

Research Article

A Linear Analysis of Coupled Wilson-Cowan Neuronal Populations

L. L. Neves¹ and L. H. A. Monteiro^{1,2}

¹Universidade Presbiteriana Mackenzie, Escola de Engenharia, São Paulo, SP, Brazil

²Universidade de São Paulo, Escola Politécnica, São Paulo, SP, Brazil

Correspondence should be addressed to L. H. A. Monteiro; luizm@mackenzie.br

Received 23 February 2016; Accepted 4 August 2016

Academic Editor: Saeid Sanei

Copyright © 2016 L. L. Neves and L. H. A. Monteiro. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Let a neuronal population be composed of an excitatory group interconnected to an inhibitory group. In the Wilson-Cowan model, the activity of each group of neurons is described by a first-order nonlinear differential equation. The source of the nonlinearity is the interaction between these two groups, which is represented by a sigmoidal function. Such a nonlinearity makes difficult theoretical works. Here, we analytically investigate the dynamics of a pair of coupled populations described by the Wilson-Cowan model by using a linear approximation. The analytical results are compared to numerical simulations, which show that the trajectories of this fourth-order dynamical system can converge to an equilibrium point, a limit cycle, a two-dimensional torus, or a chaotic attractor. The relevance of this study is discussed from a biological perspective.

1. Introduction

In the last decades, the features of action potentials propagating along axonal membranes have been related to the oscillations found in electroencephalogram (EEG) records [1–3]. From a theoretical perspective, analogies between EEG and nonlinear dynamics [4] were formulated prior to knowledge of the seminal numerical simulation by Lorenz, published in 1963, about a chaotic system [5]. All these studies aim to disclose the neural code; that is, to understand how information is represented, carried, and transformed by neurons [6]. The ambition is to explain how cognitive functions emerge from neuron spikes. In this context, here we analytically investigate the activity of a simple neuronal assembly, in order to examine how its parameter values influence its dynamical behavior.

Consider a population formed by two groups of interacting neurons, so that the synapses of the first group are excitatory and the synapses of the second group are inhibitory. Consider also that these two groups are connected. In 1972, Wilson and Cowan proposed a mathematical model

to describe the time evolution of the activity of this neuronal population [7]. In this model, the activity $g(t)$ of each group obeys a nonlinear first-order ordinary differential equation with the following form:

$$\frac{dg(t)}{dt} = -kg(t) + [1 + rg(t)]S(z(t)) \quad (1)$$

in which t denotes the time, k is a positive constant related to the natural decay of $g(t)$, r is a positive constant proportional to the refractory period, and $z(t)$ represents the input to this group, written as a linear combination of the activities of both groups plus the influence of external stimuli. Resting activity corresponds to $g = 0$; thus, a negative value of g expresses a depression from this background level. The function $S(z)$ must be sigmoidal. However, as stated by Wilson and Cowan [7], “no particular significance is to be attached to the choice of” $S(z)$. Usual choices are $1/(1 + e^{-z}) - 1/2$ [8], $\tanh(z)$ [9], and $\arctan(z)$ [10]. In several studies, the properties of a single population were explored via numerical simulations [8, 9, 11], since these usual sigmoidal functions pose difficulties to

analytical works. The dynamics of the two-population model was also already numerically investigated [10, 12–15].

Monteiro et al. suggested to use $S(z) = z/\sqrt{1+z^2}$ and some analytical results were derived for the Wilson-Cowan model [16]. Here, we also take this particular form of $S(z)$ to analytically investigate the dynamics of two coupled Wilson-Cowan populations (composed of four groups). We also assume that (1) the isolated populations are identical (in the sense that they are characterized by the same parameter values, as considered in other studies [10, 12–15]); (2) the populations are coupled by links starting from their excitatory groups (because most synapses are excitatory; for instance, they are 84% in the cat cortex [17]); (3) the refractory period is negligible (which corresponds to taking $r = 0$ in (1), as supposed in several works [9, 10, 12, 14, 18–20]).

Some authors took $S(z)$ as a piecewise linear function [14, 19–21]. In our analyses, we assume that the parameter values related to this neuronal assembly allow us to take $S(z) \approx z$; thus, the model becomes linear. From this approximation, the occurrence of a Hopf bifurcation in the nonlinear model is analytically inferred. Other bifurcations are numerically found. Recall that bifurcation corresponds to a qualitative change in the dynamical behavior of a system caused by variation of parameter value(s).

This manuscript about the dynamics of a pair of coupled Wilson-Cowan neuronal populations is structured as follows. In Section 2, the model is presented and analyzed by taking into account the linear approximation just described. In Section 3, the analytical results are compared to numerical simulations performed by considering the nonlinear version of the model. We found that, as the time passes, the variables of the model can converge to an equilibrium point, a limit cycle, a two-dimensional torus, or a chaotic attractor. In Section 4, the results of this work are discussed from a biological point of view.

2. Analytical Results

Let two identical populations be coupled by links that can be symmetrical or asymmetrical. Assume that the variables $x_i(t)$ and $y_i(t)$ denote the activity of the excitatory group and the activity of the inhibitory group, respectively, of i th population, with $i = 1, 2$. According to Wilson and Cowan, the dynamics of these coupled populations can be described by

$$\begin{aligned} \frac{dx_1(t)}{dt} &= -ax_1(t) \\ &+ \frac{wx_1(t) - by_1(t) + \alpha_1 x_2(t) + I_1}{\sqrt{1 + (wx_1(t) - by_1(t) + \alpha_1 x_2(t) + I_1)^2}}, \\ \frac{dy_1(t)}{dt} &= -dy_1(t) \\ &+ \frac{cx_1(t) - ey_1(t) + \beta_1 x_2(t) + J_1}{\sqrt{1 + (cx_1(t) - ey_1(t) + \beta_1 x_2(t) + J_1)^2}}, \end{aligned}$$

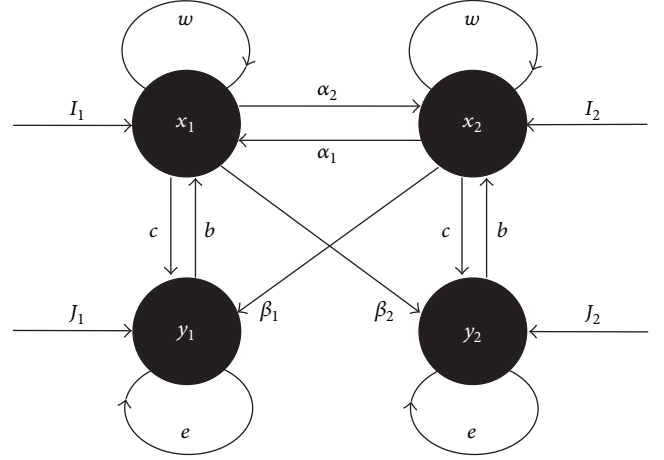


FIGURE 1: Schematic representation of the two-population model. The variables of the model are the activities of the neuronal groups x_1 , x_2 , y_1 , and y_2 . The connection strengths are denoted by b , c , e , w , α_1 , α_2 , β_1 , and β_2 . Stimuli from other sources correspond to I_1 , I_2 , J_1 , and J_2 .

$$\begin{aligned} \frac{dx_2(t)}{dt} &= -ax_2(t) \\ &+ \frac{wx_2(t) - by_2(t) + \alpha_2 x_1(t) + I_2}{\sqrt{1 + (wx_2(t) - by_2(t) + \alpha_2 x_1(t) + I_2)^2}}, \\ \frac{dy_2(t)}{dt} &= -dy_2(t) \\ &+ \frac{cx_2(t) - ey_2(t) + \beta_2 x_1(t) + J_2}{\sqrt{1 + (cx_2(t) - ey_2(t) + \beta_2 x_1(t) + J_2)^2}}. \end{aligned} \quad (2)$$

The parameters a and d represent the natural (exponential) decay. The parameters b , c , e , and w are the strengths of the connections between the excitatory and inhibitory groups of an isolated population, as shown in Figure 1: b is the inhibitory connection from y_i to x_i , c is the excitatory connection from x_i to y_i , w is the self-excitatory connection (from x_i to x_i), and e is the self-inhibitory connection (from y_i to y_i). The strengths of the connections between the populations are denoted by the parameters α_i and β_i : α_1 is the connection from x_2 to x_1 , α_2 is the connection from x_1 to x_2 , β_1 is the connection from x_2 to y_1 , and β_2 is the connection from x_1 to y_2 . All these ten parameters are positive constants. The constants I_1 , J_1 , I_2 , and J_2 stand for stimuli from external sources reaching x_1 , y_1 , x_2 , and y_2 , respectively. Thus, the dimension of the parameter space is 14.

A steady state is a stationary solution, corresponding to an equilibrium point $(x_1^*, y_1^*, x_2^*, y_2^*)$ in the four-dimensional state space $x_1 \times y_1 \times x_2 \times y_2$. The constants x_1^* , y_1^* , x_2^* , and y_2^* are obtained from $dx_1/dt = 0$, $dy_1/dt = 0$, $dx_2/dt = 0$, and $dy_2/dt = 0$. By taking into consideration the linear approximation proposed in Section 1, there is only one equilibrium point given by

$$\begin{aligned}
x_1^* &= \frac{(bc - EW)(EI_1 - bJ_1) + (EI_2 + bJ_2)(E\alpha_1 - b\beta_1)}{(bc - EW)^2 + (E\alpha_1 - b\beta_1)(b\beta_2 - E\alpha_2)}, \\
y_1^* &= \frac{(bc - EW)(cI_1 - WJ_1) + (EI_2 - bJ_2)(c\alpha_1 - W\beta_1) + (b\beta_2 - E\alpha_2)(\alpha_1 J_1 - \beta_1 I_1)}{(bc - EW)^2 + (E\alpha_1 - b\beta_1)(b\beta_2 - E\alpha_2)}, \\
x_2^* &= \frac{(bc - EW)(EI_2 - bJ_2) + (bJ_1 - EI_1)(b\beta_2 - E\alpha_2)}{(bc - EW)^2 + (E\alpha_1 - b\beta_1)(b\beta_2 - E\alpha_2)}, \\
y_2^* &= \frac{(bc - EW)(cI_2 - WJ_2) + (EI_1 - bJ_1)(c\alpha_2 - W\beta_2) + (\beta_2 I_2 - \alpha_2 J_2)(E\alpha_1 - b\beta_1)}{(bc - EW)^2 + (E\alpha_1 - b\beta_1)(b\beta_2 - E\alpha_2)},
\end{aligned} \tag{3}$$

with $W \equiv w - a$ and $E \equiv d + e$. Note that if the populations are isolated, that is, if $\alpha_i = \beta_i = 0$, then $x_i^* = (EI_i - bJ_i)/(bc - EW)$ and $y_i^* = (cI_i - WJ_i)/(bc - EW)$, for $i = 1, 2$. Obviously, if $I_1 = I_2$ and $J_1 = J_2$, then $x_1^* = x_2^*$ and $y_1^* = y_2^*$.

The local stability of an equilibrium point can be determined from the eigenvalues λ of the Jacobian matrix obtained from the system of (2) linearized around this point [22–24]. By the Hartman-Grobman theorem, such a point is locally asymptotically stable if all eigenvalues have negative real parts [22–24]. For this fourth-order system, the eigenvalues λ are the roots of the polynomial:

$$\lambda^4 + \gamma_1 \lambda^3 + \gamma_2 \lambda^2 + \gamma_3 \lambda + \gamma_4 = 0, \tag{4}$$

with

$$\begin{aligned}
\gamma_1 &= 2(E - W), \\
\gamma_2 &= (E - W)^2 + 2(bc - EW) - \alpha_1 \alpha_2, \\
\gamma_3 &= 2(E - W)(bc - EW) + b(\alpha_1 \beta_2 + \alpha_2 \beta_1) \\
&\quad - 2E\alpha_1 \alpha_2, \\
\gamma_4 &= (bc - EW)^2 + bE(\alpha_1 \beta_2 + \alpha_2 \beta_1) - b^2 \beta_1 \beta_2 \\
&\quad - E^2 \alpha_1 \alpha_2.
\end{aligned} \tag{5}$$

Routh-Hurwitz criterion states that all eigenvalues have negative real parts if $\gamma_1 > 0$, $\gamma_2 > 0$, $\gamma_3 > 0$, $\gamma_4 > 0$, $\delta_1 \equiv (\gamma_1 \gamma_2 - \gamma_3)/\gamma_1 > 0$, and $\delta_2 \equiv (\delta_1 \gamma_3 - \gamma_1 \gamma_4)/\delta_1 > 0$ [25]. For instance, in the case of isolated populations, local stability of the steady-state solution is assured if $E > W$ and $bc > EW$.

Observe that the stability conditions do not depend on the values of I_i and J_i . Thus, constant external stimuli do not modify the stability of the stationary solution (in the linear approximation). Observe also that a necessary condition for stability is $E > W$; that is, $w < w_c \equiv a + d + e$ or $e > e_c \equiv w - (a + d)$. Therefore, the equilibrium point is locally asymptotically stable only if the strength of the self-excitatory connection is below the critical value w_c and/or if the strength of the self-inhibitory connection is above the critical value e_c .

If $\alpha_1 = \alpha_2 = 0$, then γ_4 diminishes as β_1 and/or β_2 increase; if $\beta_1 = \beta_2 = 0$, then γ_2 , γ_3 , and γ_4 diminish as α_1 and/or α_2 increase. Thus, both kinds of connections

between the populations, characterized by α_i and β_i , can singly destabilize the equilibrium point, if their strengths exceed critical numbers.

If the parameter values are varied so that $\delta_2 = 0$, then a Hopf bifurcation [22–24] can occur (because two roots of (4) are purely imaginary complex-conjugate numbers and the other two roots have negative real parts; i.e., (4) can be written as $(\lambda^2 + A\lambda + B)(\lambda^2 + C) = 0$, with $A, B, C > 0$). As a consequence, a limit cycle with oscillation period $\tau \approx 2\pi/\sqrt{C}$ appears. Recall that a limit cycle is an isolated closed trajectory in the state space, corresponding to a periodic solution [22–24].

If $\alpha_1 = \alpha_2 = \alpha$ and $\beta_1 = \beta_2 = \beta$, the condition $\delta_2 = 0$ is equivalent to $\epsilon_1 \equiv (w + \alpha) - (a + d + e) = 0$. In this particular case, there is the birth of a limit cycle for $\epsilon_1 = 0$ if $\epsilon_2 \equiv b(c + \beta) - (w + \alpha - a)(d + e) > 0$. The period τ of the newborn cycle is approximately given by $\tau \approx 2\pi/\sqrt{\epsilon_2}$.

As shown in the next section, other bifurcations can take place by varying the parameter values of this dynamical system.

3. Numerical Simulations

In the simulations presented in this section, the values of $a, b, c, d, e, I_1, I_2, J_1$, and J_2 are kept fixed and the values of $w, \alpha_1, \alpha_2, \beta_1$, and β_2 are varied. Thus, we investigate how the dynamics is influenced by the strengths of the self-excitatory loops and of the links between the populations.

Here, we take $a = d = 0.01$, $b = 20$, $c = 10$, $e = 10$, $I_1 = 2$, $I_2 = 1$, and $J_1 = J_2 = 0$. In Figures 2(a)–2(i), only $x_1(t)$ is plotted in function of t . In each case, the behaviors of the other three variables are qualitatively similar; hence, their plots were omitted. For instance, if $x_1(t)$ converges to a periodic solution, then $y_1(t)$, $x_2(t)$, and $y_2(t)$ also tend to a periodic oscillation as the time passes. In (a), $w = 8$, $\alpha_1 = \alpha_2 = \beta_1 = \beta_2 = 0$; in (b), $w = 12$, $\alpha_1 = \alpha_2 = \beta_1 = \beta_2 = 0$; in (c), $w = 8$, $\alpha_1 = \alpha_2 = 1$, $\beta_1 = \beta_2 = 0$; in (d), $w = 8$, $\alpha_1 = \alpha_2 = 3$, and $\beta_1 = \beta_2 = 0$; in (e), $w = 8$, $\alpha_1 = \alpha_2 = 1$, and $\beta_1 = 0$, $\beta_2 = 3$; in (f), $w = 12$, $\alpha_1 = \alpha_2 = 1$, and $\beta_1 = \beta_2 = 0$; in (g), $w = 12$, $\alpha_1 = \alpha_2 = 1$, and $\beta_1 = \beta_2 = 2$; in (h), $w = 13$, $\alpha_1 = \alpha_2 = 3$, and $\beta_1 = \beta_2 = 0$; and in (i), $w = 13$, $\alpha_1 = \alpha_2 = 3$, and $\beta_1 = \beta_2 = 2$. In these simulations,

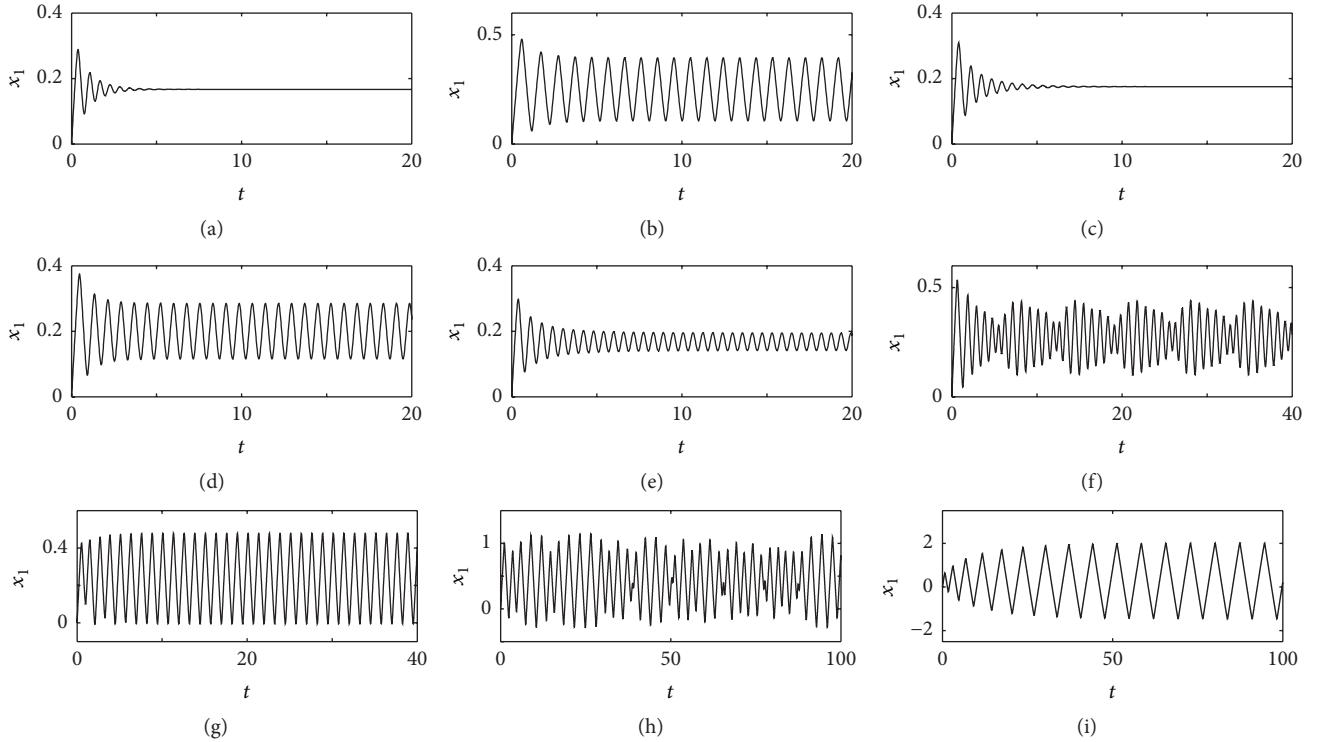


FIGURE 2: Temporal evolutions of $x_1(t)$ obtained by numerically integrating (2). In all cases, the initial condition is the origin of the state space. In all cases, $a = d = 0.01$, $b = 20$, $c = 10$, $e = 10$, $I_1 = 2$, $I_2 = 1$, and $J_1 = J_2 = 0$. In (a), $w = 8$, $\alpha_1 = \alpha_2 = \beta_1 = \beta_2 = 0$; in (b), $w = 12$, $\alpha_1 = \alpha_2 = \beta_1 = \beta_2 = 0$; in (c), $w = 8$, $\alpha_1 = \alpha_2 = 1$, and $\beta_1 = \beta_2 = 0$; in (d), $w = 8$, $\alpha_1 = \alpha_2 = 3$, and $\beta_1 = \beta_2 = 0$; in (e), $w = 8$, $\alpha_1 = \alpha_2 = 1$, $\beta_1 = 0$, and $\beta_2 = 3$; in (f), $w = 12$, $\alpha_1 = \alpha_2 = 1$, and $\beta_1 = \beta_2 = 0$; in (g), $w = 12$, $\alpha_1 = \alpha_2 = 1$, and $\beta_1 = \beta_2 = 2$; in (h), $w = 13$, $\alpha_1 = \alpha_2 = 3$, and $\beta_1 = \beta_2 = 0$; and in (i), $w = 13$, $\alpha_1 = \alpha_2 = 3$, and $\beta_1 = \beta_2 = 2$. In (a) and (c), the system converges to an equilibrium point; in (b), (d), (e), (g), and (i) to a limit cycle; in (f) to a two-dimensional torus; and in (h) to a chaotic attractor.

(2) were numerically solved by employing the fourth-order Runge-Kutta integration method with integration step of 0.01. In addition, in all simulations, the initial condition is $(x_1(0), y_1(0), x_2(0), y_2(0)) = (0, 0, 0, 0)$.

In (a) and (b), the populations are isolated. Therefore, the attractor can be either an equilibrium point or a limit cycle, because (2) split into two decoupled second-order systems. In fact, these are the attractors that can exist in the state space of second-order autonomous nonlinear systems [22–24].

In (a), the conditions for the asymptotical stability of the steady-state solution are satisfied (because $E = 10.01 > W = 7.99$ and $bc = 200 > EW \approx 80.0$). In this case, $x_1(t)$ converges to 0.167, which is the same value obtained from the analytical expression $x_1^* = (EI_1 - bJ_1)/(bc - EW)$. Thus, the linear approximation of the sigmoidal function $S(z)$ can be considered as valid to determine this equilibrium point and its stability. For $w = w_c = 10.02$, the system experiences a Hopf bifurcation. Hence, in (b), as $w = 12 > w_c$, $x_1(t)$ tends to a periodic solution.

In cases (c)–(i), the populations are coupled. Now, the nature of the attractor is determined from its Lyapunov exponents L_1 , L_2 , L_3 , and L_4 , which were numerically computed by using the algorithm proposed by Wolf et al. [26]. When $L_{1,2,3,4} < 0$, the attractor is an equilibrium point; when $L_1 = 0$ and $L_{2,3,4} < 0$, the attractor is a limit cycle; when $L_1 = L_2 = 0$ and $L_{3,4} < 0$, the attractor is a two-dimensional

torus; when $L_1 > 0$, $L_2 = 0$, and $L_{3,4} < 0$, the attractor is chaotic [22–24].

In (c), the Routh-Hurwitz criterion is satisfied (because $\gamma_1 = 4.04 > 0$, $\gamma_2 \approx 243 > 0$, $\gamma_3 \approx 465$, $\gamma_4 \approx 14300 > 0$, $\delta_1 \approx 128 > 0$, and $\delta_2 \approx 13.6 > 0$) and x_1 converges to 0.175, which matches the number calculated from (3). In this case, $L_{1,2} \approx -0.52$ and $L_3 \approx L_4 = -1.51$.

As stated in Section 2, by varying the parameter values, if δ_2 becomes equal to zero, then a Hopf bifurcation can take place. For instance, for $w = 8 < w_c$ and $\beta_1 = \beta_2 = 0$, then $\delta_2 = 0$ if $\alpha_1 = \alpha_2 = \alpha_c = 2.02$; thus, for $\alpha_1 = \alpha_2 = 3 > \alpha_c$, the attractor is a limit cycle, as shown in (d). In this case, $L_1 \approx 0.00$, $L_2 \approx -0.67$, $L_3 \approx -1.48$, and $L_4 \approx -3.32$. Recall that $\delta_2 = 0$ is equivalent to $\epsilon_1 \equiv (w + \alpha) - (a + d + e) = 0$ when $\alpha_1 = \alpha_2 = \alpha$ and $\beta_1 = \beta_2 = \beta$; thus, $\epsilon_1 = 0$ corresponds to $\alpha_c = a + d + e - w = 2.02$, which is equal to the value numerically found. For $\alpha = \alpha_c$, the oscillation period is about $2\pi/\sqrt{\epsilon_2} \approx 0.63$. In (d), for $\alpha = 3$, the period is still 0.63.

Even when $w = 8 < w_c$ and $\alpha_1 = \alpha_2 = 1 < \alpha_c$, a limit cycle arises if $\beta_2 > \beta_c = 2.25$. Hence, in (e), with $\beta_2 = 3 > \beta_c$, the asymptotical behavior is a regular oscillation. It corresponds to a limit cycle, because $L_1 \approx 0.00$, $L_2 \approx -0.31$, and $L_{3,4} \approx -2.16$.

It is easy to check that the steady state loses its stability when $W > E$, that is, when $w > w_c$. In (f), with $w = 12 > w_c$, $\alpha_1 = \alpha_2 = 1$, and $\beta_1 = \beta_2 = 0$, the trajectories in the

state space converge to a two-dimensional torus. In fact, the Lyapunov exponents are $L_1 \approx L_2 \approx 0.00$ and $L_3 \approx L_4 \approx -0.55$. Therefore, by increasing w , the system experiences a bifurcation concerning the birth of a toroidal attractor. In (g), by taking $\beta_1 = \beta_2 = 2$, the trajectories tend to a limit cycle, with $L_1 \approx 0.00$, $L_{2,3} \approx -0.54$, and $L_4 \approx -0.58$. Thus, by increasing β_1 and β_2 , the transition from torus towards cycle occurs.

In (h), for $w = 13 > w_c$, $\alpha_1 = \alpha_2 = 3 > \alpha_c$, and $\beta_1 = \beta_2 = 0$, the attractor is chaotic, because $L_1 \approx 0.28$, $L_2 \approx 0.00$, $L_3 \approx -0.62$, and $L_4 \approx -1.40$. Observe that, when compared to (d), by increasing w , a transition from regular oscillation towards chaotic solution can occur. For these values of α_i and β_i , there is chaos if $12.5 \leq w \leq 16.5$. In (i), by taking $\beta_1 = \beta_2 = 2$, the attractor comes back to be a limit cycle, because $L_1 \approx 0.00$, $L_2 \approx -0.07$, and $L_{3,4} \approx -0.15$. Thus, by increasing the values of β_1 and β_2 , a bifurcation related to the transition from chaos to periodic behavior takes place.

Starting from a stationary solution, regular oscillations can be created by increasing w (please see (a) \rightarrow (b) and (c) \rightarrow (f)), or α_i (see (c) \rightarrow (d)), or β_i (see (c) \rightarrow (e)). From a periodic behavior, chaos can appear by a similar way (see (d) \rightarrow (h)). Thus, by enhancing the excitatory connections, the activities of both populations can regularly or irregularly oscillate. However, when the strength of any excitatory connection is “too high,” the linear approximation of $S(z)$ can no longer be valid. In this scenario, a suitable approximation is $|S(z)| \approx 1$ (because $|S(z)| \rightarrow 1$ when $|z| \rightarrow \infty$). Therefore, from (2), $|x_i| \rightarrow 1/a$ and $|y_i| \rightarrow 1/d$ as the time passes. For instance, by simulating the system with $w = 20$, $\alpha_1 = \alpha_2 = 3$, and $\beta_1 = \beta_2 = 0$, the variables asymptotically converge to $x_1^* \approx x_2^* \approx 99.3$ and $y_1^* \approx y_2^* \approx 98.5$.

It is important to stress that the attractors and bifurcations reported in this section were already found in earlier works based on computer simulations on two-population models [10, 13–15]. We hope that the analytical and numerical results presented here can guide experimental researches on the detection of such attractors and bifurcations in actual neural networks, in particular the ones underlying working memory, as discussed in the next section.

4. Discussion

In this work, we explicitly derived analytical conditions concerning the stability of the steady state derived from the approximation $S(z) \approx z$. Now, we discuss the possible relevance of this result.

Neural codes based on chaotic activity have been proposed [27–29]. We observed here that chaos can be found in a simple neuronal assembly composed of two coupled Wilson-Cowan populations subject to constant stimuli, when the strengths of the excitatory connections are above critical numbers. Since excitatory synapses are more often encountered than inhibitory ones [17], then these strengths can naturally be above such critical numbers. Thus, the irregular activity commonly found in EEG would be, at least partially, a simple consequence of the neuronal connectivity, in particular, the predominance of excitatory synapses.

The strengths of excitatory connections, however, cannot be “too high,” because in this case $S(z) \approx 1$ and the activity would tend to be stationary. Thus, the balance between excitation and inhibition must satisfy constraints assuring normal oscillatory behavior. Abnormalities in this balance in the cortical circuitry have been associated with neurological disorders, such as autism and epilepsy [30, 31].

The conditions presented in Section 2 can guide laboratory experiments, in order to verify their validity. Observe that these analytical expressions were derived by supposing that $S(z) \approx z$. It is far from being trivial to find the regions in the 14-dimensional parameter space where this approximation holds. However, from (1) with $r = 0$, note that $dg/dt = 0$ implies $kg^* = S(z^*)$. Since $S(z) = z/\sqrt{1+z^2} \approx z$ only if $z \ll 1$, then $kg^* \approx z^* \ll 1$ in the steady-state condition. Hence, in the numerical simulations, we took $a = d = 0.01 \ll 1$ (obviously, k in (1) is equivalent to a and d in (2)). Neuronal populations with “slow” exponential decay (i.e., $a, d \ll 1$) can be responsible for supporting working memory [32]. Hence, the validity of our conclusions could be first tested in such populations. Perhaps, these tests can reveal the true nature of the attractors and bifurcations involved in maintaining and manipulating new information.

Competing Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

Acknowledgments

Lucas L. Neves is partially supported by CAPES and Luiz H. A. Monteiro by CNPq.

References

- [1] A. K. Engel, P. König, and T. B. Schillen, “Why does the cortex oscillate?” *Current Biology*, vol. 2, no. 6, pp. 332–334, 1992.
- [2] E. Basar, C. Basar-Eroglu, S. Karakas, and M. Schürmann, “Oscillatory brain theory: a new trend in neuroscience: the role of oscillatory processes in sensory and cognitive functions,” *IEEE Engineering in Medicine and Biology Magazine*, vol. 18, no. 3, pp. 56–66, 1999.
- [3] W. J. Freeman, “Understanding perception through neural ‘codes,”” *IEEE Transactions on Biomedical Engineering*, vol. 58, no. 7, pp. 1884–1890, 2011.
- [4] E. M. Dewan, “Nonlinear oscillations and electroencephalography,” *Journal of Theoretical Biology*, vol. 7, no. 1, pp. 141–159, 1964.
- [5] E. N. Lorenz, “Deterministic nonperiodic flow,” *Journal of the Atmospheric Sciences*, vol. 20, no. 2, pp. 130–141, 1963.
- [6] R. C. deCharms and A. Zador, “Neural representation and the cortical code,” *Annual Review of Neuroscience*, vol. 23, pp. 613–647, 2000.
- [7] H. R. Wilson and J. D. Cowan, “Excitatory and inhibitory interactions in localized populations of model neurons,” *Biophysical Journal*, vol. 12, no. 1, pp. 1–24, 1972.

- [8] R. M. Borisjuk and A. B. Kirillov, "Bifurcation analysis of a neural network model," *Biological Cybernetics*, vol. 66, no. 4, pp. 319–325, 1992.
- [9] P. E. Latham, B. J. Richmond, P. G. Nelson, and S. Nirenberg, "Intrinsic dynamics in neuronal networks. I. Theory," *Journal of Neurophysiology*, vol. 83, no. 2, pp. 808–827, 2000.
- [10] Y. Hayashi, "Numerical bifurcation analysis of an oscillatory neural network with synchronous/asynchronous connections," *Neural Computation*, vol. 6, no. 4, pp. 658–667, 1994.
- [11] D. Wang, "Emergent synchrony in locally coupled neural oscillators," *IEEE Transactions on Neural Networks*, vol. 6, no. 4, pp. 941–948, 1995.
- [12] G. B. Ermentrout and N. Kopell, "Oscillator death in systems of coupled neural oscillators," *SIAM Journal on Applied Mathematics*, vol. 50, no. 1, pp. 125–146, 1990.
- [13] G. N. Borisjuk, R. M. Borisjuk, A. I. Khibnik, and D. Roose, "Dynamics and bifurcations of two coupled neural oscillators with different connection types," *Bulletin of Mathematical Biology*, vol. 57, no. 6, pp. 809–840, 1995.
- [14] T. Ueta and G. Chen, "On synchronization and control of coupled Wilson-Cowan neural oscillators," *International Journal of Bifurcation and Chaos in Applied Sciences and Engineering*, vol. 13, no. 1, pp. 163–175, 2003.
- [15] Y. Maruyama, Y. Kakimoto, and O. Araki, "Analysis of chaotic oscillations induced in two coupled Wilson-Cowan models," *Biological Cybernetics*, vol. 108, no. 3, pp. 355–363, 2014.
- [16] L. H. A. Monteiro, M. A. Bussab, and J. G. Chaui Berlinck, "Analytical results on a Wilson-Cowan neuronal network modified model," *Journal of Theoretical Biology*, vol. 219, no. 1, pp. 83–91, 2002.
- [17] C. Beaulieu and M. Colonnier, "A laminar analysis of the number of round-asymmetrical and flat-symmetrical synapses on spines, dendritic trunks, and cell bodies in area 17 of the cat," *Journal of Comparative Neurology*, vol. 231, no. 2, pp. 180–189, 1985.
- [18] H. G. Schuster and P. Wagner, "A model for neuronal oscillations in the visual cortex-I. Mean-field theory and derivation of the phase equations," *Biological Cybernetics*, vol. 64, no. 1, pp. 77–82, 1990.
- [19] M. V. Tsodyks, W. E. Skaggs, T. J. Sejnowski, and B. L. McNaughton, "Paradoxical effects of external modulation of inhibitory interneurons," *The Journal of Neuroscience*, vol. 17, no. 11, pp. 4382–4388, 1997.
- [20] E. Ledoux and N. Brunel, "Dynamics of networks of excitatory and inhibitory neurons in response to time-dependent inputs," *Frontiers in Computational Neuroscience*, vol. 5, article 25, 2011.
- [21] A. Muir, "A simple case of the Wilson-Cowan equations," *Biophysical Journal*, vol. 27, no. 2, pp. 267–275, 1979.
- [22] J. Argyris, G. Faust, M. Haase, and R. Friedrich, *An Exploration of Dynamical Systems and Chaos*, Springer, Berlin, Germany, 2nd edition, 2015.
- [23] L. H. A. Monteiro, "Overview of dynamical systems and chaos," in *Chaotic Signals in Digital Communications*, M. Eisenkraft, R. Attux, and R. Suyama, Eds., pp. 83–109, CRC Press, Boca Raton, Fla, USA, 2014.
- [24] H. R. Wilson, *Spikes, Decisions, and Actions: The Dynamical Foundations of Neuroscience*, Oxford University Press, Oxford, UK, 1999.
- [25] K. Ogata, *Modern Control Engineering*, Prentice Hall, Upper Saddle River, NJ, USA, 2009.
- [26] A. Wolf, J. B. Swift, H. L. Swinney, and J. A. Vastano, "Determining Lyapunov exponents from a time series," *Physica D. Nonlinear Phenomena*, vol. 16, no. 3, pp. 285–317, 1985.
- [27] I. Tsuda, "Toward an interpretation of dynamic neural activity in terms of chaotic dynamical systems," *Behavioral and Brain Sciences*, vol. 24, no. 5, pp. 793–810, 2001.
- [28] H. Korn and P. Faure, "Is there chaos in the brain? II. Experimental evidence and related models," *Comptes Rendus—Biologies*, vol. 326, no. 9, pp. 787–840, 2003.
- [29] M. V. Kiselev, "Homogenous chaotic network serving as a rate/population code to temporal code converter," *Computational Intelligence and Neuroscience*, vol. 2014, Article ID 476580, 8 pages, 2014.
- [30] O. Yizhar, L. E. Fenno, M. Prigge et al., "Neocortical excitation/inhibition balance in information processing and social dysfunction," *Nature*, vol. 477, no. 7363, pp. 171–178, 2011.
- [31] C. E. Stafstrom and T. A. Benke, "Autism and epilepsy: exploring the relationship using experimental models," *Epilepsy Currents*, vol. 15, no. 4, pp. 206–210, 2015.
- [32] Z. Tiganj, M. E. Hasselmo, and M. W. Howard, "A simple biophysically plausible model for long time constants in single neurons," *Hippocampus*, vol. 25, no. 1, pp. 27–37, 2015.