

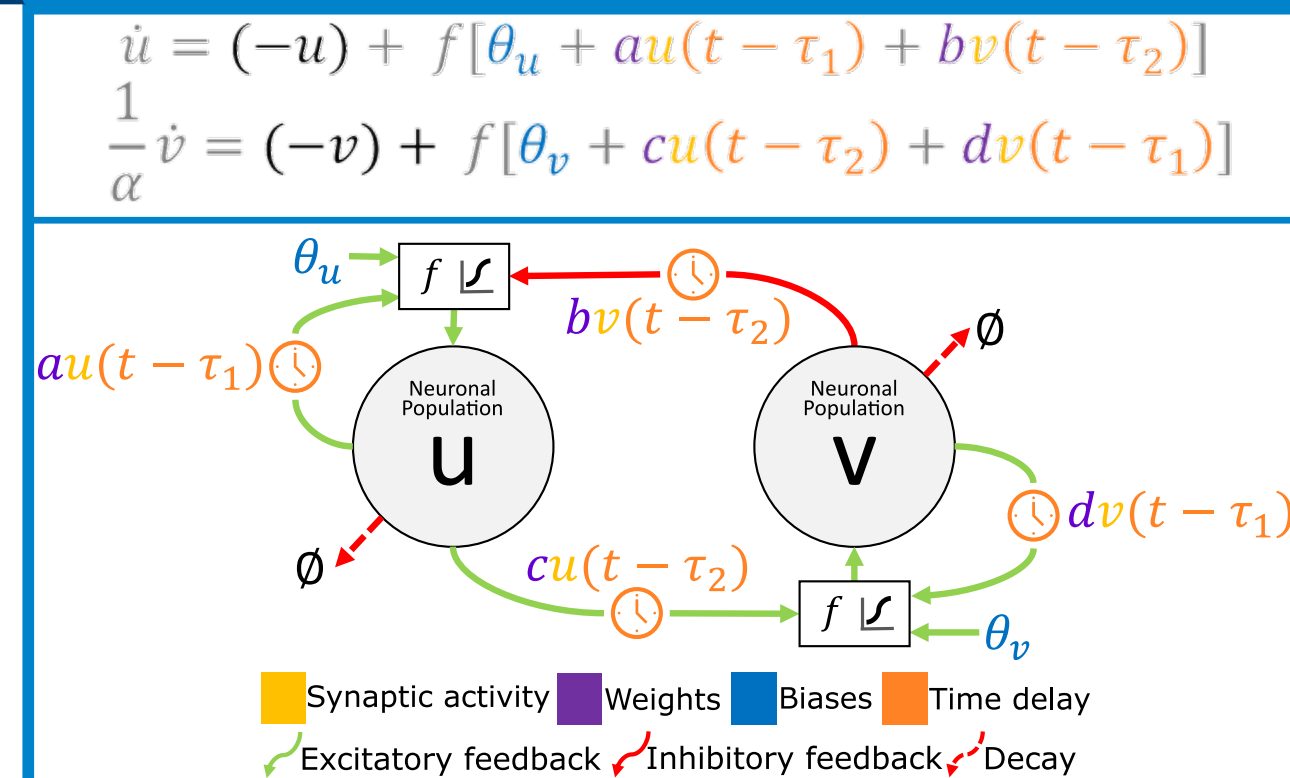


## 1. Introduction

A time delay,  $\tau$ , can be included in mathematical models to acknowledge that communication between neurones is not instant. For example, delays may arise as impulses take time to traverse an axon, or transduce at a synapse. The addition of delays in a neural model invites us to explore how they affect a dynamical system.

Coombes and Laing show that, when applied to the Wilson-Cowan model [1], two discrete delays can destabilise fixed points and lead to oscillatory and chaotic behaviour [2]. Here, we outline and simulate these findings before discussing the relevance of these behaviours and how they connect to neuroscientific research.

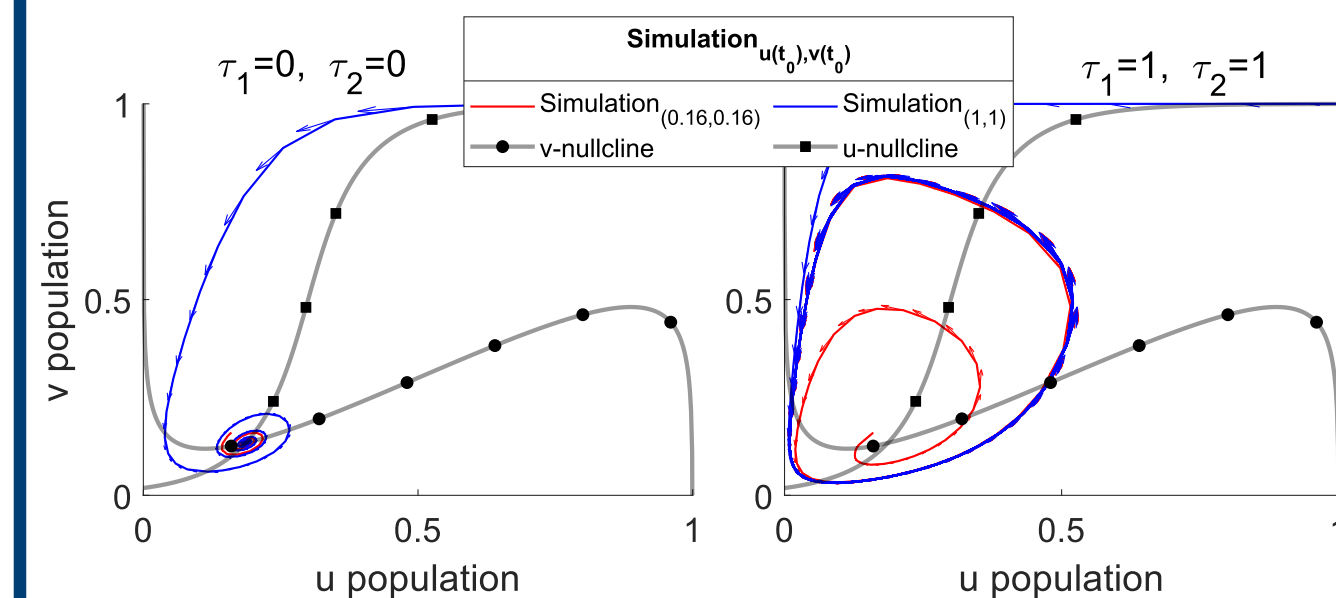
### Delayed Wilson-Cowan Model



**Figure 1:** Wilson-Cowan model, with delays, for an excitatory-inhibitory network of two neuronal populations. Self and inter-population feedback (yellow) are weighted (purple) and delayed (orange); then, alongside background drive/bias (blue), are interpreted by a firing rate function,  $f$ , to give activity.

## 2. Stability and identical delays

Steady states exist independent of delays; however, their stability can be delay-dependent. Consider a Wilson-Cowan model with weights for an excitatory-inhibitory network (Figure 1):  $a=-b=c=10$ ,  $d=2$ . By also setting parameters  $(\theta_u, \theta_v)=(-2, -4)$  and  $\alpha=\beta=1$ , we follow the work of Coombes and Laing [2] and explore varying  $\tau=\tau_1=\tau_2$ , where both delays are the same (Figure 2). Then, we change the parameters for Figure 3 to show different behaviour.

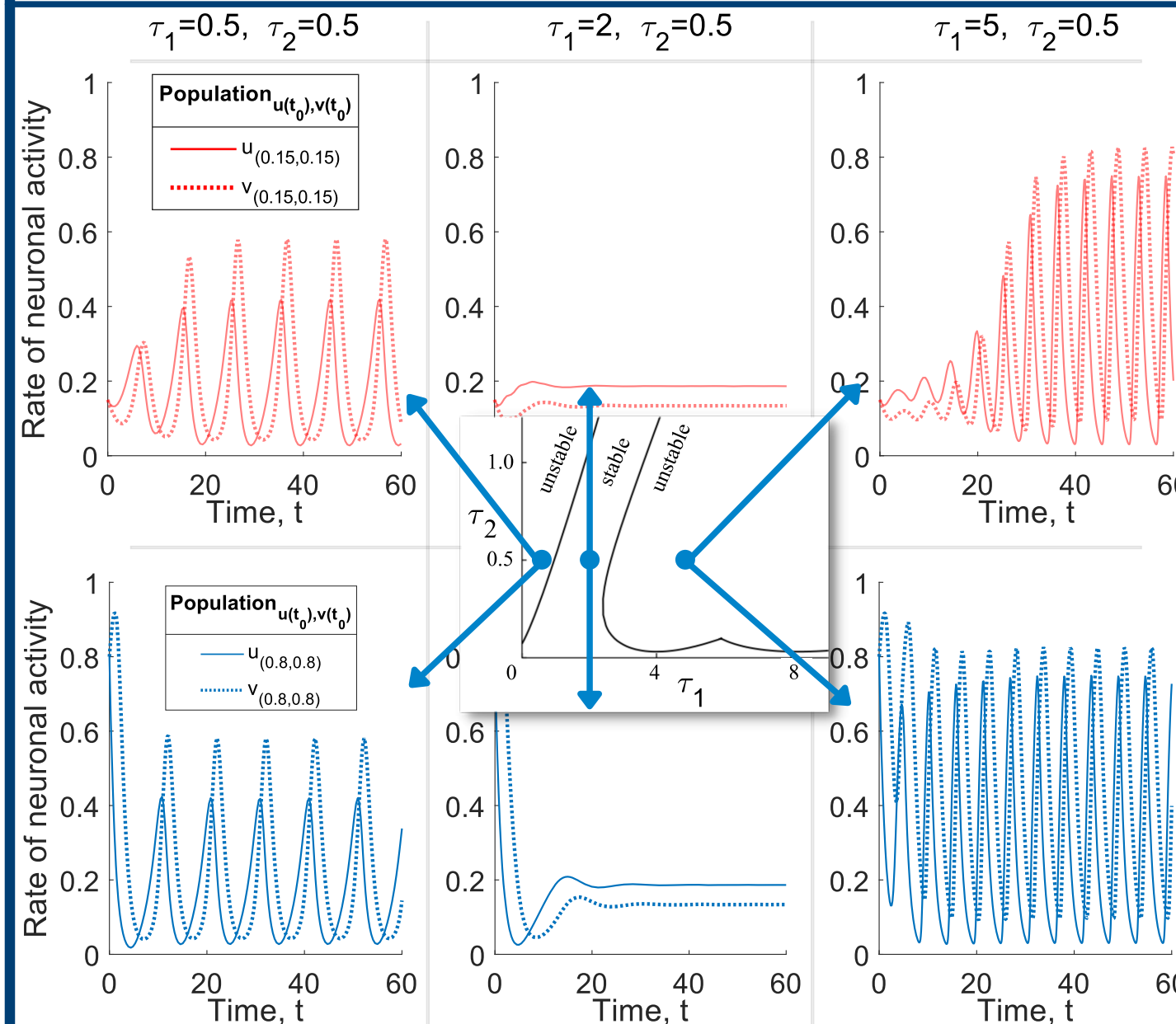


**Figure 2:** U-V phase planes with superimposed  $u$ - and  $v$ - nullclines. Steady states exist for both simulations at  $\approx(0.16, 0.16)$ . Without delays (left), oscillatory behaviour decays to a stable fixed point. However, delays (right) make this fixed point unstable and a stable, periodic limit cycle emerges.

## 3. Stability and two discrete delays

Delays associated with impulses arriving from a neighbouring neuronal population (i.e., "cross-interactions") could differ from those of a population's "self-interactions" [2]. For this reason, in this section we now independently vary  $\tau_1$  and  $\tau_2$ , keeping other parameters the same as in Section 2.

If delays are sufficiently sized, the system is unstable. However, Coombes and Laing document an "interference effect" [2], where Hopf bifurcations bring about stability if  $\tau_1$  and  $\tau_2$  are taken from a window of values. This window is sandwiched between two regions of instability (Figure 4) that, akin to that seen with identical delays, lead to unstable fixed points occurring alongside oscillatory behaviour.



**Figure 4:** Three pairs of delays  $(\tau_1, \tau_2)$ :  $(0.5, 0.5)$ ,  $(2, 0.5)$  and  $(5, 0.5)$ , are sampled from a central graph modified from Coombes and Laing's [3, see: Section 3]. We simulate two  $(u, v)$  initial conditions,  $(0.15, 0.15)$  in red and  $(0.8, 0.8)$  in blue, for these delays.

When  $(\tau_1, \tau_2) = (0.5, 0.5)$ , shown on the left, a periodic orbit found similarly in Figure 2 exists. By transitioning  $(\tau_1, \tau_2)$  to  $(2, 0.5)$ , the system settles to a stable fixed point. This stability is reversed by further increasing  $\tau_1$ , setting  $(\tau_1, \tau_2) = (5, 0.5)$ . Thus demonstrating an "interference effect" [2].

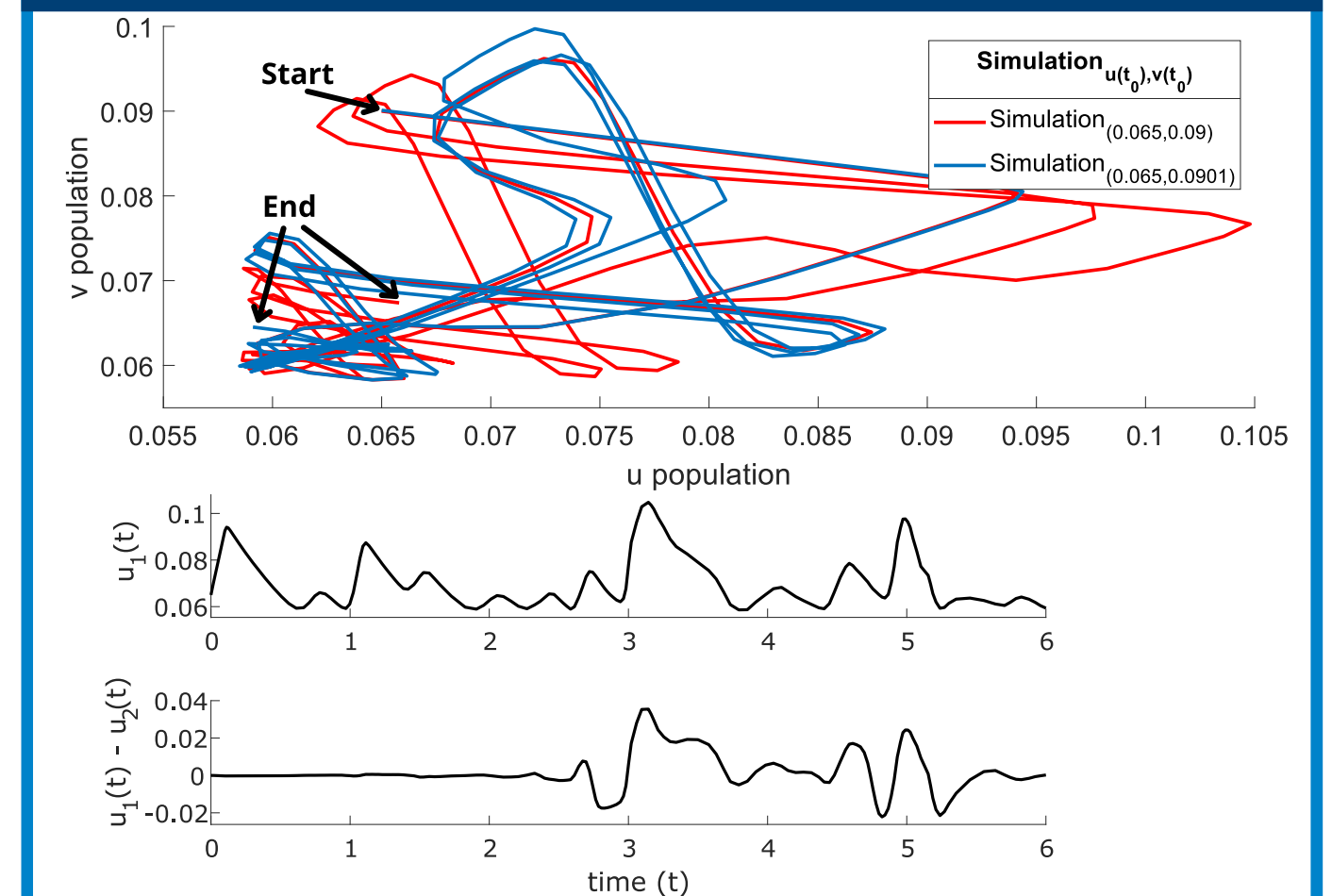
Aside from the Hopf bifurcation shown in Figure 2, the Wilson-Cowan model also supports saddle-node bifurcations (SN). Coombes and Laing show that for parameters as in Figure 3, a SN bifurcation of a fixed point occurs when  $0.56 < \theta_u < 0.83$  [2]. Thus, in Figure 3 we show SN and Hopf bifurcations to unstable fixed points (higher and lower steady states) and SN bifurcations to a periodic orbit (middle steady state).

**Figure 3:** U-V phase plane with superimposed  $u$ - and  $v$ - nullclines for an inhibitory network under 3 initial conditions  $\alpha=1$ ,  $\beta=60$ ,  $a=-1$ ,  $b=-0.4$ ,  $c=-1$ ,  $d=0$ ,  $\theta_u = 0.7$ ,  $\theta_v = 0.5$ ,  $\tau_1=\tau_2=0.2$ . All trajectories decay to stable periodic orbit. However,  $(0.6, 0.6)$ , in green, starts near an unstable periodic orbit, cycling a few times before diverging towards a stable periodic orbit.

## 4. Chaos/quasi-periodicity and weights

It is also possible for a delayed Wilson-Cowan model to exhibit behaviour that is chaotic or quasi-periodic for some parameter sets. Following [2], we see that chaos occurs with short-range inhibition ( $a=d=-6$ ) and long-range excitation ( $b=c=2.5$ ).

By definition, chaotic trajectories show sensitive dependence on initial conditions (SDIC). That is, any two similar initial conditions  $(u_1, v_1)$  and  $(u_2, v_2)$  will follow diverging trajectories with a difference that grows exponentially with time. Figure 5 uses the Wilson-Cowan model to exemplify and visualise SDIC. Additionally, and not shown here, by decreasing  $\beta$  to make the firing rate function shallower we can alter the behaviour of the system from chaotic to quasi-periodic.



**Figure 5:**  $\alpha=1$ ,  $\beta=60$ ,  $a=d=-6$ ,  $b=c=2$ ,  $\theta_u=\theta_v=0.2$ ,  $\tau_1=\tau_2=0.1$ ,  $t=6$ . Two similar initial conditions  $(0.065, 0.09)$  and  $(0.065, 0.0901)$ . Top: U-V phase plane. Bottom: Despite changes in  $u_1$ , divergence between trajectories occurs after  $t=2.5$ , demonstrating SDIC.

## 5. Discussion

Delays can support stable yet dynamic behaviours that can facilitate rhythmic activity patterns between neuronal populations (i.e., "bursting") [2,3]. In Figure 3, we propose an example of an unstable orbit causing a period of oscillatory activity, or bursts, followed by relative quiescence. Bursts are understood to be the neurological mechanism be walking, breathing and other repetitive tasks [4].

By studying neurological rhythms, we can research how the brain encodes data and appreciate the origins of cerebral dysrhythmias (eg. epilepsy and Parkinson's disease) [3,5]. Future work ought to consider models of numerous delays. After all, neuronal populations can receive multiple inputs from various distances, such as a third neuronal population. Taking this further, time delays need not be discrete, either. Finding neural models that allow for numerous delays of different types will permit more reliable and realistic approximations of brain activity.

## 6. References

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