A Very Small Chaotic Neural Net

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Abstract—Previously we have shown that chaos can arise in networks of physically realistic neurons [1], [2]. Those networks contain a moderate to large number of units connected in a spatial arrangement providing instances of so-called Cellular Neural Networks. It was proposed that the flexibility and wide range of behaviors of chaos could be of computational value, namely in spatiotemporal regimes and when coupled with a chaos control process, either in biological or artificial nets. Here we aim to find a minimal network of realistic neurons already featuring a chaotic regime. Such a small network can be computationally useful per se, or otherwise constitute the building block of larger networks with even richer dynamical regimes. Our investigation unveils the role of the interplay between a homoclinic tangency and the presence of delays in neural signal transmission in the creation of complex behavior.

I. INTRODUCTION

Chaos can be observed in neural networks even when realistic model neurons are considered [1], [2]. While careful exploration of parameter space is convenient, no purposeful mathematical gadgets need to be added in order to obtain complex behavior. Suggestions of the computational usefulness of chaos were given in [3], [4], [5], namely by showing how visual pattern processing can take place in biologically inspired networks [3], [5]. In previous work we focused on network properties requiring that a large enough number of neurons be connected in some spatial arrangement. In the present paper we downsize the network to as few as two units, actually one excitatory and one inhibitory neuron. We thus obtain a minimal chaotic building block. This can then be used either as a standalone complexity generator module or in the creation of larger networks where e.g. spatial symmetries can also be explored. The two latter applications, however, are outside the scope of this paper.

II. A MINIMAL REALISTIC MODEL

We adopt a model of the leaky integrator type, endowed with time-delays in the signal transmission between neurons [6], [2]. Passive and active membrane properties are featured. Neuron connectivity is nonlinear, via the well-known sigmoidal activation function. Details of the model derivation can be found in [2]. Here we consider a simplified version which can be viewed as either modeling an excitatory neuron interacting with an inhibitory one, or otherwise representing the uniform activity of, respectively, a population of excitatory and inhibitory neurons coupled together. Individual neurons are not oscillators under the present model. However, oscillations in electrical activity arise due to the presence of excitatory and inhibitory feedback through synaptic connections.

Mathematically, the model consists in a set of two coupled nonlinear delay differential equations,

\[
\begin{align*}
\frac{dX}{dt} &= -\gamma(X - V_L) - (X - E_1) \Omega_1 F_X[X(t - \tau)] \\
&\quad - (X - E_2) \Omega_2 F_Y[Y(t - \tau)] \\
\frac{dY}{dt} &= -\gamma(Y - V_L) - (Y - E_1) \Omega_3 F_X[X(t - \tau)] .
\end{align*}
\]

Here, \(X\) represents the membrane potential of an excitatory neuron, and \(Y\) that of an inhibitory neuron. The different equilibrium potentials have the values \(V_L = -60\) mV, \(E_1 = 50\) mV, and \(E_2 = -80\) mV. As with other parameters of the model, these values are suggested by electrophysiological experiments [6], [7]. The inverse of the membrane’s time-constant takes the value \(\gamma = 0.25\) msec\(^{-1}\). The sigmoidal activation function is of the form

\[
F(V) = \frac{1}{1 + e^{-\alpha(V - V_c)}} .
\]

The parameters \(\alpha\) and \(V_c\) are adjusted so that the neuron is silent for a potential \(V\) lower than some activation threshold, and the firing rate saturates for a certain higher value of \(V\). \(V_c\) is fixed at \(-25\) mV, whereas \(\alpha_X = 0.09\) mV\(^{-1}\) and \(\alpha_Y = 0.2\) mV\(^{-1}\). Note that there is a delay \(\tau\) in signal transmission, including the case of the excitatory neuron feeding back onto itself. The delay is a varying parameter of the model. All types of connections are present except the inhibitory-to-inhibitory one. Synaptic weights are given by \(\Omega_1 = 6.3\), \(\Omega_2 = \Omega_3 = 5\) everywhere in this text except in Fig. 2 where \(\Omega_2\) is allowed to vary. From Eqs. (1), it is clear that, in the absence of coupling, the neurons would simply relax toward the \(V_L\) equilibrium. By this we refer to the case where the self-coupling of \(X\), with delay \(\tau\), would also be absent. This would correspond to Eqs. (1) being left without any \(F_X\) or \(F_Y\) terms.

III. ROUTE TO CHAOS

By varying the parameters of system (1), a wide range of dynamical behaviors can be observed. To gain some analytical understanding of the system, one may keep most of the parameters fixed, and change the few remaining parameters. In this manner, one can identify the loci of bifurcations from steady states into other stationary states or into periodic oscillations. Such bifurcation diagrams have been calculated elsewhere [8], for typical sets of fixed parameters. Destexhe has also estimated the network size for which uniform oscillations of networks of neurons of the above type become linearly unstable [8]. However, that is a multi-unit scenario clearly different from the one we are considering. In our case, we can reduce the parameters to a single equation, in order to focus on the basic mechanisms of chaos. The results of the calculations are presented in the next section.

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case, bifurcations do lead to chaos and this happens for as few as two neurons. Let us note that different routes to chaos, namely of a spatiotemporal nature, are possible if we turn directly to the multi-unit case [8], [2].

To investigate the creation of oscillatory solutions, we considered Hopf bifurcations with different types of system parameters acting as the bifurcation parameter. These included the values of synaptic strengths $\Omega_I$, of the propagation delay $\tau$, and of the transfer functions' slopes $\alpha_X$ and $\alpha_Y$.

Figure 1 provides an example of a Hopf bifurcation unfolded by varying $\tau$ in Eqs. (1).

![Figure 1](image_url)

**Fig. 1.** Hopf bifurcation from a stationary solution of Eqs. (1). The amplitude of the cycles increases with the value of the time-delay $\tau$. Apart from $\tau$, all other parameters of Eqs. (1) are fixed and have the values as indicated in the main text.

Shown are three limit cycles obtained for different values of the delay.$^1$ The cycles with $\tau = 0.424$ and $\tau = 0.6$ are relatively close to the onset of the bifurcation. With $\tau = 4$, a solution with higher amplitude and more complex structure is seen. With the same fixed parameters as in this figure, for higher values of $\tau$, chaotic solutions are observed. This is strong evidence that time-delays are of crucial importance in neuronal modeling, and can change completely the nature of the dynamics. For instance, with $\tau < 0.42$ and all other parameters as in Fig. 1, only a stationary state can be observed asymptotically.

It is known that dynamical systems may present different routes to chaos. In the following, we identify a possible route to chaotic behavior of Eqs. (1). For finite regions of the parameter-space, the system may present bursting oscillations. Figure 2 shows some examples of bursting regimes. In this figure, $\tau$ takes different values, and the synaptic weight $\Omega_2$ is also allowed to vary from the usual value $\Omega_2 = 5$. The bursting oscillations are characterized by two different time-scales. Very fast oscillations are superimposed on a basic cycle occurring on a much longer time-scale. Following a suggestion by Gaspard (see also [9]), the bursting oscillations have been interpreted as signaling the presence of a homoclinic tangency to an unstable limit cycle [10]. The basic phenomenon concerns the graphs (a), (b) and (c) of Fig. 2, which are obtained with successively smaller values of $\Omega_2$ and all other parameters fixed. As $\Omega_2$ is decreased, the duration of the active phase, as well as the number of fast pseudo-cycles thereof, increase continuously. The intermediate segment, or silent phase, remains practically unchanged. For a critical value of $\Omega_2$ close to 4.26, an infinite-period bifurcation takes place. Exactly at the bifurcation point, the bursting phase lasts an infinite time. For lower values of $\Omega_2$ past that point,$^1$ Since the full dynamics is infinite-dimensional due to the delay terms, the figure only displays projections of that dynamics on the $(X, Y)$ space.
the dynamics collapses into a stationary solution (not shown in the figure). At the bifurcation, the stationary solution is
given by \((X, Y) = (-13.38, 43.04)\) mV. In analogy with the
case studied by Destexhe and Gaspard \[10\], the following
explanation can be proposed\(^2\). A limit cycle, called LC2,
accounts for the fast oscillations. There is another cycle,
LC1, which is born in a familiar way at a Hopf bifurcation\(^3\)
and which increases in amplitude (as \(\Omega \) is decreased) until
it “collides” with LC2. LC2 cannot exist without the inter-
mediate segment. Therefore, LC2 must be an unstable limit
cycle. Let us consider the induced discrete-time dynamics at
a Poincaré section transverse to the continuous-time cycles.
LC2 appears to be a cycle of saddle type, because orbits
like LC1 may enter the vicinity of LC2 along a path close
to the stable manifold of LC2, and then escape along a
path close to the unstable manifold of LC2. The bursting
oscillations appear when LC1 grows to a point where it
approaches significantly the region of phase-space where
LC2 exists, such that the oscillations become increasingly
LC2-like. Hence, the bursting can be interpreted as a transient
distortion of the limit cycle LC1 due to the presence of LC2.
At the critical point, or homoclinic tangency, LC1 coalesces
with LC2. If viewed at the Poincaré section, the system’s
trajectory approaches the saddle fixed point LC2 exactly
along the stable manifold of the latter, and escapes LC2
along its unstable manifold. At the bifurcation, the LC2-
like oscillations are no longer a transient process, but last an
infinite time. The exact homoclinic situation is inaccessible
experimentally. Nonetheless, the approach of the critical
situation is signaled by an increase of the bursts’ duration,
depending logarithmically on the distance to criticality \[10\].
This is illustrated as the sequence (a)→(b)→(c) of Fig. 2.
In the previous investigation \[10\], the homoclinic tangency
accounts for a succession of regular burst regimes akin
to the one illustrated by that partial sequence of Fig. 2.
Here, we extend the investigation by showing that irregular
behavior of the chaotic type may also appear, associated
with the homoclinicity. In Fig. 2, this corresponds \emph{e.g.}
to taking the system along a path in parameter-space given
by the sequence (a)→···→(g). This is one of the many
possible sequences. For higher values of the delay \(\tau\), the
succession of bursting periods gains an irregular character,
until temporal chaos is obtained (for \(\Omega_2 = 5\), in the figure).
We propose therefore that chaos is associated with the
interaction between the homoclinic tangency and the memory
effect due to the presence of the delay terms\(^4\). Let us note that
the role of homoclinic orbits in the generation of complex
temporal behavior had been pointed out previously by several
authors \[11\], \[12\].

In the remainder of the paper, we adopt the parameter
values \(\Omega_2 = 5, \tau = 16\) msec. The resulting chaotic regime
is investigated for the system (1). In Figure 3, a Poincaré
section of the dynamics of Eqs. (1) shows the lack of low-
dimensional structure of the chaotic attractor.

![Figure 3](image-url)

Figure 4 illustrates the same dynamics, but in continuous
time. We also made a comparison of the above dynamics
with that of a network comprising several chaotic neurons
of the type described. Such comparison is not detailed in
the present paper. Not surprisingly, we found that chaos is
more developed in the full network case. Notwithstanding,
by coupling several chaotic neurons we brought about a route
to spatiotemporal chaos that had not yet been investigated
by other authors. Indeed, the origin of spatiotemporal chaos
in larger networks, in our case, is somewhat different from
the one in \[8\], \[13\]. Given the route to chaos that we
identified with the smaller system, Eqs. (1), we see that
as few as two coupled neurons suffice to display chaotic
behavior. Therefore, “spatiotemporal” chaos can be studied
with arbitrarily small networks. We could thus study chaotic
networks with between 2 and 32 neurons. References \[8\],
\[13\] present a different picture. The Author’s numerical sim-
ulations, with parameter values different from ours, show that
uniform periodic oscillations of a network are destabilized
only for network sizes greater or equal to \(N_{ex} = 144, N_{in} = 36\),
which are, respectively, the number of excitatory and inhibitory
neurons. In the same numerical study, but with a less favorable connectivity pattern, the periodic bulk
oscillations are not destabilized for network sizes smaller
than \(N_{ex} = N_{in} = 1600\). In the simulations in \[8\], \[13\],
the destabilization of uniform solutions happens through a

\(^2\)Those authors have not considered the same parameters as ours —hence
they have not found chaos, but the homoclinic phenomenon is analogous.

\(^3\)With \(\tau\) fixed at 7 msec and all other parameters with the usual values,
LC1 originates from a supercritical Hopf bifurcation of the fixed point
\((X, Y) = (-75.53, -40.09)\) mV, at a critical value \(\Omega_2 = 68.6\).

\(^4\)Actually, the intermediate stages of the approach to the homoclinic
tangency, at \(\tau = 7\) msec, already show some irregularity. But chaos is only
really developed for delay values greater than about 8 msec, and appropriate
values of \(\Omega_2\).
type of spatiotemporal intermittency (see e.g. Figure 4 of Ref. [13]) that is not featured in our study of larger networks. The same Author shows theoretically that periodic bulk oscillations are destabilized for network sizes greater than $N_{ex} = N_{in} = 9$, but with special network configurations that we do not consider.

IV. CONCLUSIONS

We described a minimal network of realistic continuous-time neurons which are capable only of relaxing toward equilibrium when taken in isolation, but which display chaotic dynamics when synaptically coupled and in the presence of time-delays. Two neurons suffice to observe the chaotic behavior.

Although the paper’s emphasis is on the dynamics of a very small network, we also pointed out that such small chaotic modules could be connected in order to obtain larger networks with more complex dynamical regimes. These include spatiotemporally chaotic regimes, albeit of a different nature from the ones investigated by other authors and also from the ones proposed in other studies by the present author [2].

Chaos may become most useful computationally when coupled with some form of chaos control [5]. Such control was achieved by us with the present model, both in the two-neuron version and for larger networks. While the discussion of a particular control procedure had already been provided [1], the actual mechanism of chaos generation in the homoclinic context is discussed only in the present paper.

Dynamical features of this biologically inspired model were revealed through theoretical considerations and numerical simulations. In subsequent work, an electronic implementation could be envisaged such that dynamical and computational properties might be explored in an actual physical device.

ACKNOWLEDGMENT

The author acknowledges the partial support of Fundação para a Ciência e a Tecnologia and EU FEDER via the Center for Logic and Computation and the project ConT-Comp (POCTI/MAT/45978/2002), and also via the project PDCT/MAT/57976/2004.

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