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Assessing Alpha Band Event-related Synchronisation/Desynchronisation Using a Bio-Inspired Computational Model

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Abstract: This paper describes a study of the effects of variation of synaptic connectivity in a thalamo-cortical circuitry using a neural mass model. The oscillatory behaviour of the model output is assessed within the alpha frequency band. The model presented here is a modification of an existing model involving the introduction of biologically plausible synaptic connectivities as well as synaptic structure. Our goal is to study altered event related desynchronisation/synchronisation (ERD/ERS) patterns within the alpha band in Alzheimers disease as observed in experimental studies. ERD is an amplitude attenuation of certain EEG rhythms when an event is initiated or while a certain event is taking place in the brain. ERS is an amplitude enhancement of a certain EEG rhythm when cortical areas are not specifically engaged in a given mode of activity at a certain instant of time. EEG desynchronisation normally blocks alpha rhythms in the EEG due to sensory processing or behaviour. The results show that a decrease in synaptic connectivity induces a time lag in both ERD and ERS peaks in the model output. Furthermore, a deficiency induced in the inhibitory cholinergic pathway results in a distinct effect on time to peak in the ERD/ERS response. These observations are consistent with experimental findings in AD. Variation of the level of interconnectivity has a pronounced effect on the ERS behaviour of the model while the excitatory connectivity in the retino-geniculate pathway during the resting state is more influential on the ERD behaviour.

Key Words: Alpha rhythm, Event-related-(de)synchronisation, Alzheimer's disease, thalamo-cortical model
Category: I.6

1 Introduction

Alpha rhythm in cortical EEG is in the $8 - 12$ Hz band and is most prominent in the occipital lobe, the seat of the visual cortex, and when a person is in a relaxed state with no sensory input through the visual pathway (eyes closed) [Niedermeyer 97]. Rhythmic activity in the brain, or indeed, the whole nervous

system, is understood to be due to the synchronous activity of the underlying neuronal groups. However, occurrence of an event, for example visual perception or motor execution/imagery, brings about an alteration in this synchrony. This and other observations led to the theory of Event Related Desynchronisation (ERD) and Event Related Synchronisation (ERS), defined as "frequency specific changes in the ongoing EEG activity" induced by an event, reflecting a decrease or an increase, respectively, in synchronous activity of the underlying neuronal population [Pfurtscheller and da Silva 1999]. The term ERD/ERS was first introduced in [Pfurtscheller and Aranibar 1977] — ERD is an amplitude attenuation of a certain EEG rhythm when an event is initiated or while a certain event is taking place in the brain; ERS is an amplitude enhancement of a certain EEG rhythm when cortical areas are not specifically engaged in a given mode of activity at a certain instant of time [Pfurtscheller 1998]. EEG desynchronisation is a blocking of alpha band rhythms due to sensory processing or behaviour. ERD usually has a fairly localised topography, and can be characterised by its phasic behaviour and its frequency specificity. There is general agreement that EEG desynchronisation is a reliable correlate of the increased cellular activity in thalamocortical systems during cortical information processing [Pfurtscheller et al. 1998b].

We have been studying changes in alpha [Sen Bhattacharya et al. 2010a, Sen Bhattacharya et al. 2010b] and theta rhythms [Zou et al. 2011a] observed in EEG of Alzheimer's Disease (AD) and corresponding alterations in the underlying neuronal connectivity using computational models [Abuhassan et al. 2012, Sen Bhattacharya et al. 2011c] as well as by image analysis [Li et al. 2011]. The work presented here is based on a classic thalamocortical neural mass model proposed by Lopes da Silva [da Silva et al. 1974] and later used by Suffczyński and others [Suffczyński 2000] to study abnormal neuronal phenomena associated with epilepsy. Subsequently, they extended the model to represent an event-related 'focal-centre–antagonistic-surround' behaviour of mutually inhibiting thalamocortical neuronal populations [Suffczyński et al. 1999]. Such a phenomenon corresponding to limb-movement was reported in [Pfurtscheller et al 1998a]—a task involving hand movement brought about an ERD in the associated cortical region and an ERS in the annular surrounding region. Lateral inhibition is thought to play a crucial role in such centre-surround behaviour observed in neighbouring neurons (sensory) or neuronal populations (cortical) [Sen Bhattacharya 2008].

Suffczyński's ERD/ERS model has been used recently to study such a phenomenon in context to AD [Sen Bhattacharya et al. 2010c]. Motor dysfunction and structural damage to sensorimotor pathways have been reported in early stage AD [Goldman et al. 1999, Agosta et al. 1999]. Moreover, the ERD and ERS within the alpha band is shown to be affected in AD: the latency in cor-

tical response corresponding to an event (finger movement) is increased in AD compared to that in young healthy adults; the spatial distribution of affected cortical areas are altered in AD compared to healthy young as well as old adults [Babiloni et al. 2000]. Studies show that ERD and ERS within the alpha band correlates to tasks that demand attention for example visual perception or limb movement [Dujardin et al. 1999]. Furthermore, the thalamus, considered as a key structure in the generation of cortical alpha rhythms, is now thought to play a crucial role in linking action to perception; all sensorimotor pathways have a branch that are afferents to the thalamic nuclei [Guillery 2003]. Thus, a thalamocortical circuitry based study of ERD/ERS in the cortical alpha band seems justified. In this work, we present a modified version of the ERD/ERS model presented in our earlier work [Sen Bhattacharya et al. 2010c]. The modified model has synaptic connectivity parameters with values similar to that obtained from most recent studies on the Lateral Geniculate Nucleus (LGN) of the mammal (cat) and rodent (rat) thalamus. We observe that variation in synaptic connectivity parameters in the model affects the ERD/ERS behaviour of the output in a manner consistent with experimental findings in AD.

In Section 2, we introduce the modified ERD/ERS model. In Section 3, we detail the experimental methods followed by results and discussion with context to Alzheimer's disease in Section 4. The conclusions derived from the current work and scope for future directions are discussed in Section 5.

2 The Bio-inspired computational model

We have used the Alpha Rhythm model (ARm) to study the neural correlates of Alzheimer's disease and underlying neurophysiology by varying the model synaptic connectivity parameters and observing the associated effect on the frequency of the alpha rhythmic activity [Sen Bhattacharya et al. 2010a, Sen Bhattacharya et al. 2010b]. In [Sen Bhattacharya et al. 2010c], we have presented a preliminary study on the event related behaviour within the alpha band when the ARm is mutually coupled with another of its kind through inhibitory synaptic connectivities. Subsequently [Sen Bhattacharya et al. 2011a, Sen Bhattacharya et al. 2011b], we have extended the ARm by (a) introducing biologically plausible values for the synaptic connectivity parameters based on experimental data [Horn et al. 2000, Jones 2007, Sherman and Guillery 2006] and (b) modifying the structure of a single neuronal population in the model based on more recent work on neural mass models [Ursino et al. 2010]. In the following sections, we present the basis of the modification in the original ERD/ERS model and introduce the modified ERD/ERS model.

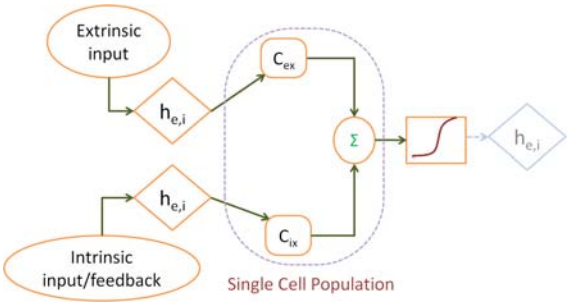


Figure 1: The structure of a single neuron in the circuit, slightly altered from ARm, and consistent with more recent research involving similar models [Ursino et al. 2010].

2.1 a modified structure for a single cell population

The structure of a single neuron population in the model is shown in Fig. 1 and is as in [Ursino et al. 2010]. Each cell population has two sets of connectivity parameters at its input where each set correspond to either an extrinsic (e.g. sensory afferents) or intrinsic input (e.g. intra-population collaterals or inter-population feedbacks). The afferent fibres to the population from a certain type of input (extrinsic or intrinsic) are collectively considered as a unit fibre having a mean firing rate. The synapse made by this fibre is then scaled up by the corresponding connectivity parameter representing the total number of released synaptic vesicles corresponding to this input. This is unlike ARm where the unit fibre carrying the mean firing rate is scaled up by a connectivity parameter representing the total number of fibres in the unit prior to making a synapse.

2.2 A modified Alpha Rhythm model

Using the cell population structure presented in Section 2.1, the original ARm is modified and is presented in Fig. 2. The model has two representative neural populations as in ARm: Thalamo-cortical Relay (TCR) cells of the Lateral Geniculate Nucleus (LGN) and cells of the Thalamic Reticular Nucleus (TRN). The TCR cells receive excitatory input N_r from the retinal cells defined as:

$$N_r = \Psi_{\mu,\varphi}, \tag{1}$$

where $\Psi_{\mu,\varphi}$ represents the resting state background firing activity of retinal neurons and is simulated with a Gaussian white noise having a mean μ and standard deviation φ.

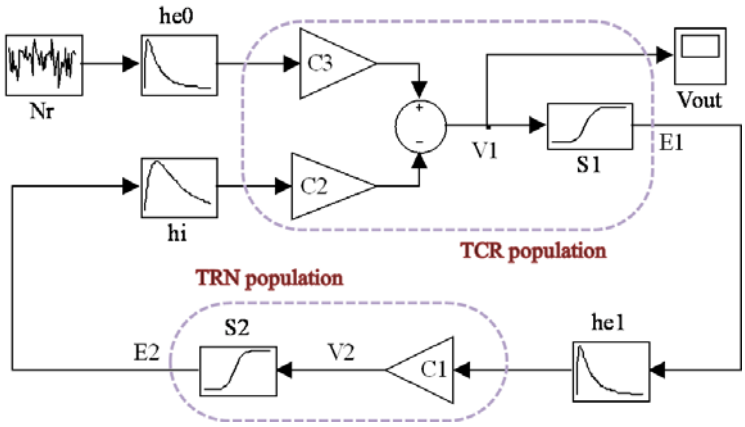


Figure 2: The modified thalamo-cortical circuitry as a modification of the ARm and as simulated in our work with Simulink® in Matlab. The connectivity parameter values in the model are based on most recent experimental data (from the mammalian and rodent Lateral Geniculate Nucleus (LGN)) available from literature [Horn et al. 2000, Jones 2007, Sherman and Guillery 2006] and represent the proportion of excitatory or inhibitory synapses from respective afferents on a single dendritic terminal of a cell. An additional connectivity parameter (w.r.t. ARm) C_3 is also incorporated in the model to represent the proportion of synapses from sensory afferents.

The collective synapses in the model are represented by a function $h(t)$ defined as:

$$h_{e,i}(t) = A_{e,i}(e^{-a_{e,i}t} - e^{-b_{e,i}t}), \tag{2}$$

where the suffixes e or i represent parameters corresponding to either excitatory or inhibitory synapses respectively; $a = \frac{1}{\tau_{rd}}$ where τ_{rd} is the rise-time; $b = \frac{1}{\tau_{fd}}$ where τ_{fd} is the decay-time.

The average membrane potential of each cell population is converted to a mean firing rate $E(t)$ using a sigmoid function S and is defined as:

$$\begin{aligned} E(t) = S\left[V(t)\right] &= g_0\, e^{\gamma[V(t)-V_0]} && \forall V \le V_0 \\ &= g_0\left[2 - e^{-\gamma[V(t)-V_0]}\right] && \forall V > V_0, \end{aligned} \tag{3}$$

where V_0 is the threshold spiking voltage of the neuronal population, g_0 and $2g_0$ are the maximum firing rates when $V(t) = V_0$ for the cases $V \le V_0$ and $V > V_0$ respectively and γ is the steepness parameter of the sigmoid function.

The output of the model is the simulated membrane potential of the TCR population and mimics occipital EEG. The values of all parameters defined in Equations (1)–(3) are given in Table 1.

Table 1: Values of the parameters defined in Equations (1)–(3) [Sen Bhattacharya et al. 2011a].

μ	φ	A_e	a_e	b_e	A_i	a_i	b_i	γ	V_0	g_0
pps	pps^2	mV	sec^{-1}	sec^{-1}	mV	sec^{-1}	sec^{-1}	mV^{-1}	mV	sec^{-1}
312	169	1.65	55	605	32	27.5	55	0.34	7	25

2.3 The modified ERD/ERS model

The ARm was extended in [Suffczyński et al. 1999] to simulate the focal-ERD–surround-ERS behaviour corresponding to motor function. They showed that when filtered within the $8 - 12$ Hz band, the model output exhibits centre-surround characteristics. Subsequently, we have validated the model results and have presented [Sen Bhattacharya et al. 2010c] a preliminary study in the context of AD. In this work, we present a modified ERD/ERS model using the modified ARm as described in Section 2.2. Figure 3 shows the block diagram of the modified ERD/ERS model. The model consists of two modified ARm modules interconnected by inhibitory connectivity parameters C_4 simulating mutual inhibition in the neighbouring population of thalamo-cortical neurons. As in the original ERD/ERS model, there is an additional input M(t) which simulates the cholinergic input to the thalamus from the brainstem associated with the occurrence of an event like visual input, motor activity or mental task and is referred to as the 'modulatory' input. It is simulated by a step function of amplitude 10 pps (arbitrarily chosen) in this work. The modulatory input makes an excitatory synapse through the connectivity parameter C_5 with the TCR cell population, and an inhibitory synapse through the connectivity parameter C_6 with the TRN cell population of the 'target module' (top). The module that does not receive direct afferents from the modulatory input is referred to as the 'neighbouring module' (bottom). The output $V(t)$ of each module simulates the EEG showing the focal-ERD–surround-ERS behaviour in two neighbouring populations of thalamo-cortical cells when in an awake attentive or working state. The biological relevance of the synaptic connectivity parameters in the model are discussed below.

2.4 Biological background of synaptic connectivity

We consider synaptic connectivity, unlike fibre connectivity considered in the original model [da Silva et al. 1974, Suffczyński 2000]. Furthermore, our choice of parameters is based on the most recent available data obtained from cat [Horn et al. 2000] and rat [Jones 2007, Sherman and Guillery 2006] LGN (dorsal). The synaptic connectivity parameters in the model are expressed as a per-

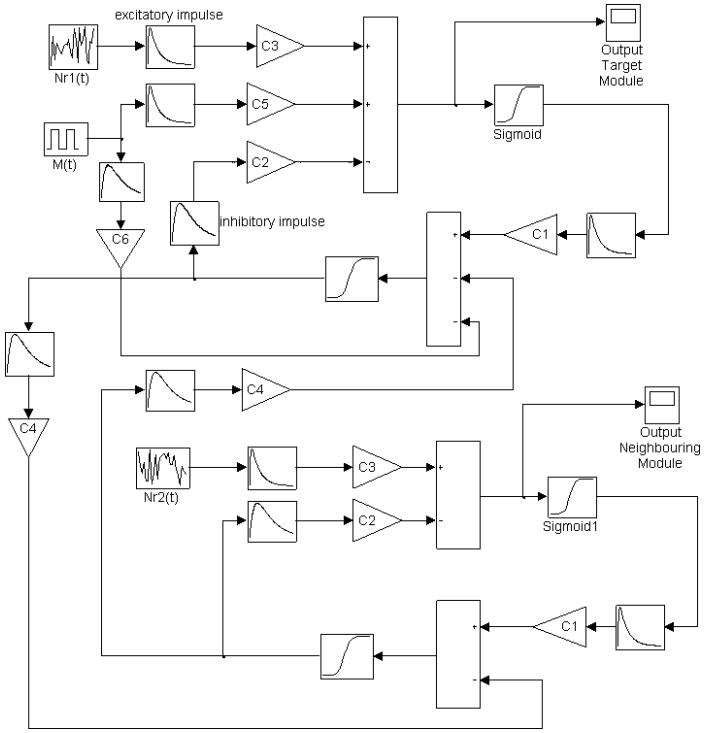


Figure 3: The modified ERD/ERS model as simulated in our work with Simulink® in Matlab. Each module in the model is a modified ARm. The modulatory input $M(t)$ represents an event-related input to the model and makes excitatory and inhibitory synaptic contact with the top module, referred to here as the 'target' module. The bottom module is referred to as the 'neighbouring' module. The connectivity parameters used in the model are give in Table 2 and is based on experimental data obtained from most recent work on the cat and rat Lateral Geniculate Nucleus (LGN) of the thalamus [Horn et al. 2000].

centage of the total number of synapses converging on a single dendrite of the population from different sources.

The pre-synaptic terminals are classified into four types depending on the shape and size of the synaptic vesicle [Jones 2007]: *RL:* Round shaped large sized vesicles, characteristic of the excitatory axonal terminals of the retinal cells; *RS:* Round shaped small sized vesicles, characteristic of the excitatory axonal terminals of the cortical cells as well as those of the BRF and other parts of the brain; *PSD/F:* Inhibitory pre-synaptic terminals of the inhibitory (TRN and IN) cells. We summarise below the quantitative data available in current literature on synaptic connectivities reported in the thalamic cell populations

Table 2: The values of the connectivity parameters in the modified ERD/ERS model shown in Fig. 3 are based on most recent available data from the Lateral Geniculate Nucleus of cat and rat (as reported in [Horn et al. 2000, Jones 2007, Sherman and Guillery 2006]). The value for the parameter C_1 is set at the mean value 35 within the biologically plausible range of $30 - 40$. The modulatory input to the TRN corresponds to the RS terminal synapses and are considered arbitrarily as $\frac{1}{3}^{rd}$ of that of the total number of RS terminals (50%) (the majority will correspond to cortical afferents). Similarly, the modulatory input to the TCR population is $\frac{1}{3}^{rd}$ of 62%, the total amount of synaptic convergence from RS terminals. Mutual coupling parameter C_5 is modelled by the inhibitory synaptic connectivity on TRN from neighbouring cell populations, which is 25% (see Section 2.4).

Connectivity Parameters	C_1	C_2	C_3	C_4	C_5	C_6	C_7
basal value	35	30.9	7.1	25	25	20.7	16.7

within the context of our current work.

- *TCR afferents:* 7.1% from RL, 30.9% from PSD/F and 62% from RS terminals.
- *IN afferents:* 47.4% from RL, 23.6% from PSD/F and 29% from RS terminals.
- *TRN afferents:* The most recent available data on synaptic afferents to the TRN are based on studies on the rat LGNd in 1999 as reported in [Jones 2007]. Both thalamocortical and corticothalamic synapses on the TRN sector corresponding to the rat visual cortex are excitatory (Glutamatergic) in nature and constitute $30-40$% and 50% of the total synapses respectively. The remaining and upto 25% of the synapses are from inhibitory (GABAergic) sources from the other parts of the brain as well as via dendro-dendritic connections among neighbouring cells within the TRN.

The basal values of the connectivity parameters in the model are based on the data above and are given in Table 2. Simulation of the model and results are discussed in the following section.

3 Experimental methods

Model simulation is implemented using the $4^{th}/5^{th}$ order Runge-Kutta ODE solver within the Simulink® environment in Matlab. Total simulation time is 9 seconds with a sampling frequency of 500 Hz. The output is an average of

20 simulations for a certain set of parameter values. The output vector Γ_A thus obtained from each module of the model is bandpass filtered between $8-12$ Hz to extract the alpha rhythm components using a linear phase remez FIR filter. The vector Γ_B thus obtained is averaged using a sliding rectangular window of 400 msec to obtain another vector Γ_C. A reference power R is calculated by averaging the elements of Γ_C corresponding to $t = 1 - 3$ seconds of the simulation time. The normalised event-related-power of the output of each model is calculated as in Equation 4:

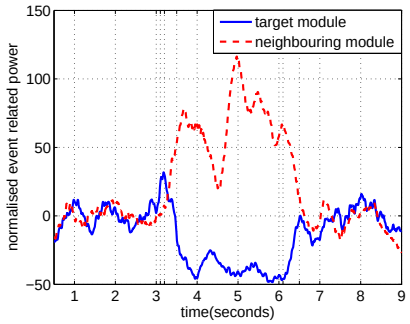
$$\frac{\Gamma_C - R}{R} \times 100. \tag{4}$$

Next, the power spectra of the bandpass filtered vector Γ_B from each module is further subdivided into six parts on the time axis: Γ_A^1 from the $1 - 1.9$ seconds, Γ_A^2 from the $2 - 2.9$ seconds, Γ_A^3 from the $3 - 4$ seconds, Γ_A^4 from the $4.1 - 6$ seconds, Γ_A^5 from the $6.1 - 7$ seconds and Γ_A^6 from the $7.1 - 8.5$ seconds. Γ_A^1 and Γ_A^2 correspond to the pre-movement period, Γ_A^3 and Γ_A^4 correspond to the movement period, Γ_A^5 and Γ_A^6 correspond to the post-movement period. The power spectral densities for each of the vectors Γ_A^1–Γ_A^6 are computed using a Welch periodogram with hamming window of segment length $1/4^{th}$ the size of the sampling frequency and overlap of 50% [Cantero et al. 2009, Sen Bhattacharya et al. 2010a, Sen Bhattacharya et al. 2010b]. Our analysis is based on the relative band power, an analytical method recommended in dementia studies [Moretti et al. 2004], obtained by dividing the absolute power at each frequency by the mean of the total power spectra. The mean of the relative power within the alpha band in each of the six relative power density vector indicate the occurrence of an ERD or ERS. Results are presented and discussed below.

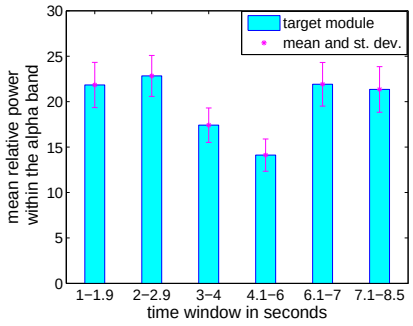
4 Results and Discussion

The ERD and ERS within the alpha band of the target and neighbouring module outputs respectively when the parameters are at their basal values are shown in Fig. 4(a). In our simulation, the event occurs ($M(t)$ is active) from the start of the 3^{rd} second to the end of the 5^{th} second, i.e. for a duration of 3 seconds. The normalised corresponding to ERD and ERS is calculated as in Equation 4.

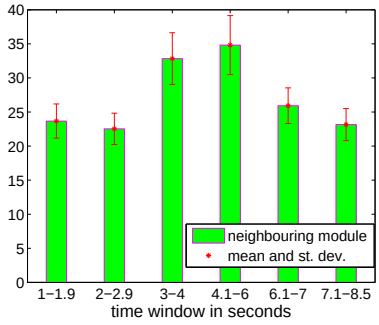
Figures 4(b) and 4(c) show the average of the relative power (defined in Section 3) within the alpha band $8 - 12$ Hz in the frequency domain power spectra of the target and the neighbouring module outputs. The first two bars show the relative power computed during the 1^{st} and the 2^{nd} second, i.e. for 2 seconds during the pre-event period when the modulatory input to the target module is 0 and the event has not yet occurred. A clear dip and rise respectively in the alpha band relative power of the target and neighbouring module outputs is shown by the 3^{rd} and 4^{th} bars. The 3^{rd} bar corresponds to the 1^{st} second



(a) ERD/ERS



(b) ERD

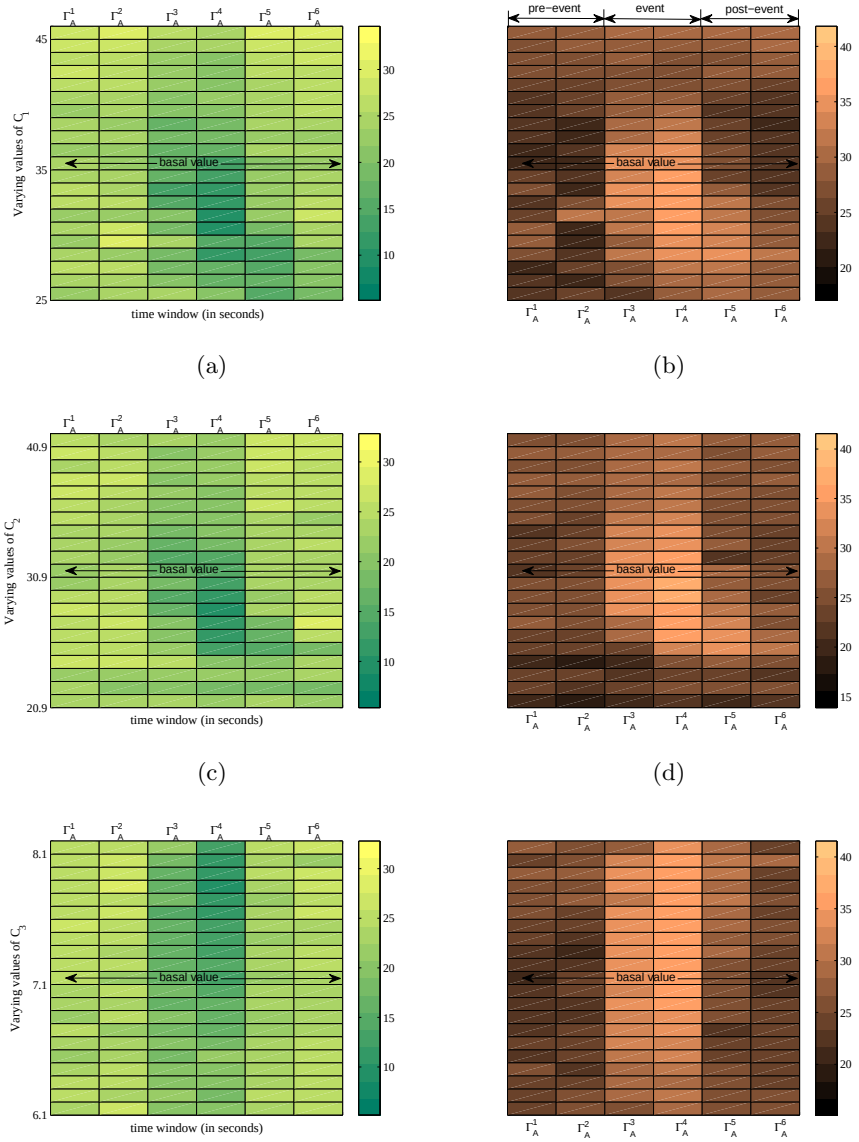


(c) ERS

Figure 4: (a)Event-related Desynchronisation and Synchronisation within the alpha band in the target and neighbouring modules respectively. Event-related (b) decrease in the relative power of the target module and (c) increase in relative power of the neighbouring module within the alpha frequency band.

after occurrence of the event and the 4^{th} bar correspond to the remaining two seconds of the event. The 1^{st} second of the post-event period i.e. after withdrawal of the modulatory input is shown in the 5^{th} bar. The 6^{th} bar shows the 2^{nd} second and 500 msec into the 3^{rd} second of the post-event period. Comparing with our previous work, we observe that the modifying the model by introducing biologically derived model parameters and structure produce a more pronounced ERD/ERS behaviour within the alpha band in the model output.

Next, we vary the connectivity parameters in the model. The parameters C_1, C_2, C_3 and C_4 are varied simultaneously in both models and through $\pm 10\%$ about their respective basal values at a resolution of 1%. Since the basal value of C_3 is in single digit (7.1), we vary it through a smaller resolution of 0.1% and across 1% about its basal value. The results corresponding to variation of



(a) (b)

(c) (d)

(e) ERD (f) ERS

Figure 5: (a)(c)(e)Event-related Desynchronisation and (b)(d)(f) Event-related Synchronisation observed in the model with variation of the parameters (a)(b) C_1 through $\pm 10\%$ of its basal value of 35, (c)(d) C_2 through $\pm 10\%$ of its basal value of 30.9 and (e)(f) C_3 through $\pm 1\%$ of its basal value of 7.1. The time window nomenclature is thus: $\Gamma_A^1 : 1-1.9$ seconds; Γ_A^2: $2-2.9$ seconds; Γ_A^3: $3-4$ seconds; Γ_A^4: $4.1-6$ seconds; Γ_A^5: $6.1-7$ seconds; Γ_A^6: $7.1-8.5$ seconds.

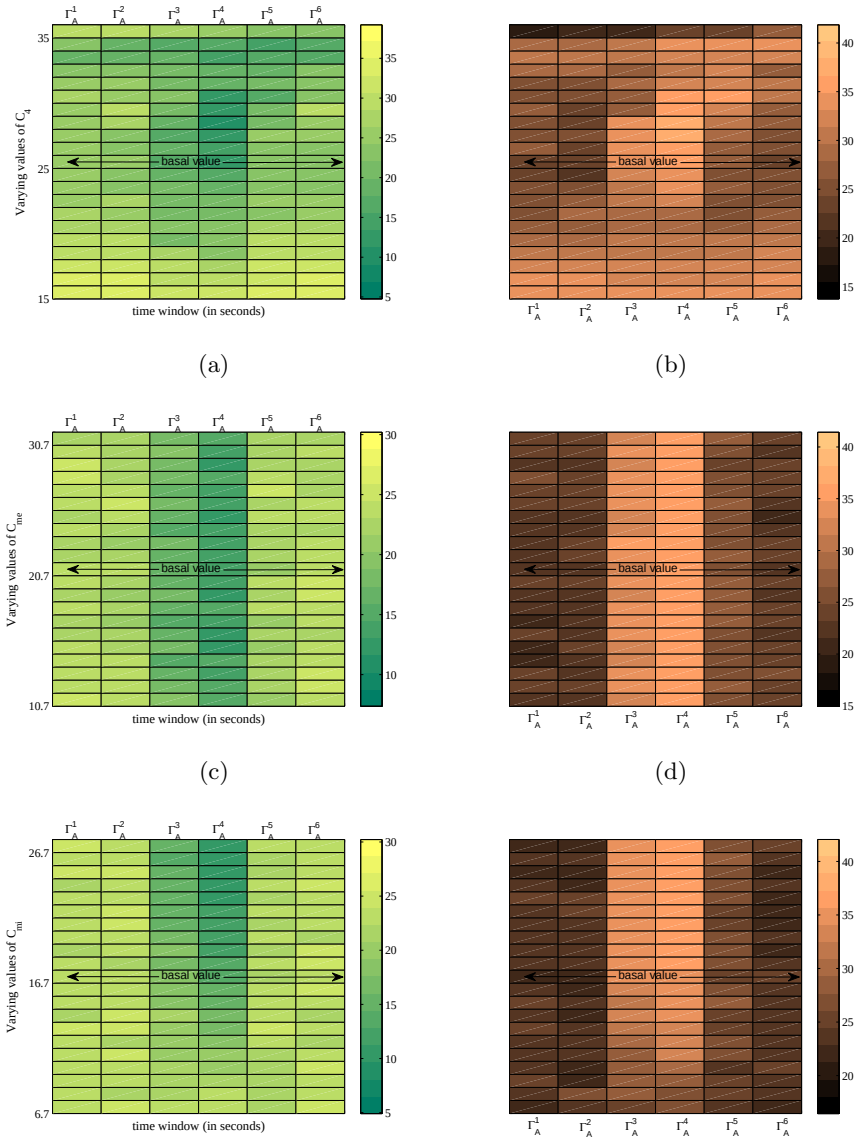


Figure 6: (a)(c)(e)Event-related Desynchronisation and (b)(d)(f) Event-related Synchronisation observed in the model with variation of the parameters (a)(b) C_4 through $\pm 10\%$ of its basal value of 25, (c)(d) C_5 through $\pm 10\%$ of its basal value of 20.7 and (e)(f) C_6 through $\pm 10\%$ of its basal value of 16.7. The time window nomenclature is as in Fig. 5.

the parameters $C_1 - C_3$ are shown in Fig. 5 while that for parameters $C_4 - C_6$ are shown in Fig. 6. The computation of the six columns in each grid plot correspond to the six bars in Figures 4(a) and 4(b), while the rows correspond to the variation of the parameter values about their respective basal values.

In Figures 5(a)–5(d), we observe a similar ERD/ERS behaviour with variation of the intra-module excitatory (C_1) and inhibitory (C_2) connectivity parameters. The module outputs show more pronounced ERD/ERS behaviour with a decrease of both parameters up to about -5% of their respective basal values. Beyond this range, the change in power within the alpha band (corresponding to event related changes) is diminished. We observe that the decrease in the parameter values causes an increased time lag with the ERD or ERS becoming more pronounced in the $4 - 6$ second period of simulation time. This is an interesting result with context to AD and indicates that a decrease in synaptic connectivity of local neuronal population may lead to delayed reactivity in AD as observed in experimental studies [Babiloni et al. 2000].

In Fig. 5(e) we see a distinct ERD behaviour in the target module output for parameter (C_3) values greater than the basal value. A pronounced ERS behaviour in the neighbouring module is seen not only for increasing values of C_3 but also for up to -0.7% of its basal value. Moreover, with decrease in C_3, the ERS behaviour within the alpha band is homogeneous during the event occurrence period, unlike that for the basal value and above, when the ERS behaviour is more pronounced in the later two seconds of the three second event duration. In a previous work [Sen Bhattacharya et al. 2011a] we have shown a proportional relationship between the parameter C_3 values and the power within the alpha band, with the latter decreasing (slowing of alpha rhythms) with decreasing values of the former. A similar decrease in power in the 4^{th} column of Fig. 5(f) is seen with decreasing values of C_3 while still undergoing a synchronised behaviour. This indicates an overall decrease in the ERS peak power within the alpha band with decreasing values of C_3. We speculate that a similar distinct ERD behaviour for decreasing values of C_3 might be offset by the over-riding effects of the modulatory input on the target module . The result indicates that the retino-geniculate pathway associated with the resting state actively affects the neighbouring neuronal populations while being suppressed in the target module during the occurrence of an event.

In Figures 6(a)and 6(b) we observe an increase in the ERD and ERS behaviour in the target and neighbouring modules respectively of the model with increasing values of the inter-module inhibitory connectivity parameter C_4. Furthermore, in the neighbouring module, a distinct time lag in ERS peak is observed for higher values of the parameter. This is in contrast to a similar time lag seen in both ERD and ERS with increase in the intra-module excitatory and inhibitory parameters. The ERD plot corresponding to the increase in C_4

however do not show any remarkable time lag. Again, it might be speculated that the connectivity related to the modulatory input offsets the effect of the inter-module connectivity in the target module, while the neighbouring module remains largely unaffected as it do not have direct afferents from the event related pathway. Based on the results, we speculate that an increase in mutual inhibitory connectivity among neighbouring neuronal populations further enhances the event-related antagonistic behaviour in surrounding neuronal population, thus indirectly emphasising the event-related behaviour in the target module.

The variation of the excitatory connectivity parameter C_5 in the modulatory input pathway show a homogeneous ERD as well as ERS behaviour in the target and neighbouring modules respectively for varying values of the parameter across the range observed in this work as shown in Figures 6(c) and 6(d). It would be of interest to observe the parameter variation over a larger range in future works.

In Fig. 6(e), we see a distinct decrease in the ERD behaviour with decreasing inhibitory connectivity from the modulatory input to the target population. In other words, with fewer inhibitory synaptic inputs, the extent of desynchronisation within the alpha band is reduced, i.e. power is retained within the alpha band and the thalamocortical circuitry shows a reluctance to move out of the relaxed state into a state of cognitive alertness. It is worth mentioning here that the inhibitory input to the TRN cell population of the target module from the modulatory input models the inhibitory cholinergic input from the brain stem to the TRN population of the thalamus. Both cholinergic pathway and inhibitory network connectivities in the thalamocortical circuitry is thought to play a crucial role in abnormal rhythmic activity observed in EEG of AD patients [Sen Bhattacharya et al. 2011c]. The result thus relates to experimental studies in AD and indicates that reduced levels of cholinergic input from the brainstem might induce a state of 'inertia within the alpha band' into the thalamocortical neurons, leading to cognitive and perceptual deficiencies observed in the disease.

In Fig. 6(f), however, distinct ERS is observed up to about -8% of the basal value. Moreover for a range between around $5-6\%$ below the basal parameter value, a distinct lag in ERS peak is seen. Beyond this, alpha band power within the event-related time window falls. We speculate that cholinergic deficiency in the thalamocortical circuitry affects the local population of cells involved in a task in a more pronounced way than it does to the neighbouring population.

5 Conclusions and Future Work

In this work, we have presented a study of Event-Related Desynchronisation and Synchronisation (ERD/ERS) within the alpha band in the context of Alzheimer's disease (AD) using a bio-inspired computational model. Compared to a similar

study that we presented earlier [Sen Bhattacharya et al. 2010c], we have since modified the model by introducing bio-inspired synaptic structure as well as synaptic connectivity. Two instances of the model are mutually connected in a biologically plausible manner; the model thus formed has two 'modules'. Results indicate that a decrease in intra-module synaptic connectivity induces a time lag in reactivity to the occurrence of an event in both modules. This conforms to the time lag observed in movement related reactivity in patients with AD [Babiloni et al. 2000]. Decrease in the event-related pathway inhibitory connectivity has a more pronounced effect on the target module compared to the neighbouring module. This parameter models the cholinergic input from the brainstem to the inhibitory cell population of the thalamus, both of which are believed to behave anomalously in Alzheimer's disease. Our results indicate that a deficiency in the cholinergic input to the inhibitory thalamic cell population might lead to sensorimotor related deficiencies observed in AD. The inter-module inhibitory connectivity, on the other hand, shows a comparatively greater impact on the behaviour of the neighbouring module with increasing response time lag corresponding to increasing inhibitory connectivity. The excitatory synaptic pathway in the event-related pathway shows little variation with alterations in the extent of excitation across the range observed in this work. We speculate that the excitatory connectivity would only affect event related behaviour in a thalamocortical population if varied significantly beyond its normal range.

The results summarised above gives meaningful future directions to the work, the most important of which is to induce increased biological plausibility in each of the thalamocortical modules in the model. We would like to modify the model presented in this work by upgrading each module in the model to a (relatively) more biologically-plausible thalamo-cortico-thalamic model in [Sen Bhattacharya et al. 2011c]. This may shed more light on the inter-module pathways affecting the event related response and behaviour, both in the functional and dysfunctional brain of AD. Furthermore, study of cholinergic pathway is crucial to neuronal network studies in AD. However, this is lacking in the thalamo-cortical models that we have presented thus far. This aspect is being dealt with in our current work. Once developed, we intend to introduce this into the ERD/ERS model in near future.

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