population activity over time is influenced by its own activity (decay term) and a firing rate function that considers: External drive (θ_u/θ_v) , recurrent activity (delayed by τ_1) and inter-population activity (delayed by τ_2). Using two delays lets us consider that recurrent and inter-population signals might have different time-frames of propagation, and we might expect that $\tau_1 < \tau_2$

The weighting variables (a, b, c, d) can determine the network architecture, allowing each inter-population and recurrent connection to be either excitatory/inhibitory, or completely absent (weight set to 0). The firing rate function f(z) is a sigmoid function, with a slope parametrised by Beta, as:

$$f(z)=rac{1}{1+e^{-eta z}}$$

This non-linear function can complicate our analysis of this model. However, we can make progress by linearising the system locally around its fixed points. Alternatively, we could also consider f(z) as a Heaviside step function, which we explore later in this work.

Linear Stability Analysis

In the model, a fixed point (u*, v*) is present whenever the rates of change of both state variables are zero, so that the delayed value of each state variable will be equal to its current value. Thus, fixed points satisfy:

$$heta_u = f^{-1}(u^*) - au^* - bv^*, \quad \ heta_v = f^{-1}(v^*) - cu^* - dv^*,$$

With the inverse sigmoid function defined as:

$$f^{-1}(z) = eta^{-1} \ln \left(rac{z}{1-z}
ight)$$

To linearise the system, we can Taylor expand the system around a fixed point and guess that perturbations in these regions will evolve exponentially. After rearranging to create a condition to solve for lambda, we arrive at:

$$egin{bmatrix} \lambda + 1 - aeta u^*(1-u^*)e^{-\lambda au_1} & -beta u^*(1-u^*)e^{-\lambda au_2} \ -ceta v^*(1-v^*)e^{-\lambda au_2} & rac{\lambda}{lpha} + 1 - deta v^*(1-v^*)e^{-\lambda au_1} \end{bmatrix} egin{bmatrix} \delta u \ \delta v \end{bmatrix} = egin{bmatrix} 0 \ 0 \end{bmatrix}$$

For perturbations to the system to be non-trivial, the determinant of the matrix has to equal zero. Importantly, despite not influencing the values of (U*, V*), the delays will influence the behaviour of the fixed points (as the delays are present in this matrix). The next steps are to explore this influence, the Wilson and Cowan model displays oscillatory behaviour, which we try to locate in the following section.

Hopf Analysis

To locate oscillatory behaviour we search for $\lambda=i\omega ext{ for } \omega
eq 0, ext{ where } \omega \in \mathbb{R}.$ Hopf bifurcations, at which:

Substituting this and expanding the determinant of the matrix, gives equations for the real and imaginary parts of lambda, that have to be satisfied to indicate a Hopf Bifurcation:

$$0=(1-k_1\cos(\omega au_1))(1-k_2\cos(\omega au_1))$$
 $-(\omega+k_1\sin(\omega au_1))(\omega/lpha+k_2\sin(\omega au_1))-k_3\cos(2\omega au_2)$ Real $0=(1-k_1\cos(\omega au_1))(\omega/lpha+k_2\sin(\omega au_1))$
 $+(\omega+k_1\sin(\omega au_1))(1-k_2\cos(\omega au_1))+k_3\sin(2\omega au_2)$ Imaginary

With K: $k_1 = a eta u^* (1 - u^*), \quad k_2 = d eta u^* (1 - v^*), \quad k_3 = b c eta^2 u^* v^* (1 - u^*) (1 - v^*)$

We can plot continuations of the Hopf bifurcation branches, with the time delays as free parameters, by using the bifurcation tool XPP [4]:

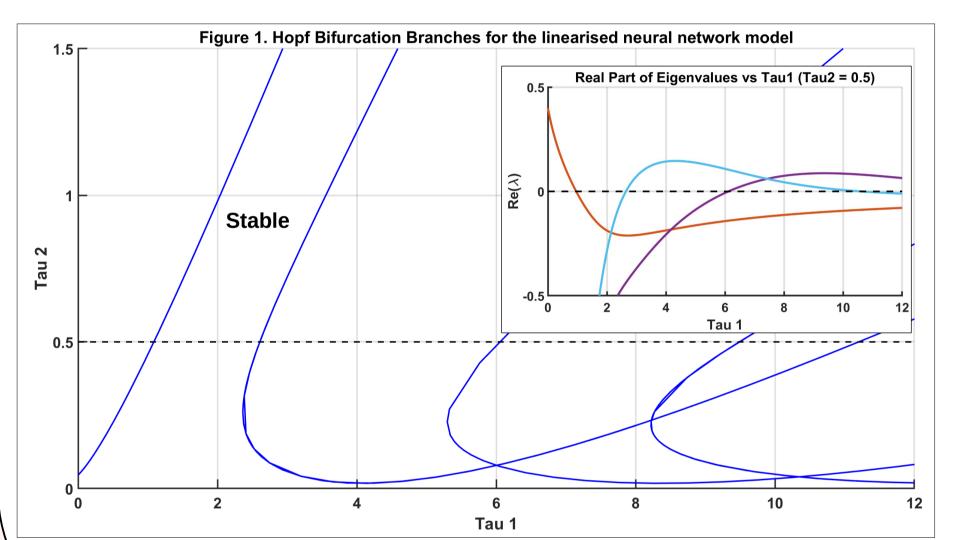


FIGURE 1. Main- Hopf Analysis diagram in τ_1 and τ_2 . Region of stability is indicated. **Inset-** Shows the real parts of the 3 most dominant eigenvalues at each τ_1 (τ_2 fixed at 0.5). Parameters: a=10; b=-10; c=10; d=2; alpha=1; beta=1; $\theta_u=-2$; $\theta_v=-4$;

DDE-BIFTOOL

To study the model in its original form, we use the numerical bifurcation tool DDE-BIFTOOL [5]. We consider a different network architecture than above, see figure heading.

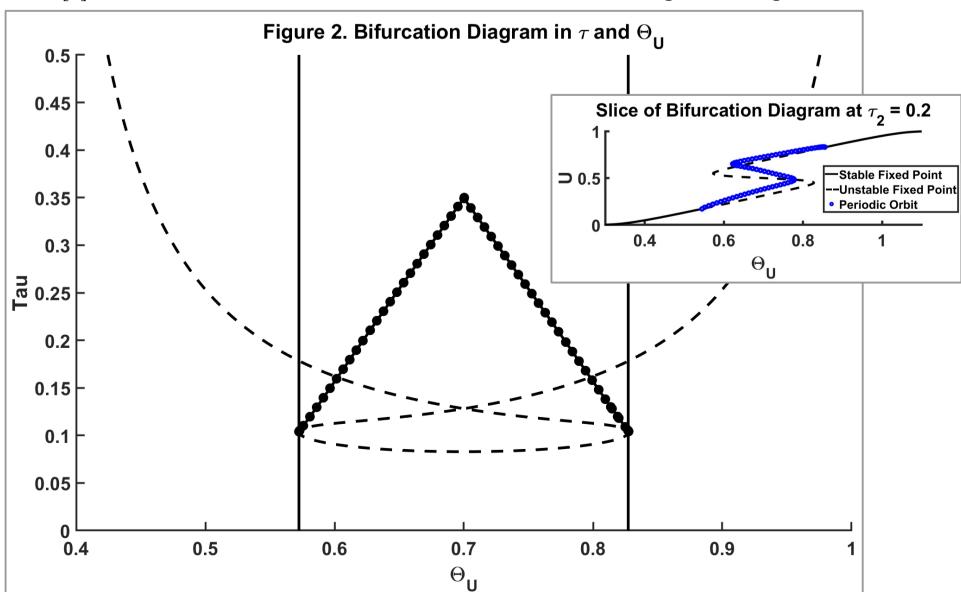


FIGURE 2- Main -Bifurcation diagram in θ_u and τ ($\tau_2 = \tau_1$). Circled Line = Saddle node bifurcation of periodic orbit, Dashed line = Hopf Bifurcation, Solid line = Saddle node bifurcation of fixed points. **Inset** – Horizontal cut through main diagram at $\tau_2 = 0.2$. Parameters: a=-1; b=-0.4; c=-1; d=0; alpha=1; beta=60; θ_v =0.5;

Oscillatory Activity

We now turn to numerical simulations (DDE23 in MATLAB) and analytical solutions to demonstrate oscillatory $\dot{}$ behaviour. To generate analytical solutions the firing threshold function has been taken as a Heaviside step function (beta $\rightarrow \infty$). We consider a fully-inhibitory network architecture (a,b,c,d < 0), with excitatory activity only being derived externally (θ_u and θ_v).

Figure 3. Co-Existing Synchronous and Asynchronous solutions for the delayed neural network model

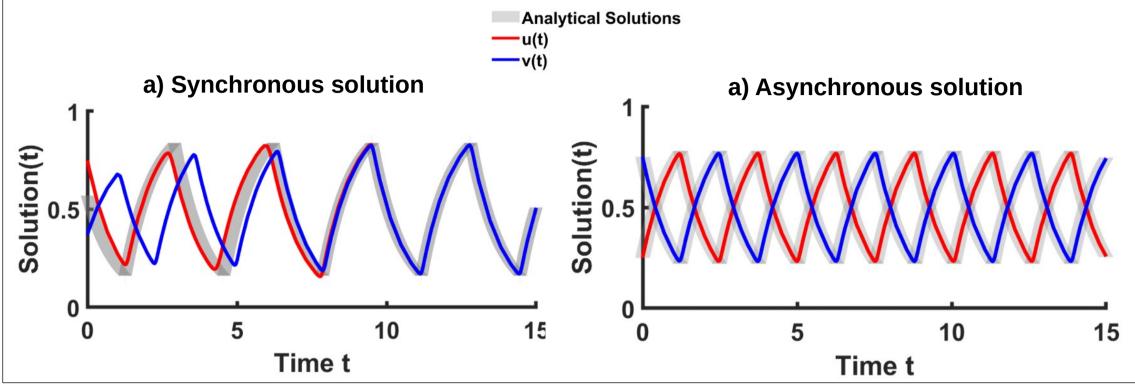


FIGURE 3. Synchronous (a) and asynchronous (b) solutions to the delayed, fully inhibitory neural network model

Parameters: a = -1; b = -0.4; c = -0.4; d = -4; alpha = 1; $\theta_u = 0.7$; $\theta_v = 0.7$; $\tau_1 = 1$; $\tau_2 = 1.4$. **Initial Conditions**: a) v0 = 0.37; u0 = 0.75; b) v0 = 0.75; u0 = 0.25; **Beta**: Analytical= ∞ ; Numerical=60

The analytic solutions represent the behaviour after a stable oscillation has been reached. For the anti-synchronous solution this can be derived by solving equations describing the oscillation amplitudes (A+/A-) and periods (T1 =Ascending, T2 = Descending):

$$T_1=\ln\left(rac{s+ heta_u+a+b}{ heta_u}
ight) \quad T_2=\ln\left(rac{ heta_u-s}{ heta_u+a+b}
ight) \qquad egin{aligned} A_-=A_+e^{-T_1}\ A_+=1+(A-1)e^{-T_2} \end{aligned}$$

Analytical solutions are derived in a similar fashion for the synchronous oscillations [1], S is defined as:

$$s=-(ae^{T_1}+be^{T_2})$$

Irregular Oscillations

Beyond regular oscillations (consistent period and amplitude) we also see more 'disordered' oscillatory behaviour.

Figure 4. Irregular solutions to the Delayed Neural Network Model

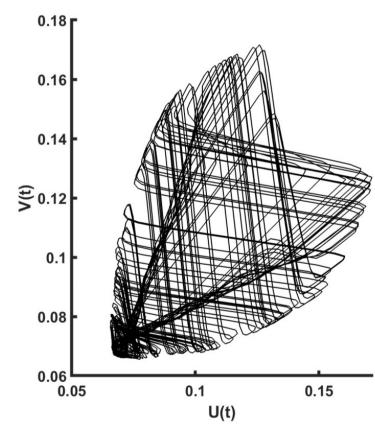


FIGURE 4. Numerical Solutions to the delayed neural network model, plotted in the U,V phase plane. Parameters: a=-6; b=2.5; c=2.5; d=-6; theta u=0.2; theta v=0.2; tau1=0.1; tau2=0.1; Time-span=0-70; Alpha=1.

Conclusions

Incorporating delay terms into a neuronal network model replicates an important constraint of actual neuronal systems, that of the time taken for signals to generate and propagate from one population to the next.

This poster highlighted that introducing delays into the Wilson Cowan model [2] still allows for oscillatory activity, as well as seemingly chaotic behaviour under certain conditions.

To increase the biological accuracy of the model, future work could consider:

1) Space dependent delays: Neuronal populations that are spatially further apart, will take longer to signal between each other. This approach has been analysed in a more complicated small-world network model [6].

2) State dependent delays: As the activity of a Neurone population changes, it might switch between fast and slow synaptic receptor currents, changing the timescale of its response. This type of delayed neuronal network can be seen in work by Zhang and Chen [7].

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