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Dynamic control for synchronization of separated cortical areas through thalamic relay

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Abstract

Binding of features and information which are processed at different cortical areas is generally supposed to be achieved by synchrony despite the non-negligible delays between the cortical areas. In this work we study the dynamics and synchronization properties of a simplified model of the thalamocortical circuit where different cortical areas are interconnected with a certain delay, that is longer than the internal time scale of the neurons. Using this simple model we find that the thalamus could serve as a central subcortical area that is able to generate zero-lag synchrony between distant cortical areas by means of dynamical relaying (Vicente et al., 2008). Our results show that the model circuit is able to generate fast oscillations in frequency ranges like beta and gamma bands triggered by an external input to the thalamus formed by independent Poisson trains. We propose a control mechanism to turn “On” and “Off” the synchronization between cortical areas as a function of the relative rate of the external input fed into dorsal and ventral thalamic neuronal populations. The current results emphasize the hypothesis that the thalamus could control the dynamics of the thalamocortical functional networks enabling two separated cortical areas to be either synchronized (at zero-lag) or unsynchronized. This control may happen at a fast time scale, in agreement with experimental data, and without any need of plasticity or adaptation mechanisms which typically require longer time scales.

Key words: dynamic relaying, thalamocortical circuit, zero-lag synchronization, correlation, firing pattern, thalamus, reticular thalamic nucleus.

1 *Introduction*

2 In the central nervous system (CNS) it is assumed that the information is
3 mainly represented by the activity of neurons transmitted to other neurons
4 through synaptic links. The extent of the neural network activated by a spe-
5 cific “piece of information” is a never ending matter of investigation but it
6 is accepted that both average levels of discharges, firing rate (Gollo et al.,
7 2009), and precise spike timing contribute to neural coding. Spatiotemporal
8 firing patterns (Villa et al., 1999b; Hayon et al., 2005) and coherent oscilla-
9 tory neural activity (Fries et al., 2007) associated to sensory and behavioral
10 events support the hypothesis that temporal information plays a key role in
11 brain processing. Empirical phenomena and extensive experimental data val-
12 idated across different species (Gray et al., 1989; Engel et al., 1991; Castelo-
13 Branco et al., 2000; Tiesinga et al., 2008) emphasize the importance of emerg-
14 ing cortico-cortical synchrony as a major phenomenon for binding features
15 distributed neural activity (von der Marlsburg, 1973; Fries, 2005; Desbordes
16 et al., 2008). Despite the success of physical models to reproduce oscillatory
17 patterns of neural activity it is not clear whether the synchronization is the
18 result of network processing exclusively limited to cortico-cortical interactions
19 or subcortical structures might also intervene (Contreras et al., 1996; Traub
20 et al., 1996; Vicente et al., 2008; Chawla et al., 2001), for a recent review
21 please refer to Uhlhaas et al. (2009).

22 The thalamus is a structure of CNS that could play an important role to let
23 the emergence or to control cortico-cortical synchronization because the ex-
24 change of information between the thalamus and cerebral cortex is a general
25 feature of all ascending sensory pathways but olfaction (Jones, 1985; Sher-
26 man, 2005). The connectivity pattern between thalamus and cortex is usually
27 viewed as been characterized by thalamocortical integration and corticotha-
28 lamic feedback (Steriade and Llinas, 1988; Villa et al., 1999a; Villa, 2002).
29 Multiple thalamocortical modules characterized by the same basic connectiv-
30 ity may be assumed to work in parallel and include three main components
31 (see Fig. 1): (i) dorsal thalamic neurons (e.g. from the medial geniculate body
32 for the auditory pathway or from the lateral geniculate body for the visual
33 pathway) recipient of the sensory input from the periphery; (ii) cells of the
34 thalamic reticular nucleus (R), a major component of the ventral thalamus;

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35 (iii) the cortical area receiving the corresponding thalamic input. The thala-
36 matic reticular nucleus receives collateral inputs from both thalamocortical and
37 corticothalamic fibres and sends its inhibitory projections to the dorsal tha-
38 lamus, thus regulating the firing mode of the thalamocortical neurons. The
39 thalamic reticular nucleus receives inputs also from several forebrain and mid-
40 brain areas known to exert modulatory functions (McCormick and Bal, 1994),
41 in particular from nerve growth factor responsive basal forebrain cholinergic
42 cells (Villa et al., 1996) that are involved in many cognitive functions and
43 whose dysfunction is associated to Alzheimer’s Disease. In the auditory sys-
44 tem evidence exist that corticofugal activity regulates the response properties
45 of thalamic cell assemblies by changing their bandwidth responsiveness to
46 pure tones (Villa et al., 1991) thus allowing to selectively extract informa-
47 tion from the incoming sensory signals according to the cortical activity (Villa
48 et al., 1999a). This model suggests that the thalamocortical circuit carries
49 embedded features that enable the build-up of combined supervised and un-
50 supervised information processing akin to produce an adaptive filter (Tetko
51 and Villa, 1997) aimed to select behaviorally relevant information processing
52 (von Kriegstein et al., 2008).

53 The current study is not aimed at simulating any detailed thalamocortical cir-
54 cuit, but rather to assess the role of simple variables that could play a major
55 role in controlling the emergence and maintenance of synchronized activity
56 in distributed cortical areas that project to the same thalamic nuclei. Our
57 model predicts that small changes in the cortical neurons firing rate, due to
58 non-correlated background synaptic activity in the thalamic region, is capable
59 of generating single or multi-frequency oscillations along with zero-lag syn-
60 chronization between distant cortical regions. We quantify this synchronized
61 state by measuring the signal-to-noise ratio which does not monotonically in-
62 crease with the firing rate. According to our model, thalamic activity plays
63 a key role in controlling the appearance of lag free synchronization between
64 cortical areas. In addition, despite its simplification, the model provides hints
65 about the conditions necessary to achieve that synchronization. We report an
66 efficient control set as the ratio of dorsal over ventral thalamus external input
67 activity to switch on thalamocortical synchronous dynamics. That switch oc-
68 curs at a fast time scale, without any need of synaptic plasticity which would
69 require longer time scales (Fries, 2005). The type of control that we suggest is
70 not limited to an “On”-“Off” switch, but it allows to control the appearance
71 of synchronous activity over an extended range of frequencies despite the de-
72 lays involved in the long-range cortico-cortical interactions (Ringo et al., 1994;
73 Vicente et al., 2009).

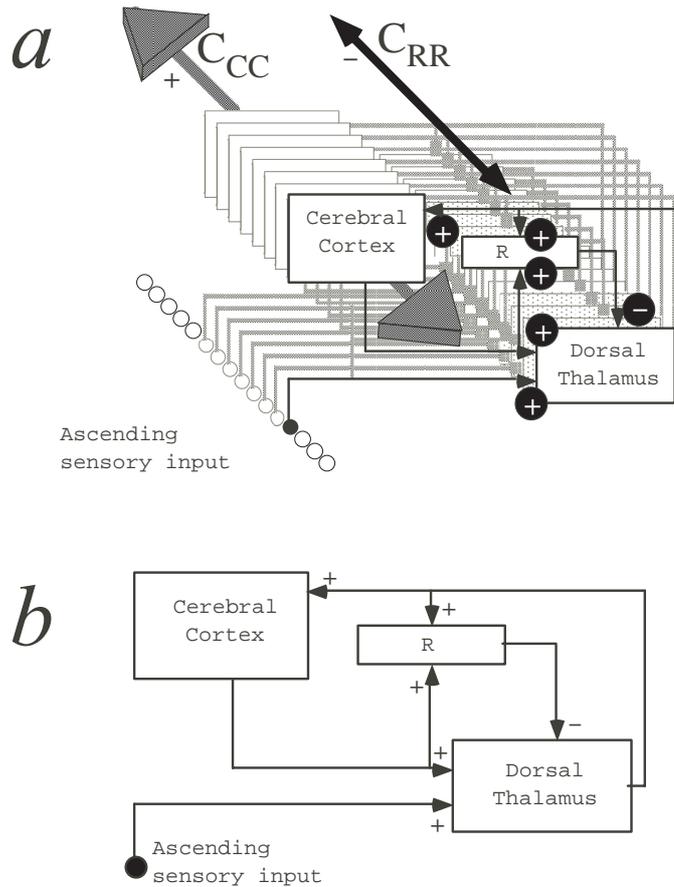


Fig. 1. **(a)** A functional scheme of the modular organisation of the typical thalamocortical sensory pathway (somatosensory, visual, sensory). The signs indicate the nature of the connections, (+) excitatory and (-) inhibitory. Notice the big arrows labeled C_{CC} corresponding to long-range excitatory cortico-cortical connections and C_{RR} corresponding to the inhibitory connections within the reticular and perigeniculate nucleus of the thalamus (R). Note the excitatory input from the ascending sensory pathway to the dorsal thalamus, the excitatory projection from the thalamus to the cortex with a collateral to R, and the excitatory projection of the cortex to the thalamus with a collateral to R. The only output of R is an inhibitory back-projection to the thalamus. **(b)** Explicit connections within one thalamocortical module.

74 Methods

75 To study the synchronization of cortical activity facilitated by the thalamic
 76 relay we conducted extensive numerical simulations of a reduced thalamocortical
 77 model of spiking integrate-and-fire neurons subject to background noise
 78 and an external driving. The model includes both local synapses and long-
 79 range interactions with different delays according to functional connectivity
 80 in a four populations motif (Milo et al., 2002) (Fig.2). The simulations were
 81 performed using NEST, the neuronal simulation tool (Brette et al., 2007) with

82 the PyNEST interface (Eppler et al., 2009).

83 **Neuronal model.** The integrate-and-fire neuron model (Brunel, 2000) for
 84 each neuron i satisfies the following dynamical equation for the membrane
 85 potential $V_i(t)$:

$$86 \quad \tau_{mem}(m) \frac{dV_i(t)}{dt} = -V_i(t) + RI_i(t), \quad (1)$$

87 where $\tau_{mem}(m)$ is the membrane time constant of neuron i belonging to the
 88 population m (as in Fig. 2); $I_i(t)$ is the total current arriving to the soma.
 89 The last term in the above equation is given by the sum of all postsynaptic
 90 potentials (PSP) of neurons belonging to the network plus the total postsy-
 91 naptic potentials of all external neurons, the latter being modeled as a Poisson
 92 process. Thus,

$$93 \quad RI_i(t) = \tau_{mem}(m) \sum_j J(j) \sum_k \delta(t - t_j^k - \tau(z, m)) + V_{ext}. \quad (2)$$

94 The first sum is taken over all presynaptic neurons j , each neuron receives
 95 $C_e(m, z)$ excitatory synapses and $C_i(m, z)$ inhibitory synapses and they de-
 96 pend on the inter-population (long-range) connections z if both neurons belong
 97 to different populations or otherwise on the population m to whom they be-
 98 long. t_j^k is the time of the k -th spike received by neuron i from its neighbor
 99 j . The axonal conduction delay is given by $\tau(z, m)$, which corresponds to a
 100 spike of a presynaptic neuron j that reaches neuron i . $J(j)$ stands for the
 101 PSP and depends on whether its presynaptic neighbor neuron j is excitatory
 102 ($J(j) = J_e$) or inhibitory ($J(j) = J_i$). V_{ext} is the postsynaptic potential gen-
 103 erated by neurons from outside the thalamocortical network. It is given by an
 104 independent and homogeneous Poisson process of N_{ext} external neurons, each
 105 one firing with a fixed average rate $\nu(m)$. The external spike contributes with
 106 a change of the membrane potential by J_{ext} whenever it impinges upon neu-
 107 ron i . The dynamics of the neurons can be described as following: the neurons
 108 start at a rest potential $V_r(m)$ which can be changed by the synaptic current.
 109 If the potential $V_i(t)$ of the i -th neuron reaches the threshold $\theta(m)$ a spike
 110 is generated and its membrane potential is reset to $V_r(m)$ after an absolute
 111 refractory period ($\tau_{rp} = 2$ ms).

112 After a brief parameter search and according to the range of values described
 113 in the literature we have set characteristic parameters for each population m
 114 presented in Table 1. The rationale of our choice was to preserve the simplicity
 115 of an oversimplified model of the thalamocortical circuit, though retaining the
 116 main dynamical features. The values of the threshold, the resting membrane

117 potential, and the membrane time constants were selected such that the neu-
118 rons in R were the most excitable and those in T were the least excitable
119 because T neurons are meant to receive the external input arising from the
120 ascending sensory pathways. For the sake of simplicity, the refractory period
121 and the excitatory/inhibitory postsynaptic efficacies were chosen to be the
122 same for all neurons.

Table 1

Neuronal parameters for the neurons in population m . *Each neuron receives also
afferences from a random neuron of the same population.

population (m)			Parameter	
C_1, C_2	R	T		
800	0	200	$N_e(m)$	# of excitatory neurons
200	40	0	$N_i(m)$	# of inhibitory neurons
20	25	15	$\tau_{mem}(m)$	membrane time constant (ms)
20.5	24.65	15	$\theta(m)$	threshold value (mV)
2	2	2	τ_{rp}	refractory period (ms)
10	12.5	7.5	$V_r(m)$	membrane rest potential (mV)
80	0	5	$C_e(m)$	# of excitatory synapses*
20	10	0	$C_i(m)$	# of inhibitory synapses*
1.5	2	1	$\tau(m)$	synaptic delay (ms)
0.05	0	0.05	J_e	excitatory postsynaptic efficacy (mV)
-0.2	-0.2	0	J_i	inhibitory postsynaptic efficacy (mV)

123 **Thalamocortical model.** The topology of the model is characterized by
124 two thalamic and two cortical neural populations (Shepherd, 1998; Huguenard
125 and McCormick, 2007). The overall layout of our model is depicted in Fig. 2.
126 The thalamus is composed by two separate populations, one of excitatory
127 thalamocortical principal cells (T) and another of inhibitory neurons corre-
128 sponding to the thalamic reticular and perigeniculate nuclei (R). The two
129 thalamic populations are also characterized by recurrent intrathalamic con-
130 nections. The cortical populations are formed by an excitatory cell type with
131 local, long range cortical, and feedback corticothalamic projections and by an
132 inhibitory type characterized by only local efferent projections. In addition,
133 the two cortical populations are distributed in two “areas” (C_1 and C_2) which
134 may or may not be interconnected (following the value of parameter C_{CC}).
135 It is a hierarchical network, with both an intra-population random structure
136 and a simple inter-population pattern of connectivity with longer delays. The
137 populations have both internal and external connectivity. Then, the topology

138 satisfies the following constrains: both R (C_{CR}) and T (C_{CT}) populations re-
139 ceive cortical feedback, the cortical populations are innervated by T (C_{TC})
140 but do not receive inhibitory feedback from R. There are also direct connec-
141 tions from R to T (C_{RT}) and from T to R (C_{TR}). Long range cortico-cortical
142 connections are determined by C_{CC} . Assuming that the thalamus is composed
143 by both R and T populations, the thalamocortical model may also be reduced
144 to a three populations network formed by a central thalamic region (T and R)
145 and two balanced cortical areas. Each neuron of a given population receives
146 the same amount of postsynaptic connections. The presynaptic neurons are
147 set randomly, therefore, the postsynaptic distribution is binomial for each type
148 of neuron (excitatory or inhibitory) within a given population.

Table 2

Parameters for inter-population (long-range) connections z between any two regions. Each neuron of the target population receives input from a randomly selected neuron belonging to the efferent population.

inter-population connectivity (z)						Parameter	
CR	CT	TC	RT	TR	CC		
30	20	20	0	80	0-110	$C_e(z)$	# of excitatory synapses*
0	0	0	25	0	0	$C_i(z)$	# of inhibitory synapses*
8	8	5	2	2	5	$\tau(z)$	synaptic delay (ms)

149 The connectivity parameter values described in Table 2 were set arbitrarily in
150 order to maintain the relative proportion of cell types usually described in the
151 literature (Jones, 1985; Sherman, 2005). The number of connections were set to
152 keep 160 afferences to each neuron of C, 75 afferences to each neuron of T and
153 150 afferences to each neuron of R. This pattern of convergence-divergence is
154 meant to preserve the known anatomical thalamocortical and corticothalamic
155 pattern of connectivity (Jones, 1985; Sherman, 2005). The specific proportion
156 of afferences generated by each population is indicated in the boxes at the
157 bottom of Fig. 2. The delays were set to account for typical axonal delays de-
158 scribed in the thalamus and cortex of mammals (Swadlow, 2000; Knoblauch
159 and Sommer, 2004). Despite the fact that we have not systematically inves-
160 tigated all ranges of axonal delays, we observed that the results are robust
161 against these delays. The most critical parameter is the delay between the
162 thalamus and the cortical areas (τ_{TC}) which must be kept identical for all
163 ascending projections. If this delay is not the same for all TC connections
164 the maximum number of coincident spikes in the cross-correlograms does not
165 occur at zero-lag but at a lag that depends on the difference between the TC
166 time delays. It is worth mentioning that a constant latency between thala-
167 mus and cortex irrespective of the distances has been reported due to regional
168 myelination differences that compensate for the conduction velocities (Salami
169 et al., 2003).

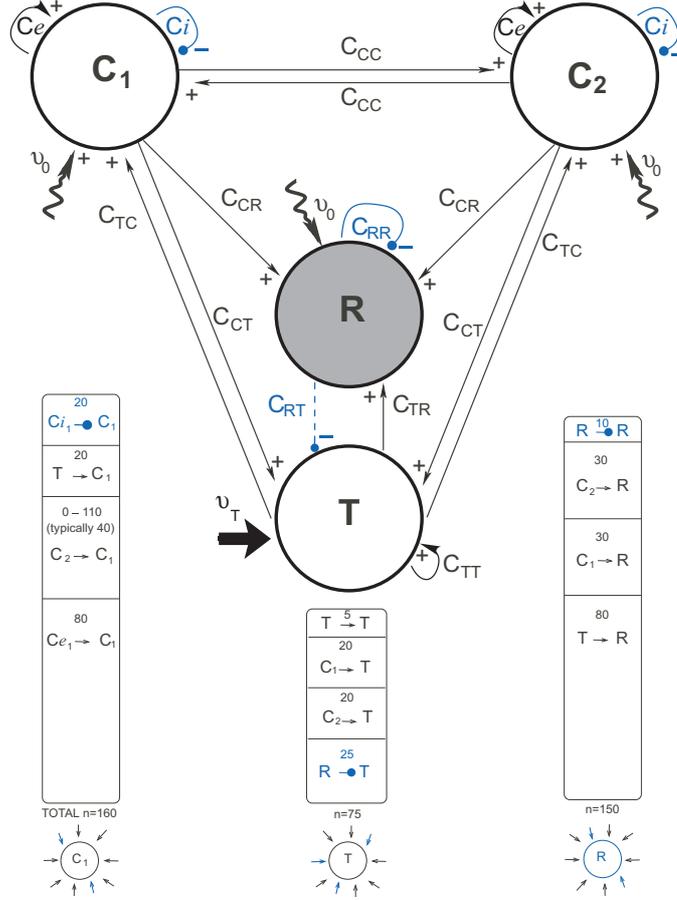


Fig. 2. Circuit layout. The sign at the arrow tip indicate the effect of the connection either excitatory (+) or inhibitory (-). Notice that the inhibitory projections are represented with a rounded shape tip. The boxes at the bottom of the figure show the pattern of the afferences of a cortical area (C_1 , bottom left), of principal thalamic neurons (T, bottom centre), and of thalamic reticular neurons (R, bottom right). The thalamus is formed by two neuronal populations, the excitatory thalamocortical projecting neurons (T) and the inhibitory reticular and perigeniculate neurons (R) which are reciprocally interconnected (C_{TR}, C_{RT}). In addition, there are local excitatory connections (C_{TT}) between thalamic principal cells and local inhibitory connections (C_{RR}) between reticular thalamic cells. Two cortical “areas” (C_1 and C_2) are connected to the same thalamic region. Each cortical area includes both excitatory (80%) and inhibitory (20%) neurons. The cortical excitatory neurons send feedback projections to the thalamus (C_{CT}, C_{CR}), and establish long range corticocortical projections (C_{CC}) and local connections (C_e). The cortical inhibitory neurons establish only local connections (C_i). The inter-population connectivity is described by the parameters of Table 2. The background activity at rate ν_0 and the external input at rate ν_T consist of independent Poisson trains with parameters of Table 3. Neurons in T are the only ones receiving an external input meanwhile all other neurons receive background activity. The external input is uncorrelated and defines the key parameter: $\frac{\nu_T}{\nu_0}$.

Table 3

Parameters of the background and external afferences.

0.1 mV	J_{ext}	external synaptic efficacy
10.0 Hz	ν_0	external driving Poisson mean rate to C and R
8.0-45.0 Hz	ν_T	overall external driving Poisson process to T
450	N_{ext}	number of external afferences

170 **Background activity and external input.** To model the background
 171 activity we assume that each neuron in the network is connected with N_{ext}
 172 excitatory external neurons subject to an independent random Poisson pro-
 173 cesses with average rate ν_0 for neurons of all regions. The thalamic region
 174 (T) receives the background activity combined with an external input also
 175 modeled by independent Poisson process, such that both the overall external
 176 input to T is a process characterized by rate ν_T . The parameters used for the
 177 Poisson background and the external driving are presented in Table 3.

178 **Cross-correlation analysis.** We run extensive simulations and analyze
 179 the spike trains over several trials. In order to quantify the results from the
 180 numerical simulations, we define two values from the cross-correlogram: a) its
 181 mean value representing the “noise” level quantifying the expected number of
 182 coincidences by chance; b) the peak of the cortico-cortical cross-correlogram
 183 (typically at zero-lag) that stands for the “signal”. Those quantities are used
 184 to compute the signal-to-noise ratio for different values of ν_T and different
 185 strengths of cortical interconnectivity (C_{CC}). The results are averaged over
 186 100 trials during 2, 000 ms in a stationary regime after 500 ms of transient dy-
 187 namics. The averaged result is condensed in a single cross-correlogram, which
 188 measures the mean number of coincidences (in a 2 ms bin) of 3, 000 randomly
 189 selected neuron pairs belonging to different populations and also averaged
 190 over the trials. This procedure allows us to assess the mean behavior of the
 191 dynamics and eliminate single trial fluctuations.

192 The “noise” is determined by the mean over the time lag in the averaged cross-
 193 correlogram. It can also be calculated analytically considering the activity of
 194 the two populations just as been independent: Let $F(p)$ be the mean firing
 195 rate of a population p and b the bin size of the computed cross-correlogram,
 196 therefore the mean cross-correlogram (noise) of two arbitrary populations i
 197 and j is given by $\langle XCOR_{i-j} \rangle = F(i)F(j)b$. For a typical thalamocortical
 198 circuit the two cortical areas have either maximum synchrony at zero-lag or
 199 no synchrony (unless C_{CC} is greater than the number of internal excitatory
 200 cortical connections C_{eC}). Thus the “signal” of the cortico-cortical dynamics
 201 is defined as the number of coincidences in the cross-correlogram at zero time
 202 lag.

203 Results

204 We have simulated the activity of large populations of interacting neurons
205 with delayed connections. We used a simple integrate and fire (I&F) neu-
206 ronal model in order to keep the problem easily computationally tractable.
207 The model retains threshold dynamics and if the membrane potential reaches
208 the threshold a spike is fired. The membrane potential is reset after the fir-
209 ing to its resting potential with an absolute refractory period (2 ms). The
210 spike is transmitted to all target neurons which receive an excitatory or in-
211 hibitory postsynaptic potential according to the type of synapse. The spike
212 is transmitted with a delay depending on the connection type. Large delays
213 are associated with inter-population connections and short delays with local
214 connections within each population. The results analyze the firing rate, cross-
215 correlation indicators, oscillation and synchronization information calculated
216 from the spike trains of individual neurons and neuron populations. It is worth
217 mentioning that the neuronal spike times were reliably reproduced despite the
218 simplicity of the I&F model.

219 **Thalamocortical circuit dynamics.** In the most symmetrical case, the
220 T region is set in order to receive external driving with the same rate as the
221 other populations ($\nu_T = \nu_0$). The firing rate in R is higher than in the cor-
222 tex which is also higher than in T. For a typical number of cortico-cortical
223 interaction, say $C_{CC} < 40$, due to the network connectivity and the difference
224 in the neuronal parameters, there is no correlation among the different areas,
225 and the activity is random and irregular. For $\nu_T > \nu_0$ other scenario takes
226 place. The raster plots of 150 neurons randomly chosen among all neuronal
227 populations illustrate the network dynamics. Such a typical raster plot is de-
228 picted in Fig. 3a. It shows the case in which the cortico-cortical connections
229 are set as $C_{CC} = 40$ and the thalamus is receiving an external input of mean
230 rate $\nu_T = 7/3\nu_0$. The neurons within the populations T and R are synchron-
231 ized at a high frequency. The two cortical areas exhibit a large number of
232 coincidences at zero-lag, meaning that they are synchronized and *in-phase*.
233 The cross-correlograms (see Methods section for details) between the cortical
234 areas and between the thalamus and one cortical area are shown in Fig. 3b,c.
235 The graphic clearly indicates *in-phase* correlation among cortical areas while
236 the thalamus and the cortical area are out of phase (with the cortical area
237 delayed by 6 ms).

238 The synchronization of the cortical regions depends on the external input to
239 T. Fig. 4 shows the raster plot of a single trial characterized at $t = 50\text{ ms}$
240 by a sudden increase of the T activity from the mean rate ν_0 to $7/3\nu_0$. The
241 synchrony does not occur in the system for low values of input ν_T , for instance
242 $\nu_T = \nu_0$, from 0 ms to 50 ms or after the input is switched off, say for time

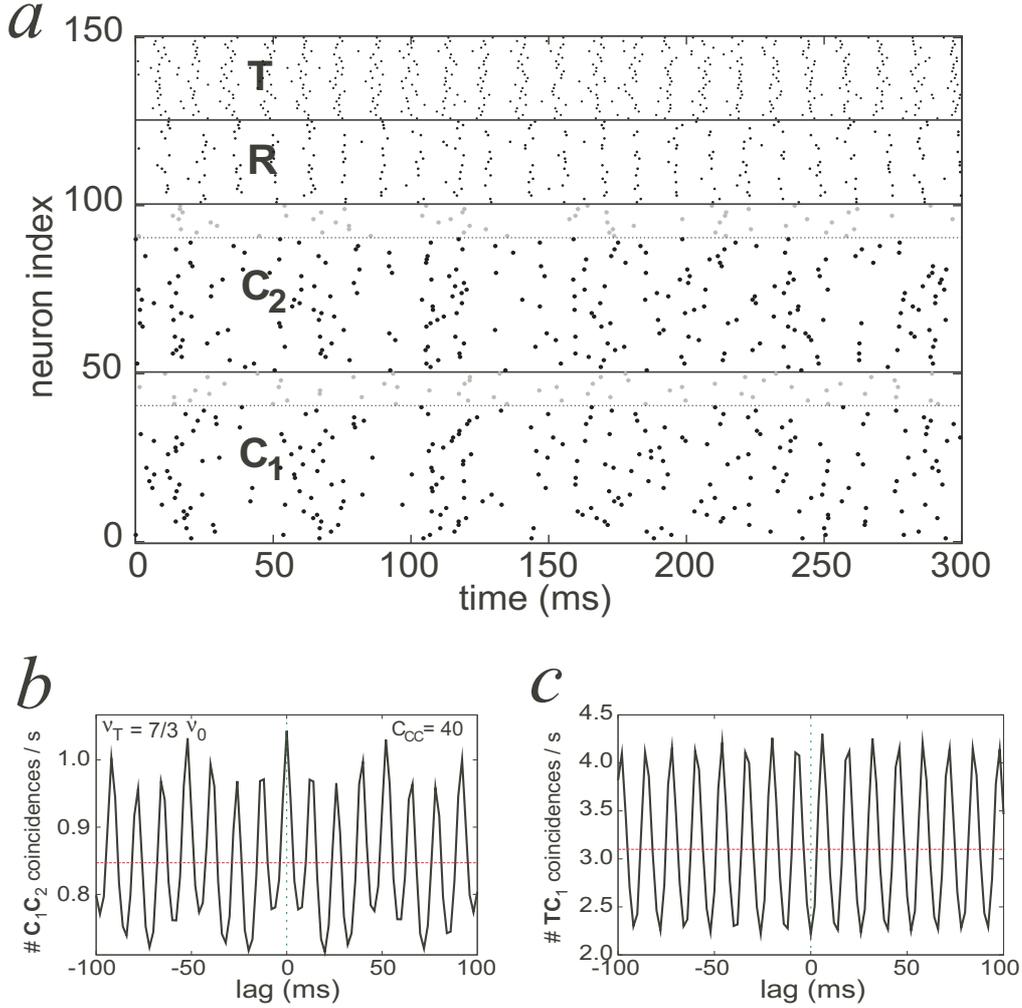


Fig. 3. Thalamocortical dynamics. **(a)** Raster plots of 150 neurons randomly chosen (50 from each cortical population and 25 neurons from R and T). The firing times of the local cortical inhibitory neurons are represented by grey dots. R, C₁, C₂ receive a background Poissonian noise at rate ν_0 Hz. T receives a Poissonian noise at rate $\nu_T = \frac{7}{3} \nu_0$. **(b)** Averaged cross-correlogram of 3,000 randomly selected neuronal pairs of different C₁ and C₂ populations averaged over 100 trials. Bin size 2 ms. The horizontal line corresponding to the mean value stands for the *noise*. The peak at zero-lag stands for the *signal*. These values are used to compute the *signal-to-noise* ratio (see text for details). **(c)** Averaged cross-correlogram of 3,000 randomly selected neuronal pairs of different T and C₁ populations averaged over 100 trials. Same labels as panel (b). Notice that the maximum of C₁-C₂ cross-correlation occurs exactly at zero-time lag while the maximum of T-C₁ occurs at a lag of 6 ms.

243 $t > 250$ ms.

244 The mean firing rate of T, C, and R neurons, computed over 2,000 ms, in-
 245 creases monotonically as a function input rate ν_T (Fig. 5a). The dependency

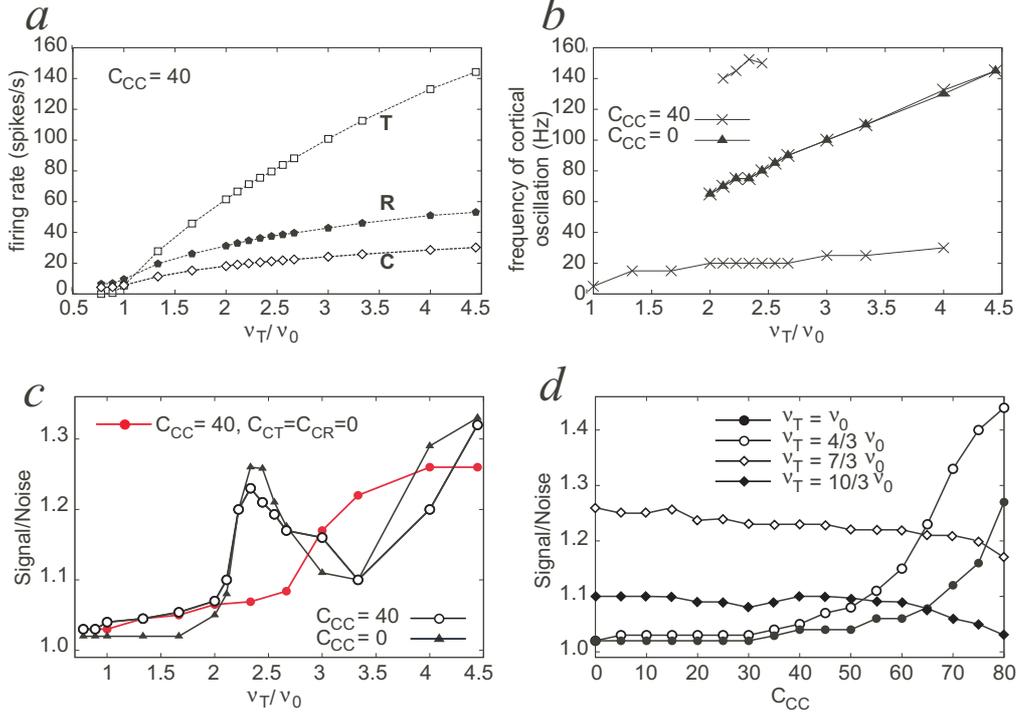


Fig. 5. Unveiling the dynamics - 100 trials analysis. **(a)** Diagram of the mean firing rate of T (open squares), R (bold dots) and C (open diamonds), as a function of the external input ν_T . **(b)** Diagram of the frequencies of cortical oscillations for increasing ν_T . The frequencies correspond to the peaks of the spectrum calculated from the Fourier transform of the cross-correlograms. Crosses indicate the data calculated with coupled cortical areas ($C_{CC} = 40$) and solid triangles were calculated in the absence of cortico-cortical coupling ($C_{CC} = 0$). **(c)** Signal-to-noise ratio as a function of the external input ν_T . Cortico-cortical coupling was set to zero (open dots) and $C_{CC} = 40$ (solid triangles). A special case with no corticofugal connectivity ($C_{CR} = C_{CT} = 0$) is plotted (solid dots) for comparison. **(d)** Signal-to-noise ratio as a function of the strength of cortico-cortical connectivity. We compare the curves for different values of ν_T .

246 of the cortical oscillation frequency as a function of ν_T/ν_0 is shown in Fig. 5b
 247 for directly interconnected ($C_{CC} = 40$) and disconnected ($C_{CC} = 0$) cortical
 248 areas. The frequencies are determined from the power spectrum analysis of the
 249 cross correlograms. Only those components whose power is larger than 20% of
 250 the maximum power are considered here. In the disconnected case, the cortical
 251 areas oscillate at a single frequency close to the thalamic firing rate (see rate in
 252 Fig. 5a). In the interconnected case ($C_{CC} = 40$) a single frequency dominates
 253 the oscillatory dynamics only if $\nu_T < 2\nu_0$. Beyond this threshold at least two
 254 frequencies of oscillation appear. For $\nu_T = \frac{7}{3}\nu_0$ three different frequencies are
 255 observed (as in Fig. 3b). The lowest frequency is related to the firing rate of
 256 the neurons within the cortical areas. The intermediate frequency is related
 257 to the thalamic firing rate like in the disconnected case. An increase of the
 258 oscillatory frequency in the cortical areas is due to greater interaction between

259 the cortex and the thalamus as a function of a larger input fed into the thala-
 260 mus. The highest frequency component in the interconnected case ($C_{CC} = 40$)
 261 is likely to be related to the inverse of the delay time of the cortico-cortical
 262 connection. However, this frequency component is observed only for a very
 263 small range of input values.

264 The signal-to-noise ratio, as defined in the Methods section from the cross-
 265 correlograms, as a function of ν_T/ν_0 is illustrated in Fig. 5c. The firing rate and
 266 the “signal” increase monotonically with the external rate of the input, but
 267 interestingly SNR is characterized by a local maximum for uncoupled cortical
 268 areas as well as for coupled cortico-cortical areas with connectivity $C_{CC} = 40$.
 269 The signal-to-noise was quite flat for low values of ν_T , then increases until
 270 reaching the local maximum. After decreasing from the local maximum the
 271 signal-to-noise increases again monotonically for very large values of the rate
 272 ν_T . To gain insight whether the synchronization among the cortical areas is
 273 induced by the T-R circuit into this aspect, we allowed the system to evolve
 274 with the whole connectivity and suddenly cut the cortico-thalamic connections
 275 ($C_{CR} = C_{CT} = 0$). The results are shown with solid dots in Fig. 5c. This curve
 276 shows that for $2 < \nu/\nu_0 < 3$ the SNR is much smaller than the one obtained
 277 with the whole connectivity, indicating that the synchronization is not driven
 278 by the thalamus circuit. Instead, a true collective behavior emerges from the
 279 whole interaction. For $\nu/\nu_0 \sim 3$ the curve increases suddenly, thus indicating
 280 that the synchronization starts to be driven by the activity of the thalamus.
 281 The signal-to-noise ratio as a function of the strength of the cortico-cortical
 282 connection for different values of ν_T/ν_0 is illustrated in Fig. 5d. Interestingly,
 283 for low values of ν_T/ν_0 the signal-to-noise response is flat but increases for
 284 large C_{CC} while it is flat but decreases for higher values of ν_T/ν_0 .

285 **Effect of the cortico-cortical connection.** The mean firing rate F of
 286 the three neuronal populations as a function of the strength C_{CC} at an input
 287 level $\nu_T = 7/3\nu_0$ is illustrated in Fig. 6a. This figure shows that the cortical
 288 firing rate is indeed the most affected rate and increases monotonically with
 289 an increase in the cortico-cortical connectivity. The dominant frequencies of
 290 cortical oscillations determined by the power spectrum analysis are displayed
 291 in Fig. 6b as a function of cortical connectivity and for two levels of external
 292 input to the thalamus. For a value $\nu_T = 5/3\nu_0$ a single frequency appears
 293 almost constant and independent of the C_{CC} strength. On the contrary, at
 294 $\nu_T = 7/3\nu_0$ three frequency components appear for $C_{CC} > 35$. Like in Fig 5b
 295 the lowest frequency is associated to the cortical firing rate and the interme-
 296 diate frequency is associated to the firing rate of population T. The highest
 297 frequency could also be associated to the inverse of the delay time in the
 298 cortico-cortical connection and became more important for higher values of
 299 C_{CC} . The presence of multiple oscillatory frequencies can be clearly observed
 300 in the cross-correlogram for $C_{CC} = 60$ and $\nu_T = 7/3\nu_0$ (Fig. 6c), whereas a

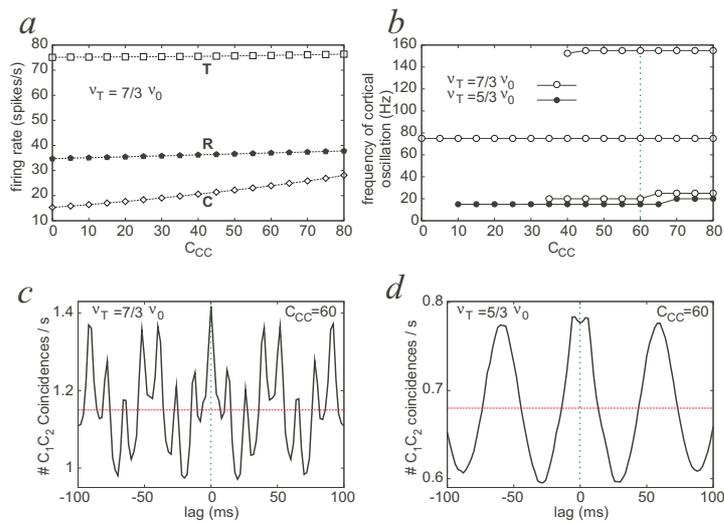


Fig. 6. Effect of the cortico-cortical connectivity. **(a)** The firing rate of T, R and C as a function of the cortico-cortical strength for $\nu_T = 7/3\nu_0$. **(b)** Frequencies of cortical oscillations for increasing values of C_{CC} for two different values of ν_T ($5/3\nu_0$; $7/3\nu_0$). **(c)** Cross-correlogram between C_1 and C_2 for $C_{CC} = 60$ and $\nu_T = 7/3\nu_0$. Notice the local maxima next to zero-lag are located at ± 12 ms. **(d)** Same as panel (c) but for $\nu_T = 5/3\nu_0$. Notice that the maxima are not exactly at zero-lag but at ± 6 ms.

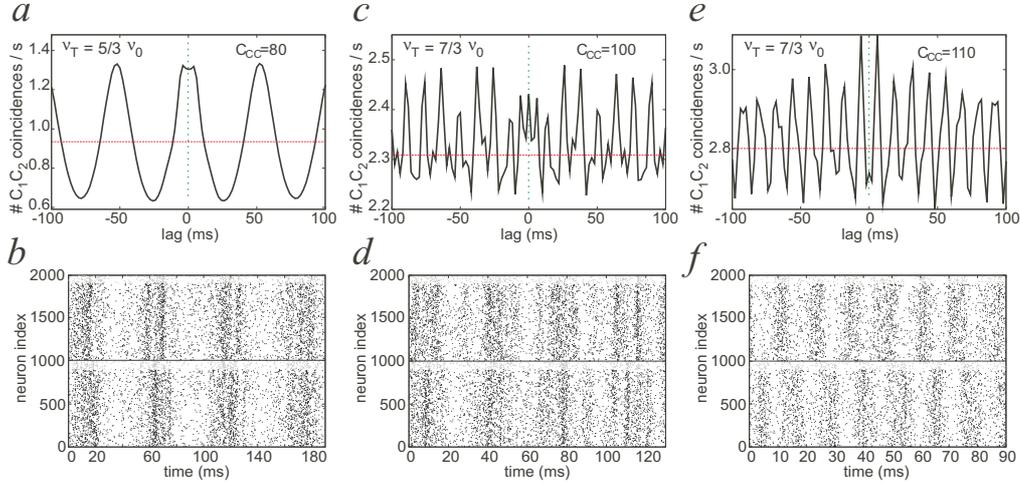


Fig. 7. Dynamics of the cortical area as a function of the cortico-cortical interaction strength. **(a,b)** The upper panel shows the cross-correlogram for $\nu_T = 5/3\nu_0$ and $c_{cc} = 80$. The lower panel shows the corresponding raster plot for all cortical neurons. C_1 neurons are indexed from 1 to 1000 and C_2 neurons are indexed from 1001 to 2000. **(c,d)** Same as previous for $\nu_T = 7/3\nu_0$ and $c_{cc} = 100$. **(e)** Same as previous for $\nu_T = 7/3\nu_0$ and $c_{cc} = 110$.

301 single frequency component dominates the dynamics for $\nu_T = 5/3\nu_0$ (Fig. 6d).

302 The observation of the raster plots and of the cross-correlograms illustrates
 303 further the dynamics emerging from the interaction between the cortical areas.
 304 In Fig. 7a, b it can be observed that for $C_{CC} = 60$ and $\nu_T = 5/3\nu_0$ the slow
 305 frequency component related to the cortical firing frequency is predominant.
 306 The peak is not sharp, at ± 4 ms from the zero-lag, and a “master-slave”
 307 dynamics can be observed in the region of high instantaneous firing rate (say
 308 from 50–80 ms after the external input onset). With parameters of $C_{CC} =$
 309 100 and $\nu_T = 7/3\nu_0$ multiple frequencies are observed in the raster plot and
 310 in the cross-correlogram (Fig. 7c,d). In this case, both the zero-lag cortical
 311 synchronization and the leader-ladder dynamics present a strong competition.
 312 At very large values $C_{CC} = 110$ the cortico-cortical connection dominates
 313 and gives rise to an out-of-phase cortical synchronized dynamics between the
 314 two areas (Fig. 7e,f) The signature of this dynamics appears both in a double
 315 peak at ± 6 ms (corresponding to the cortico-cortical coupling time in the cross
 316 correlation function) and in the raster plot where zero-phase synchronization
 317 does not occur between the cortical areas.

319 We have presented the dynamics of a simplified thalamocortical circuit. Our
 320 results suggest that the thalamus could be a central subcortical area that
 321 is able to trigger the emergence of zero-lag synchrony between distant corti-
 322 cal areas due to a dynamical relaying (Fischer et al., 2006; Vicente et al.,
 323 2008). According to this phenomenon a central element can enable two popu-
 324 lations to synchronize at zero-lag. Other subcortical areas such as the brain-
 325 stem (Scheller et al., 2009) and the hippocampus are likely to play a similar
 326 role in dynamical relaying. However, the peculiar recurrent connections of the
 327 thalamic reticular nucleus (Jones, 1985; Sherman, 2005) might provide the
 328 thalamocortical circuit with specific features that do not account just for the
 329 synchronized pattern, but also for switching “on” or “off” the asynchronous
 330 state. Furthermore, considering that large scale integration may occur as a
 331 consequence of neuronal coherence, the critical question about how the dy-
 332 namical selection of integrated areas is achieved remains open (Salinas and
 333 Sejnowski, 2001; Fries, 2005; Vicente et al., 2008; Uhlhaas et al., 2009). We
 334 suggest that an increase in the external activity fed into the T population with
 335 respect to that of R yields the cortical areas synchronize at zero-phase lag as
 336 depicted in Fig 3. That means the thalamus would be able to control the cor-
 337 tical synchronous state and regulate large scale integration. This control can
 338 occur at a fast time scale in agreement with experimental data and without
 339 any need of plasticity or adaptation mechanisms which typically require longer
 340 time scales. The main input sources to T are the ascending sensory input and
 341 the descending cortico-fugal pathway, thus suggesting that both inputs may
 342 play an important role in controlling cortical synchrony. This hypothesis for
 343 the cortico-petal projections is complementary to the hypothesis of “adaptive
 344 filtering” suggested elsewhere for the cortico-fugal projections (Villa et al.,
 345 1991, 1999a; Tetko and Villa, 1997).

346 According to our model, see Figs. 5b, 6b, the thalamocortical circuit is able
 347 to generate fast oscillations in frequency ranges like beta and gamma bands
 348 triggered by an external input to the thalamus formed by independent Poisson
 349 trains. The question of how to generate such fast oscillations has been largely
 350 discussed in the literature (Traub et al., 1996; Doiron et al., 2003; Doiron
 351 et al., 2004; Börgers et al., 2005; Marinazzo et al., 2007; Börgers et al., 2008)
 352 but, as recently pointed out (Nikolić, 2009), empirical phenomena like the
 353 cycle skipping were not satisfactorily described. The cycle skipping is observed
 354 experimentally in the current thalamocortical model when each cortical neuron
 355 spikes according to a gamma frequency modulation but with a smaller firing
 356 rate. In the raster plots of Fig. 3a it is possible to observe that few neurons
 357 spike at a given gamma cycle. Then, the oscillations are in fact shared by a
 358 whole population while single neurons skip cycles. As shown in Figs. 5a, b the
 359 cortical oscillations, for instance at a signal-to-noise ratio local maximum $\nu_T \simeq$

360 $\frac{7}{3} \nu_0$, occur at frequencies near 80 Hz for disconnected areas and in multiple
361 frequencies for $C_{CC} = 40$, while the average firing rate is approximately $\frac{1}{4}$
362 of it, 20 *spikes/s*. In general, the firing rate of the cortical populations (see
363 Figs. 5a, 6a) were found to be related to the lowest frequency component in
364 case of multiple frequency oscillations. Otherwise the cortical firing rate tends
365 to be much lower than the single frequency of oscillations (e.g., Fig. 5b) or
366 close to it for low external driving ($\nu_T = \frac{5}{3} \nu_0$), e.g., Fig. 6b.

367 The current results emphasize the hypothesis that the thalamus could control
368 the dynamics of the thalamocortical functional networks enabling two sepa-
369 rated cortical areas to be either synchronized (at zero-lag) or unsynchronized.
370 Correlations in the output firing rate of two neurons have been shown to in-
371 crease with the firing rate (de la Rocha et al., 2007). Indeed we observed that
372 for increasing input rates (ν_T) the firing rate of all populations increase mono-
373 tonically, accordingly to an expected sigmoidal function (Fig. 5a). König and
374 collaborators (König et al., 1995) reported physiological evidence of long-range
375 synchrony with oscillations, whereas short-range synchrony may occur with or
376 without oscillations. Our results, especially for low number of cortico-cortical
377 inter-population synapses (say smaller than the internal connectivity), are in
378 agreement with this finding. However, synchrony without oscillations in lo-
379 cal circuit may appear due to extensive sharing of common excitatory inputs
380 which typically generate the zero-lag coincidence observed when neurons are
381 fire at high rates (de la Rocha et al., 2007). Conversely, neurons correlated by
382 long-range connections are likely to share very few synaptic driving, such that
383 synchrony without oscillations should be very rare.

384 In order to suggest an insight of the model with the anatomical pattern of
385 the circuit one should consider that the thalamocortical and corticothalamic
386 projections are reciprocal to a great extent but corticothalamic projections are
387 characterized by a dual pattern of synapses on the thalamic neurons. Small
388 endings formed the major corticothalamic terminal field, whereas giant termi-
389 nals were less numerous and formed additional terminal fields together with
390 small terminals. (Rouiller and Welker, 2000; Takayanagi and Ojima, 2006).
391 The modal switch of corticothalamic giant synapses controlled by background
392 activity was recently reported (Groh et al., 2008). We speculate that this find-
393 ing and our results may suggest that each pattern of corticothalamic synapse
394 might correspond to a different function. One synaptic type might be involved
395 in assessing the circuitry necessary for the build-up of cortico-cortical synchro-
396 nization. The other synaptic type would be involved in transmitting stimulus-
397 related information. Which is which is a question that the current study is
398 unable to answer. We must also consider the fact that our model of individual
399 dynamics of the integrate-and-fire neurons does not produce burst discharges
400 (Sherman, 2001; Krahe and Gabbiani, 2004). This is a clear limitation and the
401 inclusion of a more physiologically realistic model as well as greater neuronal
402 diversity (Buia and Tiesinga, 2008) are scheduled for our future work. Despite

403 the simplification of our circuitry and the neuronal network modeling in gen-
404 eral the robustness of our model is an interesting outcome of this study. The
405 zero-lag synchrony between the cortical areas depends only on the identical
406 axonal delays $\tau(TC)$. If these delays are not the same for all TC connections
407 the maximum number of coincident spikes in the cross-correlograms does not
408 occur at zero-lag but at a lag that depends on the difference between the TC
409 time delays. However, it is worth mentioning that regional myelination that
410 compensates for changes in the conduction velocity has been reported as a
411 mechanism that could keep constant latency between thalamus and cortex
412 irrespective of the distances. Moreover, our results are in agreement with the
413 suggestion reported by Chawla et al. (2001) about the key role of the thalamus
414 favoring the zero-lag synchronization.

415 We have arbitrarily kept the external input ν_0 over R and the cortex popula-
416 tions fixed but we might have kept fixed T and the cortex populations with a
417 variable external input into R (ν_R). In fact it is the dependency on the variable
418 $\frac{\nu_T}{\nu_R}$ which represents the control key of the dynamic activity of the system as
419 both rates of external inputs (ν_T, ν_R) are varying over time (McAlonan et al.,
420 2008; Yu et al., 2009). The importance of uncorrelated inputs can be viewed
421 as emphasizing the role of so-called “background activity”, which was already
422 reported to play an important role in controlling the thalamocortical circuit
423 dynamic state (Wolfart et al., 2005). We are convinced that further simula-
424 tions with more accurate details of the neuronal models and with embedded
425 models of the dual cortico-fugal connectivity may provide critical clues for
426 better understanding the mechanisms of the dynamical control subserving the
427 synchronization of cortico-cortical distributed activity.

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