Phase dynamics matching structure in plastic neuronal network with transmission delays

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Synaptic time delays and plasticity are intrinsic characteristics present in real neuronal networks, and the interplay between both can promote complex dynamics in the brain. In this work, we build connected plastic subnetworks of Hodgkin-Huxley neurons where the subnetworks are composed of excitatory neurons and the connectivity modifications follow a spike-time dependent rule. We define an internal-delay among neurons communicating within the same subnetwork, an external-delay for neurons belonging to distinct subnetworks, and study how these communicating delays affect the entire network dynamics. In the absence of plasticity the subnetworks can exhibit synchronous behaviour with different collective patterns that depend on the synaptic external delays. With plasticity, we observe that the neuronal network exhibits a specific connectivity configuration for each synchronised pattern. Our results show how synaptic delays and plasticity work together to promote the formation of structural coupling among the neuronal networks. Besides that, collective synchronous activities strongly reflect the underlying topological connectivity of the neurons in a plastic neuronal network where delays provides the rich environment for plasticity to shape the network topology. Finally, plasticity promotes equivalence between function and structure.

Key-words: Synaptic plasticity; Hodgkin-Huxley model; Neural dynamics; Synchronization

1 Introduction

Signal transmission delays are an intrinsic property of neuronal communication and are extremely relevant in brain activities [32, 39]. [4] highlighted the importance of realistic time delays, emphasising that this property has just been marginally taking in account in the used models over the last decades. Different transmission or synaptic delays can generate desynchronised and synchronised neuronal activities [27, 25, 34, 14]. [43] demonstrated that certain intra and inter-networks delays can facilitate fast regular firings. [23] pointed to a possible role of facilitation dynamics to compensate such delays in motor neurons by a systematic approach. [6] reported the role of delayed feedback to control burst synchronisation in a scale-free neuronal network.

Synaptic plasticity is the fundamental ability of the neurons to change their connection intensities due to spike activities [37]. It has been reported that memory and learning functions are supported by synaptic plasticity in the brain [2]. Experimental data suggest that synchronous spike dynamics patterns can be intensified due to the plasticity, being associated with long-term memory processing. However, it is not completely clear how plasticity and synchronisation are related [13].

[18] studied the stochastic spike synchronization in a small-world neural network in the presence of excitatory spike time-dependent plasticity (STDP). They observed a Matthew effect in synaptic plasticity due a positive feedback mechanism. In a similar framework, they also study the effect of inhibitory STDP for the same network topology showing that both depression and potentiation of inhibitory connections can occur [19]. Studying the cerebellar ring network with synaptic plasticity, they still found that phase, anti-phase, and complex out-of-phase activities are involved in the long-term depression [20]. [12] demonstrated the relationship between local Hebbian plasticity and learning using a computational approach focus on the noise and weight saturation.

[3] demonstrated the self-organisation phenomena in a recurrent network of oscillators in the presence of synaptic...
plasticity identifying phase, anti-phase, coherent and chaotic activities. Phase, anti-phase and phase-lock activities are the main types of synchronisation observed between brain regions [16, 15, 12, 36, 21] and reported the importance of phase synchronisation in various cognitive processes. Phase synchronisation has been observed in distant cortical areas with long conduction delays [22]. Some works also showed that time delay, synaptic types and connection densities play an important role in anti-phase synchronisation [20, 8].

In this work, we study the emergence of synchronised symmetric patterns between the subnetworks depend on the absence and presence of long-term plasticity, and on internal and external transmission delays. Considering that internal delays among neurons inside a subnetwork are smaller than the external ones between subnetworks, we show that distinct patterns of synchronisation can be achieved as we change these delays. In the presence of plasticity, the synaptic connections between the subnetworks evolve from initially random to a more structured configuration where we can identify the stronger connections between the subnetworks associated to specific synchronised patterns. The increase of internal delays reduce the synchronous patterns inside the subnetworks. External delays, on the contrary, can promote collective synchronisation. Subnetworks in one group symmetry whose neurons are connected with small external delays are less synchronous than those neurons connected via large delays. Besides that, the synchronisation in one group is higher than for two, three, and four group symmetry. Our results also suggest that synchronous behaviour reveals the structure being created by the plastic network. More specifically, functional communities and their connections inferred by measurements of neural phase synchronization reflect the subnetworks and their linkage structure provided by the structural topology of synapses.

The paper is organised as follows: In Section 2 we introduce our plastic neuronal network of coupled HH neurons and the diagnostic tool to identify synchronisation. In Section 3 we present the results of our study about the effects of synaptic delays between neuronal subnetworks. In the last section, we draw our conclusions.

## 2 Plastic neuronal network

### 2.1 Hodgkin-Huxley model

We consider the type-II neuron model proposed by [15]. The individual dynamics of each Hodgkin-Huxley (HH) neuron in the network is given by

\[
CV_i = I_i - g_K n_i^4(v_i - E_K) - g_{Na} m_i^3 h_i (v_i - E_{Na}) - g_L (v_i - E_L)
+ (V_i^+ - V_i) \sum_{j=1}^N g_{ij} f_j(t - \tau_{ij}), \tag{1}
\]

\[
\dot{n}_i = \alpha_n(v_i)(1 - n_i) - \beta_n(v_i)n_i, \tag{2}
\]

\[
\dot{m}_i = \alpha_m(v_i)(1 - m_i) - \beta_m(v_i)m_i, \tag{3}
\]

\[
\dot{h}_i = \alpha_h(v_i)(1 - h_i) - \beta_h(v_i)h_i, \tag{4}
\]

\[
\dot{f}_i = \frac{-f_i}{\tau_s}, \tag{5}
\]

Equation (1) represents the membrane dynamics of the neuron \(i\). \(C (\mu \text{F/cm}^2)\) is the membrane capacitance and \(I_i (\mu \text{A/cm}^2)\) is a constant current density chosen in the interval \([10, 11]\). The parameters \(g_K, g_{Na},\) and \(g_L\) are the conductance of the potassium, sodium, and leak ion channels, respectively. \(E_K, E_{Na},\) and \(E_L\) are the reversal potentials for these ion channels. \(V_i^+\) corresponds to the excitatory reversal potential. \(g_{ij}\) is the excitatory coupling strength from the presynaptic neuron \(j\) to the postsynaptic neuron \(i\) with maximum and minimum value within the interval \([0.0, 0.01]\). We consider that the neuron has no self-connections, implying \(g_{ii} = 0\). \(f_i(t)\) is the normalised synaptic current from the neuron \(j\) to \(i\). The state variable \(f_i\) decays exponentially and it is updated to the unity \((f_i \to 1)\) at the spike time \(t_i\) of the neuron \(i\) [33, 9]. Equation (5) corresponds to an exponential decay on the evolution of \(f_i(t)\). The parameter \(\tau_s\) is the synaptic time decay and \(\tau_{ij}\) the delay on the synaptic transmission [5, 30, 31]. The intensity of synaptic current with time delay on the signal transmission depends on the state of the pre and postsynaptic neuron. \(\tau_{ij}\) assumes values \(\tau_{int}\) and \(\tau_{ext}\) for internal and external connections between the subnetworks, respectively. In Eqs. (2) and (3), the functions \(m(v_i)\) and \(n(v_i)\) represent the sodium and potassium activation, respectively. In Eq. (4), \(h(v_i)\) is the function for sodium inactivation. The functions \(\alpha_n, \beta_n, \alpha_m, \beta_m,\)
\( \alpha_n \) and \( \beta_n \) are given by

\[
\alpha_n(v) = \frac{0.01v + 0.55}{1 - \exp\left(-0.1v - 5.5\right)}, \quad (6)
\]

\[
\beta_n(v) = 0.125 \exp\left(\frac{-v - 65}{80}\right), \quad (7)
\]

\[
\alpha_m(v) = \frac{0.1v + 4}{1 - \exp\left(-0.1v - 4\right)}, \quad (8)
\]

\[
\beta_m(v) = 4 \exp\left(\frac{-v - 65}{18}\right), \quad (9)
\]

\[
\alpha_h(v) = 0.07 \exp\left(\frac{-v - 65}{20}\right), \quad (10)
\]

\[
\beta_h(v) = \frac{1}{1 + \exp\left(-0.1v - 3.5\right)}, \quad (11)
\]

where \( v = V/[\text{mV}] \). In our simulations, we consider \( C = 1 \mu\text{F/cm}^2 \), \( E_{\text{Na}} = 50 \text{ mV} \), \( E_K = -77 \text{ mV} \), \( E_L = -54.4 \text{ mV} \), \( g_{\text{Na}} = 120 \text{ mS/cm}^2 \), \( g_K = 36 \text{ mS/cm}^2 \), \( g_L = 0.3 \text{ mS/cm}^2 \), and \( \tau_s = 2.728 \text{ ms} \). The reversal potential for excitatory connections is \( V_r^+ = 20 \text{ mV} \). For the numerical integration, we use the Runge-Kutta fourth-order method with a time step equal to \( \delta t = 0.01 \text{ ms} \).

### 2.2 Spike-time dependent plasticity

Spike-time dependent plasticity (STDP) is a process that produces changes in the synaptic strength. It is calculated taking into consideration the times between the spikes of the postsynaptic neuron \( t_i \) and the presynaptic neuron \( t_j \). The change in the excitatory synaptic weights \( \Delta g_{ij} \) due to the time difference \( \Delta t_{ij} = t_i - t_j \) is given by \[ \Delta g_{ij} = \begin{cases} A_1 \exp\left(-\frac{\Delta t_{ij}}{\tau_1}\right), & \text{if } \Delta t_{ij} \geq 0 \\ -A_2 \exp\left(\frac{\Delta t_{ij}}{\tau_2}\right), & \text{if } \Delta t_{ij} < 0 \end{cases} \] (12)

where \( A_1 = 1 \), \( A_2 = 0.5 \), \( \tau_1 = 1.8 \text{ ms} \), and \( \tau_2 = 6 \text{ ms} \). The synaptic weights are updated according to Eq. (12), where \( g_{ij} \rightarrow g_{ij} + G \cdot \Delta g_{ij} \). The change rate of the synaptic weight is considered as \( G = 10^{-5} \text{ mS/cm}^2 \). The initial value of all excitatory synaptic weights is given by \( g_{ij} = 0.001 \text{ mS/cm}^2 \).

Figure 1 displays the plasticity curves described by Eq. (12) (red line) as a function of \( \Delta t_{ij} \).

![Figure 1: Plasticity curves as a function of \( \Delta t_{ij} \) for excitatory synapses. Negative and positive plus zero values of \( \Delta t_{ij} \) correspond to synaptic depression and potentiation, respectively.](image-url)
2.3 A network of subnetworks

We consider $N = 400$ non-identical HH neurons separated into $S = 4$ subnetworks with $N_{\text{sub}} = 100$ neurons each one ($N = S \cdot N_{\text{sub}}$). The heterogeneity in the system is given by the neuron currents $I_i$. To facilitate the visualisation and interpretation of our results, we sorted neurons in each subnetwork in ascending order according to their spiking frequency (or $I_i$). Therefore, the neuron $i = 1$ has the slowest spiking frequency and the neuron $i = 100$ has the highest one. The neurons are connected by means of excitatory synapses. For the initial coupling configuration, each subnetwork has an internal all-to-all topology without self-connections (autapses). The connections between subnetworks or external one are random distributed with a certain probability. Thus, the internal and external probability of connections are given by $p_{\text{int}} = 1$ and $p_{\text{ext}} = 0.05$, respectively. New connections are not allowed between subnetworks, however, changes in the weights of initial external connections are permitted. The network does not evolve to a configuration of only one community due to the plasticity due to the fixed internal connection probability between the subnetwork. With regard to the subnetworks, we consider an internal and external transmission delay given by $\tau_{\text{int}}$ and $\tau_{\text{ext}}$, respectively.

2.4 Measuring synchronisation and symmetries

In order to study neuronal synchronisation and symmetries, we compute the order parameter. Firstly, we use the traditional Kuramoto order parameter as a diagnostic tool for the whole network, that is given by

$$R_T(t) = \left| \frac{1}{N} \sum_{j=1}^{N} e^{i\phi_j(t)} \right|,$$  \hspace{1cm} (13)

where "i" is the imaginary unit $\sqrt{-1}$ and $\phi_j(t)$ is the neural phase associated with the spikes of each neuron $j$, given by

$$\phi_j(t) = \frac{2\pi t - t_{j,k}}{t_{j,k+1} - t_{j,k}},$$  \hspace{1cm} (14)

t$_{j,k}$ is the time when a $k$-th spike ($k = 0, 1, 2, \ldots$) happens in the neuron $j$ ($t_{j,k} < t < t_{j,k+1}$).

The time-average order parameter for the network is given by

$$\bar{R} = \frac{1}{t_{\text{fin}} - t_{\text{ini}}} \sum_{t_{\text{ini}}}^{t_{\text{fin}}} R_T(t),$$  \hspace{1cm} (15)

in which $t_{\text{fin}} - t_{\text{ini}}$ is the time window set to measure the phases, where $t_{\text{ini}}$ and $t_{\text{fin}}$ correspond to the initial and final time of the analyses, respectively. In our simulations, we consider $t_{\text{ini}} = 80$ s and $t_{\text{fin}} = 100$ s. The magnitude of the time-average order parameter tends to the unity when the network has a globally synchronised behaviour. For uncorrelated spiking phases, the order parameter is close to 0.

The traditional Kuramoto order parameter for each subnetwork, $s = 1, 2, 3, 4$, is described as

$$R_{(s)}(t) = \left| \frac{1}{N_{\text{sub}}} \sum_{j=(s-1)N_{\text{sub}}+1}^{sN_{\text{sub}}} e^{i\phi_j(t)} \right|.$$  \hspace{1cm} (16)

In order to quantify and distinguish the different symmetric synchronisation patterns, we calculate the so-called $m$-th moment of the order parameter $R^m$ ($m$ is an index), that is a variation of Eq. \hspace{1cm} (13) with $m = 1, 2, \ldots, S$ \hspace{1cm} (17)

$$R^m = \left| \frac{1}{N} \sum_{j=1}^{N} e^{im\phi_j(t)} \right|.$$  \hspace{1cm} (17)

This measure allows us to quantify the number of synchronised neuronal groups and consequently the symmetry of their phase distributions. For the particular case where $m=1$, Eq. \hspace{1cm} (17) is the same as Eq. \hspace{1cm} (13). For the network composed of $S = 4$ subnetworks, we calculate all the moments $m \in [1, 4]$. The highest moment of order parameter with the gives us information about the synchronisation and type of symmetry configuration. For instance, if $R^1 (m = 1)$ has the highest value (close to 1), the subnetworks have neurons forming effectively a single large
Figure 2: Raster plots of all neurons $i$ for fixed internal ($\tau_{\text{int}}$) and external ($\tau_{\text{ext}}$) time delays when synaptic plasticity is active. Different colours denote each subnetwork. The left, center, and right columns display the raster plots for the internal time delay equal to $\tau_{\text{int}} = 0$ ms, $\tau_{\text{int}} = 3$ ms, and $\tau_{\text{int}} = 6$ ms, respectively. The first, second, third, and last rows display the raster plots for external time delay equal to $\tau_{\text{ext}} = 0$ ms, $\tau_{\text{ext}} = 4$ ms, $\tau_{\text{ext}} = 6$ ms and $\tau_{\text{ext}} = 10$ ms, respectively. For small internal delays, high synchronous patterns are observed in each subnetwork, while bigger internal ones generate less synchronised patterns.

network (approximately 0 phase difference between subnetworks). If $R^2 (m=2)$ is the highest value, the neurons in subnetworks are synchronised in 2 groups in an anti-phase pattern (phase difference of $\pi$). The same idea applies for $m = 3$ and $m = 4$, where there are 3 and 4 groups, and the neurons in the groups have $2\pi/3$ and $\pi/2$ phase differences, respectively. By this measure, the location of each neuron of these $m$ groups in each subnetwork cannot be solely specified by the $m$-th order parameter measure. However, for any given pair of subnetworks, if neurons in a subnetwork have an approximate phase difference of $2\pi/m$ to the other neurons in the other subnetwork and there will be a number $m$ of pairs of subnetworks were this happens, then the highest order parameter will be the one with order $m$. Table 1 exhibits the standard range of parameters that we consider in our simulations.

3 Results and Discussions

In this work, we consider internal ($\tau_{\text{int}}$) and external ($\tau_{\text{ext}}$) delays in the subnetworks with and without the presence of STDP. Without plasticity, for small $\tau_{\text{int}}$ and varying $\tau_{\text{ext}}$, we observe different patterns of synchronisation between the subnetworks. However, our main goal is to investigate how these patterns of synchronisation affect the weights of network connections when plasticity is active. To do this, we consider different parameters and dynamics of the network.
Table 1: Descriptions of the standard parameters and range values considered in our simulations.

| Description                              | Parameter | Value       |
|------------------------------------------|-----------|-------------|
| Number of subnetworks                    | $S$       | 4           |
| Neurons per subnetwork                   | $N_{\text{sub}}$ | 100       |
| Internal connect. prob.                  | $p_{\text{int}}$ | 1.0         |
| External connect. prob.                  | $p_{\text{ext}}$ | 0.05        |
| Internal time delay                      | $\tau_{\text{int}}$ | [0, 6] ms  |
| External time delay                      | $\tau_{\text{ext}}$ | [0, 12] ms |
| Exc. synaptic conductance                | $g_{\text{ext}}$ | [0, 0.01] mS/cm² |
| Membrane capacity                        | $C$       | 1.0 μF/cm²  |
| Potassium conductance                    | $g_{\text{K}}$ | 36 mS/cm²   |
| Sodium conductance                       | $g_{\text{Na}}$ | 120 mS/cm²  |
| Leak conductance                         | $g_{l}$    | 0.3 mS/cm²  |
| Potassium rev. potential                 | $V_{\text{K}}$ | -77 mV      |
| Sodium reversal potential                | $V_{\text{Na}}$ | 50 mV      |
| Leak reversal potential                  | $V_{l}$    | -54.4 mV    |
| Excitatory reversal potential            | $V_{+}$    | 20 mV       |
| Constant current                         | $I_{i}$    | [10, 11] μA/cm² |
| Change rate of synap. weight             | $G$       | $10^{-5}$ mS/cm² |
| Time step integration                    | $\delta t$ | $10^{-2}$ ms |
| Initial time for analyses                | $t_{\text{ini}}$ | 80 s |
| Final time for analyses                  | $t_{\text{fin}}$ | 100 s |
| Internal time delay                      | $d_{\text{int}}$ | [0.1] ms |
| External time delay                      | $d_{\text{ext}}$ | [0, 12] ms |
| Time step integration                    | $\delta t$ | $10^{-2}$ ms |

Figure 2 shows the raster plots for fixed internal and external time delays ($\tau_{\text{int}}$ and $\tau_{\text{ext}}$) when the synaptic plasticity is on. In the left side, we consider $\tau_{\text{int}} = 0$ ms, (a) $\tau_{\text{ext}} = 0$ ms, (b) $\tau_{\text{ext}} = 4$ ms, (c) $\tau_{\text{ext}} = 6$ ms, and (d) $\tau_{\text{ext}} = 10$ ms. For small internal time delays, we verify synchronised symmetric patterns. In the center column, we consider $\tau_{\text{int}} = 3$ ms, (e) $\tau_{\text{ext}} = 0$ ms, (f) $\tau_{\text{ext}} = 4$ ms, (g) $\tau_{\text{ext}} = 6$ ms, and (h) $\tau_{\text{ext}} = 10$ ms. For these parameters, we observe no firing coherence, but the fastest neurons (higher $I_{i}$) in each subnetwork start firing and subsequently the slower neuron. In the right side, we use $\tau_{\text{int}} = 6$ ms, (i) $\tau_{\text{ext}} = 0$ ms, (j) $\tau_{\text{ext}} = 4$ ms, (k) $\tau_{\text{ext}} = 6$ ms, and (l) $\tau_{\text{ext}} = 10$ ms. Although some synchronisation can be noticed, it is lower than in the case for $\tau_{\text{int}} = 0$ ms. For $\tau_{\text{int}} = 0$ ms, we identify an equal pattern for the case with and without plasticity, as shown in Figure 2(a-d).

We focus on the most synchronised symmetric patterns. In Figure 2(a), neuron spikes in a single group (almost complete phase synchronisation) without delay between the subnetworks. Highest order parameter is the one with order $m=1$, indicating all neurons spiking nearly synchronously. Figure 2(b) displays neurons spiking in two groups for an external time delay equal to $d_{\text{ext}} = 4$ ms. Highest order parameter is the one with order $m=2$. In Figure 2(c), the neurons spike in four groups for an external time delay equal to $d_{\text{ext}} = 6$ ms. Highest order parameter is the one with order $m=4$. For a delay close to the average period between spikes ($\approx 14$ ms) the network returns to a single group, as shown in Figure 2(d). In this case, the neurons of all subnetworks exhibit a strong phase synchronisation. These patterns can also be obtained for different parameters when there is no plasticity.

Figure 3(a) displays the initial matrix connections $g_{ij}$ of the subnetworks. In Figure 3(b), we plot the initial neuronal phases where each colour represents a subnetwork. The coloured arrows are the initial order parameters showing that initially the neurons are not synchronised. Figure 3(c) exhibits the final coupling matrix after the plasticity actuates by 100 s, considering different values of the external delays and fixed internal delay $\tau_{\text{int}} = 0$ ms. For $\tau_{\text{ext}} = 0$ ms, we see a strong coherent dynamics among the subnetworks, all organised in a single group. The resulting network present some hierarchical organisation where the directed connections between some subnetworks were reinforced as highlighted by the dashed blue squares in Figure 3(c) (first column). For $\tau_{\text{ext}} = 4$ ms, the network forms effectively two pairs of subnetworks with neurons belonging to different subnetworks having a constant phase difference around $\pi$ radians (anti-phase synchronisation). In this case, connections between in-phase subnetworks are potentiated and depressed for the anti-phase ones. For $\tau_{\text{ext}} = 6$ ms, the network presents effectively four groups where neurons within each pair of groups have a phase difference around $\pi/2$ radians and the network is set to a
Figure 3: The panel (a) displays the initial matrix configuration for all simulations, where the red colour represents the initial intensity of non-null synaptic connections. Black indicates no synapses and yellow approximates to the maximal synaptic conductance $g_{ij}^{\text{max}}=0.01$ mS/cm$^2$. The panel (b) shows the initial phases of all neurons in the network. The top panel in (c) exhibits the resultant matrix for fixed internal and external time delays. In bottom in panel (c), the traditional Kuramoto order parameter for the entire network is represented by a black arrow $(R_T(t))$, as well as the same order parameter considering each subnetwork is denoted by the red, violet, cyan, and green arrows $(R_{(S)})$.
Figure 4: Resultant mean synaptic weight (left) and schematic representation of resultant connections between the subnetworks (right) for $\tau_{\text{int}} = 0$ ms. The edges in the graphs plotted on the right column panel represent the most significant average in each row of the matrices in the left column. We consider (a) $\tau_{\text{ext}} = 0$ ms, (b) $\tau_{\text{ext}} = 4$ ms, (c) $\tau_{\text{ext}} = 6$ ms and (d) $\tau_{\text{ext}} = 10$ ms.
moments of the order parameter is a suitable diagnostic of symmetry between the synchronised subnetworks.

**Figure 5:** The panels (a), (b), and (c) show the time evolution of the order parameter moments. The panels (d) and (e) display the symmetric patterns found in the parameter space of $\tau_{\text{int}}$ and $\tau_{\text{ext}}$ without (center-top) and with plasticity (center-down). The panels (f) and (g) show the highest value of the order parameters moments in the space parameter of $\tau_{\text{int}}$ and $\tau_{\text{ext}}$ for the case without (right-top) and with (right-down) synaptic plasticity. We compute the time evolution of the order parameter moments for $\tau_{\text{int}} = 0$ ms, (a) $\tau_{\text{ext}} = 0$ ms, (b) $\tau_{\text{ext}} = 4$ ms, and (c) $\tau_{\text{ext}} = 6$ ms.

The left side of Figure 4 shows the mean synaptic coupling between the subnetworks computed with the matrices from Figure 3(c). The schematic representations of the stronger mean connection between the subnetworks are displayed on the right side. The strongest weight connections between the subnetworks can be associated to the average matrix, as highlighted by the blue dashed squares. The black line with a triangle in the end corresponds to the connection direction from a presynaptic subnetwork to a postsynaptic one. In Figure 4(a), the strong phase synchronisation potentiate connections between the subnetworks in an asymmetric way. In this case, the neurons in the subnetwork 1 spike first, followed by subnetworks 2, 4, and 3. STDP is responsible for reshaping the network leading to the configurations depicted in Figure 4(a), where the connection reinforcement follows the order of spikes. All connections from subnetwork 1 to 2, 3, and 4 are reinforced. The same happens with the connections from subnetwork 2 to 3 and 4, and from subnetwork 4 to 3. Figure 4(b) exhibits two groups in anti-phase. The connections are reinforced between in-phase subnetworks and weakened between the two groups. In Figure 4(c), the subnetworks show a phase-lock synchronisation with average phase difference of $\pi$ radians. These patterns lead to a ring network configuration. The connections are potentiated in a cyclic way. The subnetworks are in-phase synchronisation in Figure 4(d) in a more coherent state than in Figure 4(a). This strong synchronisation promotes an all-to-all connection organization among subnetworks with an average reinforcement of the connections.

To better understand how plasticity promotes synchronised patterns, in Figure 5 we calculate the $m$-th moments for $\tau_{\text{int}} = 0$ ms, (a) $\tau_{\text{ext}} = 0$ ms, (b) $\tau_{\text{ext}} = 4$ ms and (c) $\tau_{\text{ext}} = 6$ ms. We observe a higher 1-st moment in Figure 5(a), which corresponds to a one-group phase synchronisation between the subnetworks. In this case, we verify that other moments are relatively lower than the first one. Figure 5(b) displays a higher 2-nd moment due to an anti-
phase synchronization in two major groups. In Figure 5(c), we identify a higher 4-th moment, which is associated with a phase-lock synchronisation between the subnetworks, as shown in Figure 3(c). The higher moment of the order parameter indicates the symmetry of the synchronised patterns. Figures 5(d) and (e) exhibit the symmetric synchronised patterns in the parameter space $\tau_{\text{ext}} \times \tau_{\text{int}}$ without and with synaptic plasticity, respectively. In the case without plasticity and small external time delay, we find one group symmetry with phase synchronisation. Increasing the external delays, we identify predominantly 2 and 4 groups symmetry, respectively. For larger external delay (around 10 ms), the network returns to a one synchronised group. These cases are found for small internal time delays (less than 1 ms). For higher internal time delays (more than 1 ms), we observe less synchronised patterns. On another hand, synaptic plasticity shifts to the left in $\tau_{\text{ext}}$, in which the regions where one, two, and four group symmetries are found. Moreover, the area in parameter space covering the existence of order 2 and 4 phase patterns is enlarged by plasticity. So, not only plasticity allows for more complex patterns to emerge for smaller $\tau_{\text{ext}}$, but also the livelihood of its appearance for a large range of delays. Thus plasticity promotes complexity as measured by the emergence of symmetric synchronous patterns. Figures 5(f) and 5(g) show the higher values of the order parameters moments for the case without and with synaptic plasticity, respectively. We verify that the highest values of $R_m$ correspond to one group symmetry.

To summarize, spiking synchronous patterns are strongly related to the network connection between subnetworks in a plastic neuronal network with time delay. For small external delays, one group configuration exhibits subnetworks spiking in a specific order, this order promotes synaptic potentiation from the subnetworks spiking first to the subsequent ones. Increasing the external delay, two groups pattern generate potentiation in in-phase subnetworks, while a synaptic depression among the ones from distinct groups. For major delays, four groups (a phase-lock synchronisation between subnetworks) show synchronised state, in which all subnetworks spiking almost at the same time and without any preferential order. This dynamical behaviour promotes a final global network configuration with an average increase of all the synaptic strengths. In all cases, the potentiation between the neuronal areas can also depend on the internal time delay. The less recurrent pattern found in our simulations is the 3 groups organisation, which can be associated with a transient behaviour.

4 Conclusions

In this work, we consider a network of subnetworks to study the effects of internal and external transmission delays on the generation of symmetric dynamics patterns, as well as the potentiation and depression on the synaptic weights due to the presence of plasticity. To do that, we consider Hodgkin-Huxley neurons coupled by means of excitatory chemical synapses and a time dependent plastic rule. To achieve synchronised patterns, the internal delayed transmission of neurons communication in the subnetworks assumes small values while the external one assumes higher values. When the internal transmission delay of neurons in the subnetworks assumes higher values, non synchronised patterns are observed.

Without plasticity and depending on the delay transmission between the subnetworks, we verify that synchronisation among subnetworks can be observed in different patterns. These regimes can be detected by means of the $m$-th moment of the order parameter which provides the information about how the dynamics of neurons in the subnetworks are correlated in the phase space of spiking times. Due to the plasticity effect, we verify that the final connectivity configuration between subnetworks is strongly reflected on the symmetric synchronised patterns. We show that the synaptic transmission delays play an important role in the generation of symmetric synchronised patterns. In addition, we also show that the phase, anti-phase, and symmetric phase-lock synchronised firing patterns influence the synaptic changes of the weight connections among the subnetworks. These results help us to understand the relation between the synchronized firings patterns, delays and connection structure.

All these paving the way for us to conclude that plasticity - described by a pairwise function that regulates synapse strength by the time intervals between two spiking neurons - in fact promotes the creation of evolved network structures whose subnetworks of intra connected neurons and their inter connections is strongly reflected in the global synchronization patterns measured by the phase dynamics of the neurons. The plastic neural network has a strong match between phase activity and graph structure.

Conflict of Interest Statement

The authors declare that there is no conflict of interest.
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