

Neuronal structures and network behaviour

(xxx): calculate it

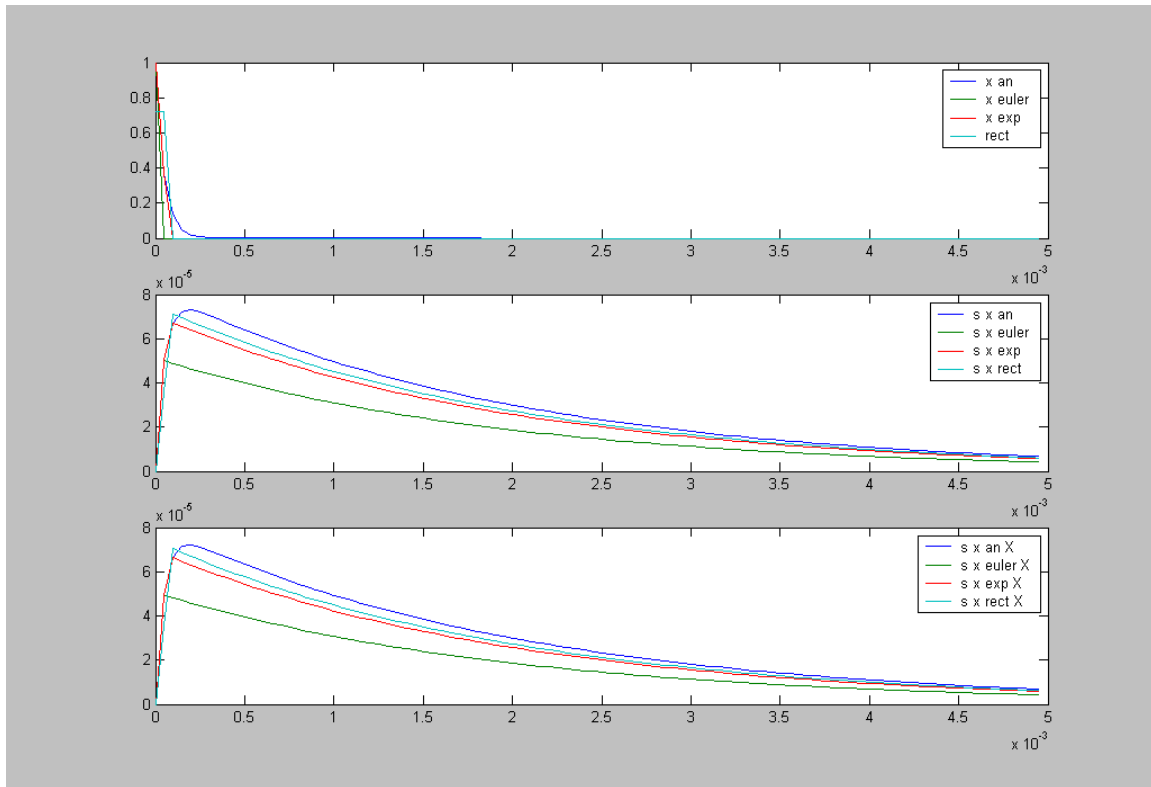
(yyy): simulate it for expectation estimation

Sampling

The minimum problem to be avoided with sampling is that the decay of a variable x can be larger than x . $x' = x / \tau_x$, so if $(x / \tau_x) * \text{step}_x > x$ then illegal values are obtained. Hence, for legal values, $(x / \tau_x) * (1 / F_s) < x \iff F_s > 1 / \tau_x$, with F_s the sampling frequency. Wang's fastest τ_x is 0.05 ms, hence this consideration requires $F_s > 20$ kHz. Rocky4 uses an exponential integration method which improve performance.

As long as simulations do not turn out to be too cumbersome I prefer to use Wang's original quick AMPA time constant together with a high (20 kHz) sampling frequency, since the precise effects of manipulating τ_x and conductances to reduce the necessary F_s are unknown (e.g. interaction with the manipulation of delays), and such manipulation only provides linear improvements to simulation time. It could also be so (xxx or yyy) that approximating the AMPA x dynamics by a rectangular function provides a satisfactory subsequent approximation to the PSP.

A rectangular approximation could be set up as follows. The magnitude of the x variable as measured in energy can be held constant by setting the area under the rectangle to the area under the decay function. Since $x(t) = \exp(-t / \tau)$, the integral from 0 to a equals $[-\tau \exp(-t / \tau)][0, a]$ and hence, for as a goes to infinity, the integral goes to τ . This leaves the height - width ratio and the location of the rectangle unspecified. Keeping the location of the means equal provides a first step. The time t_m at which half the effect of the exponential is spent is found by the equation $[-\tau \exp(-t / \tau)][0, t_m] = \tau / 2$, hence $t_m = -\tau \ln(0.5)$, which is $3.4657e-002$ ms for a τ of .05 ms. If the rectangular function responds to input with an immediate step, as does the exponential function, equality of the means specifies a width of $2 * t_m$, and hence a height of $\tau / (2 * t_m) = \tau / (-\tau \ln(0.5)) = -\ln(0.5) = 0.6931$. At 20 kHz, a rectangular x -pulse seems to approximate the effect of a decaying x on s better than exponential integration.



Interestingly, approximations all seem to systematically underestimate the analytical solution, which suggests the existence of a formula for increasing the initial step size of x so that this underestimation is corrected. The approximations converge to the analytical solution as F_s increases. Since synaptic weights will be heavily manipulated at later stages to evoke sensitive network behaviour, this relation between optimal step size and F_s does not seem worth pursuing, except from mathematical interest. For the same reason, the rectangular approach itself doesn't seem to offer substantial improvements over exponential integration at 20kHz.

Parameters

Time constants: AMPA: x : 0.05 ms, s : 2 ms. NMDA: x : 2 ms, s : 80 ms. GABA: s = 10 ms.

Conductances: AMPA: 0.3. NMDA: 0.05. GABA: 0.1.

Normalization: divide by average number of incoming synapses per incoming module.

Weights: often show critical regions, where small changes strongly influence network behaviour. E.g. in *ie*, excitatory to inhibitory strength determines whether a winning population continues to oscillate or eventually spikes continuously.

Architectures and behaviour

Syntax describing pair of populations (sets) A and B:

A: Neuron type in A (number of modules in A) [internal connection type in A] =A to B synapse types> <B to A synapse types= B:neuron type in B [internal connection type in B] (number of modules in B).

Synapse types 0, 1 and 2 are AMPA, NMDA and GABA respectively.

E.g., the binary system of 5 internally NMDA connected excitatory modules undergoing recurrent inhibition from a single inhibitory population is described as:

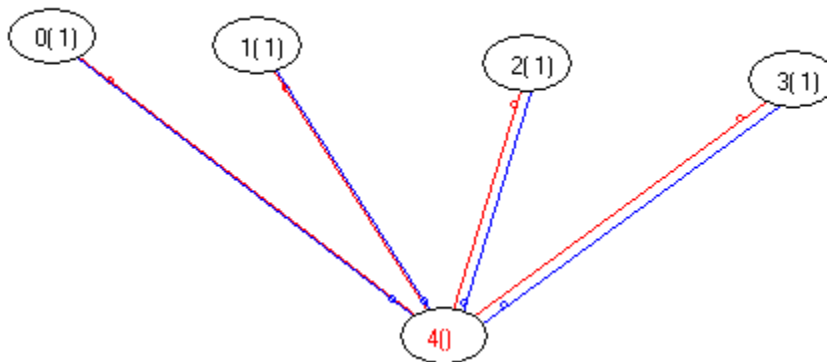
A: exc (5) [1] =0> <2= B: inh (1)

The general theoretical approach taken here is to find hypotheses that determine relations between functions that can be plugged into sensory input or motor output and specific patterns of neuronal behaviour, especially but not exclusively oscillations and phase-locking. The hypotheses take the form of architectures of neuronal populations that respond to simple activation with flexible and complex adaptive behaviour. The whole story and line of argument concerning these hypotheses will eventually lean heavily on anatomical reality, various kinds of psychophysiological data and behavioural characteristics (stability, speed, etc). The physical origin of the necessary simple inputs will be related, if possible, to prefrontal cortex, thereby specifying its role in relation to posterior areas and targeting the next step in hypothesis generation. That is, first possible physical / neuronal functions of the PFC will be studied in relation to neuronal structures with specific behavioural characteristics, and subsequently the same approach will be applied to the mechanisms by which the PFC adaptively performs those functions in the correct context. As a general theoretical idea, these mechanisms within PFC are expected to be relatively simple, only having to blindly induce some kind of disturbance in neuronal architectures which respond to such disturbances in a functional manner, e.g. switching patterns or resolving competitions. That is, the "intelligence" of the system is locked in the systems to be controlled, while the controlling structure in a sense knows least of all what it's doing.

Many-to-one-to-many recurrent inhibition (eie).

A: exc (4) [1] =0> <2= B: inh (1).

e.g. \comp_osc\compOsc1.txt

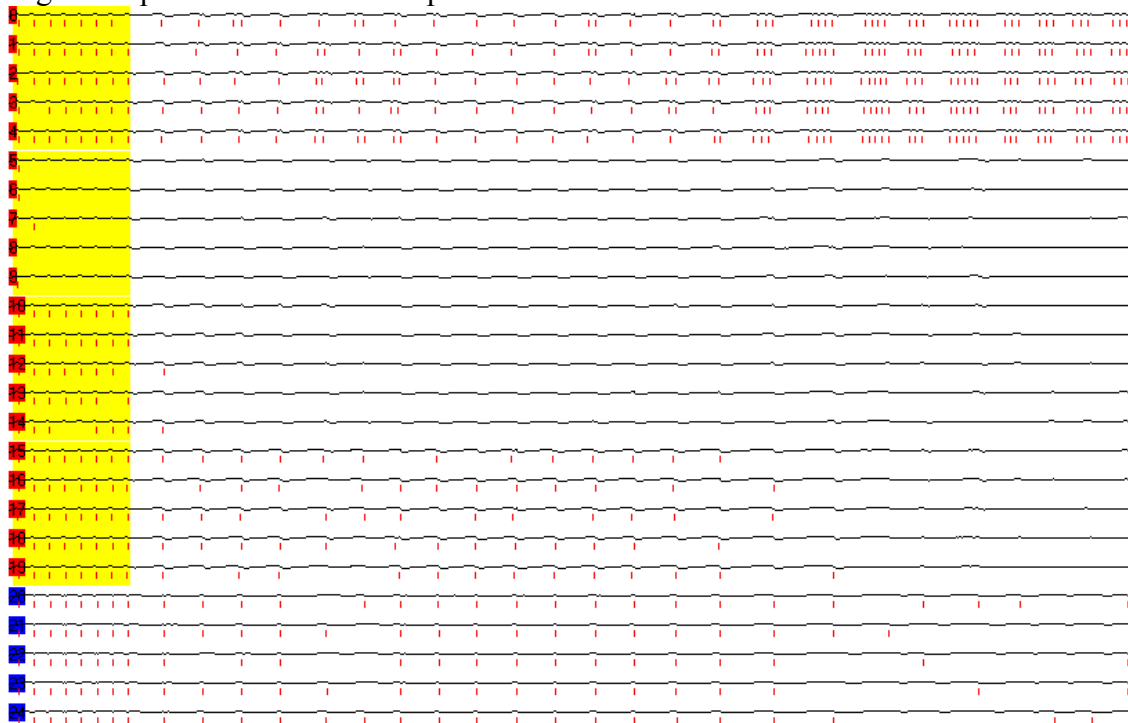


Neuroscientific relevance: resolution of competition (for phase / spiking), hence task-set selection; logical links between ERD and movement initiation.

a. Basic behaviour:

Stimulating all exc pop's leads to initial in-phase oscillation, followed by competition resolution so that a subset remains oscillating. The mechanism is an earlier firing of (a subset of) one population, leading to total inhibition of other populations that have not yet fired (as often).

Once competition is resolved and a stable, part-oscillating state is achieved, changing the state demands more activation / external disturbance than was needed to initiate it. This is consequence of the periodic inhibition preventing externally activated populations from firing in the presence of afferent input.



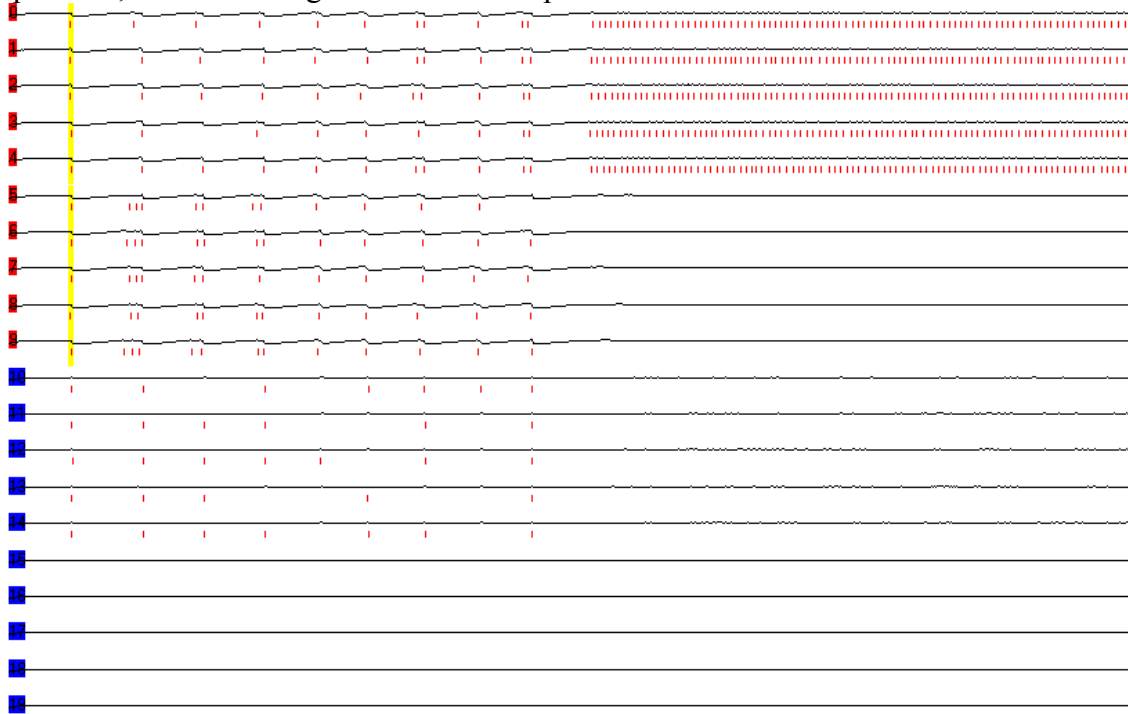
The input onto a specific inhibited population can change the state if it is strong enough that an inhibited population that has just been inhibited will fire sooner due to afferent input than the active population will fire due to internal NMDA dynamics. The input must also be present long enough to survive a period of inhibition; that is, to be sure of a change, afferent input must be present twice as long as would be necessary to lead to a spike, since in the worst case inhibition occurs at the time a spike would have occurred.

Blind input to all excitatory populations can similarly change the state if it strong enough to let at least one inhibited population fire. At that time, recurrence occurs and the inhibitory advantage of the previously activated population is lost.

b. Effect of changing weights:

Reducing the NMDA weight introduces more variability to the internal dynamics, increasing the tendency to resolve competition. With a high NMDA weight, the noise can be too weak for populations to fall behind.

If the exc2inh activation is too weak for a single winner to initiate inhibition, the oscillation is lost and the winner fires at a rate limited only by internal dynamics. That is, oscillation reflects ongoing competition. Resolution involves a loss of oscillation and an increase of rate. Restoring stability could involve activating the whole excitatory population, re-establishing oscillation / competition.



The process requires weights in a certain logical range. The exc2inh weight w_{e2i} must evoke a prompt recurrent inhibition so that being first to fire does tend to inhibit populations with somewhat later spikes. However, the weight must also be low enough that a winning population does not initiate inhibition.

If w_1 is the weight necessary for a single module to just activate the inhibitory population, w_{e2i} must be below this. It must also be above the weight w_2 at which two or more modules firing in phase are just unable to activate the inhibitory module. In the current simulator, an AMPA weight of 0.4 and an NMDA weight of 0.2 results in an oscillation-to-constant firing pattern (compOsc2.txt); that is, w_{e2i} exists (xxx).

c. Effect of internal AMPA connections:

With weights as above, internal connections prevent constant spiking of the winner by initiating two spikes in succession, thereby activating the recurrent inhibition.

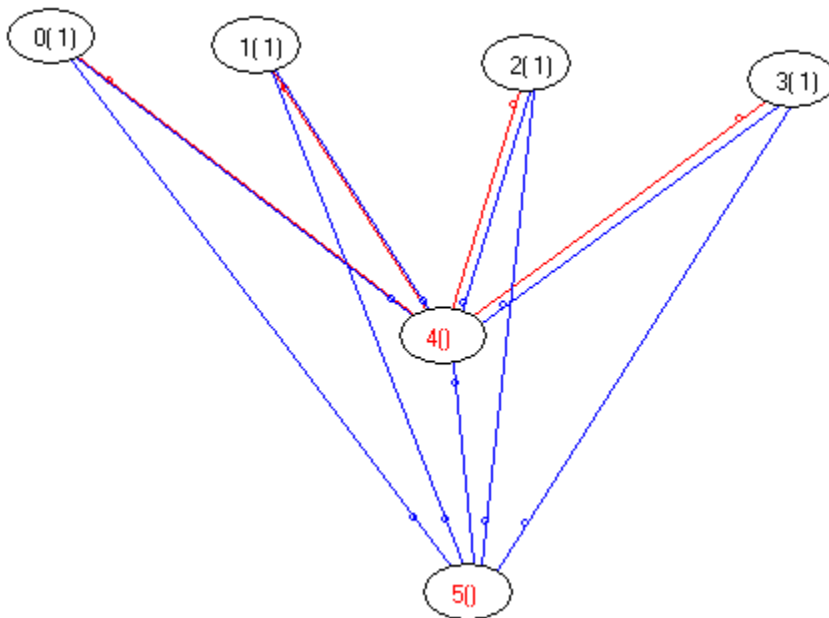
2. Pass oscillation, stop constant firing (2i, thresholded inhibition)

compOsc3.txt, compOsc4.txt.

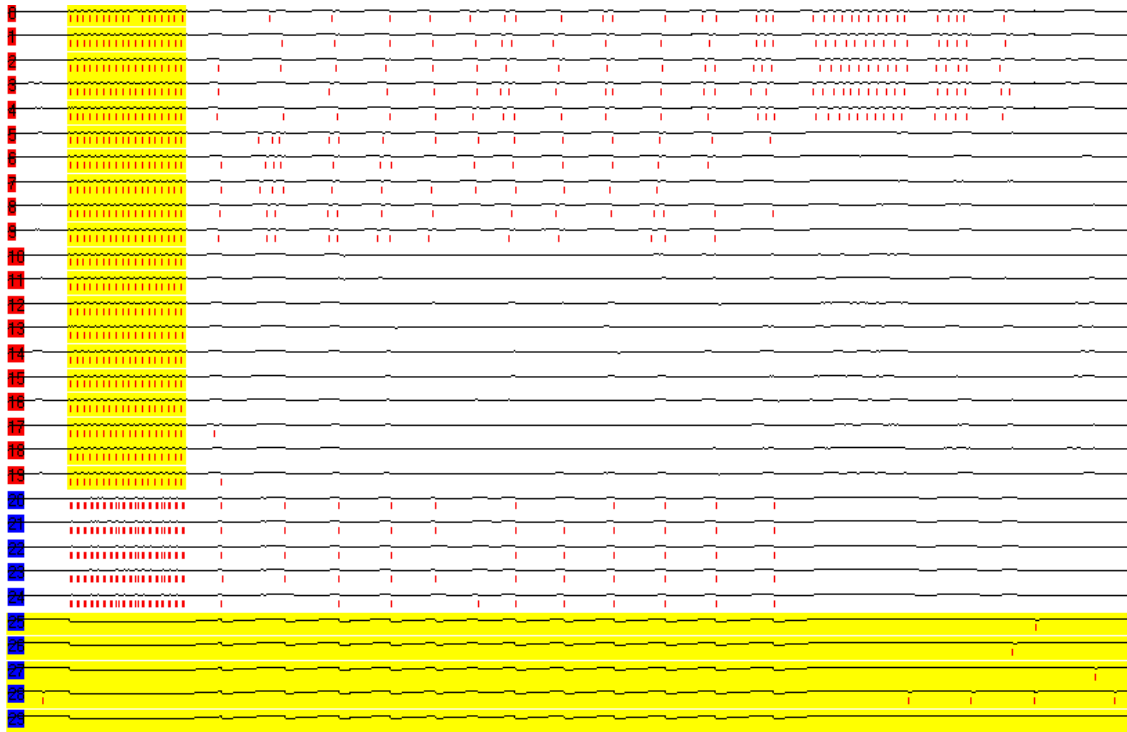
Neuroscientific relevance: meaning of ERD and ERS, strategic / adaptive opportunities in control of ERD / ERS.

After a winning subset goes into constant spiking, what self-regulatory mechanism at the network level (NLSR) could control that? Such a network mechanism could be embedded into transient function and adaptation, as neuron-level mechanisms (e.g. STD) could not. One approach to NLSR would be through disinhibition of inhibition (2i, compOsc3.txt). While the common inhibitor (CI) is controlling the excitatory populations, it might also inhibit a second inhibitory population that receives constant activation. In syntax:

A: exc (5) [1] =0> <2= B: inh (1)
B =2> C: inh (1)
C =2> A



For this architecture to work, the activation of C must be weak enough to be inhibited by B. The time between the last inhibitory spikes from B and the first sufficiently in-phase firing of C determines how long the active excitatory population spikes after winning the competition. The influence of C on A must be high enough to prevent internal NMDA dynamics from arising. After the active excitatory population is inhibited, only C continues to fire.

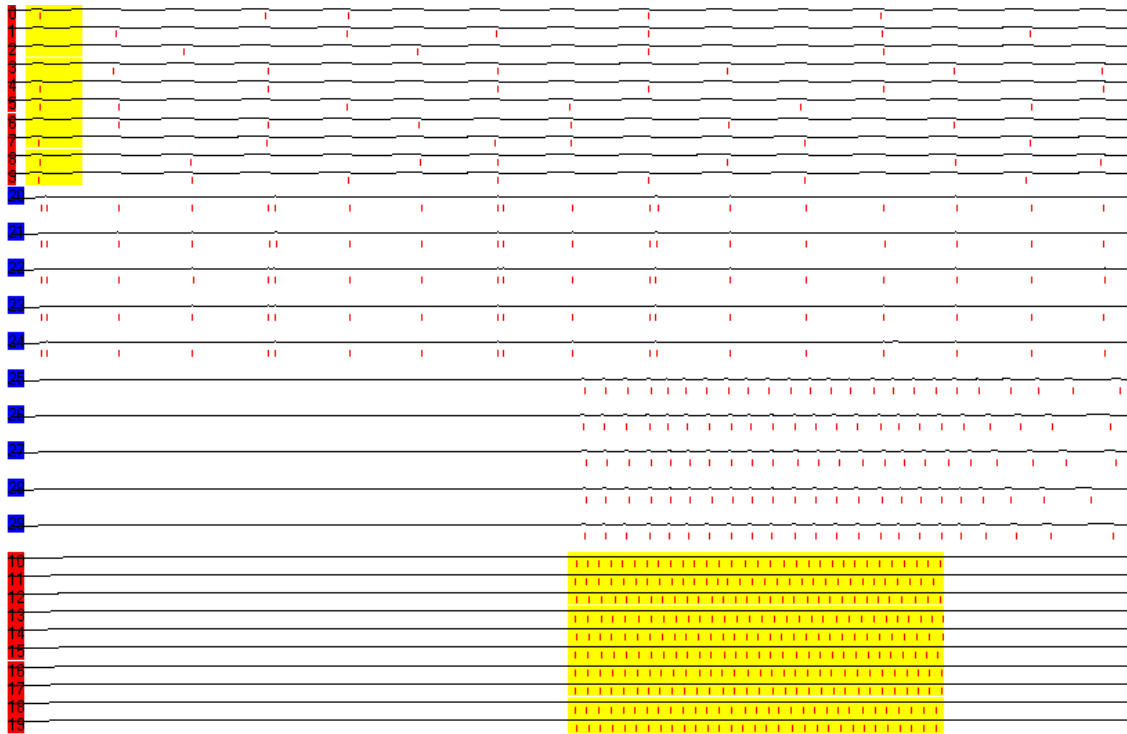


This 2i architecture seems reasonably stable (yyy). It lacks a role for ERS, and the only interesting activity occurs just after cessation of activation. As with eie, the pattern of weights determines network behaviour. This pattern is determined by logical ranges. E.g., weaker activation of C leads to a longer constant firing period. The range of C activation in which behaviour is sliding is quite narrow (0.1 - 0.11), bounded by the requirements to spike only after a number of spaced inhibitory inputs, while still eventually spiking (xxx). The consequences of AB weight patterns are as in the basic eie architecture.

Differential responses to slow oscillations and constant firing should also provide a hypothetical pass oscillation / stop constant mechanism. oscCon1.txt shows the existence of such a weight (xxx as function of PSP dynamics?) in a low-NMDA ei (INei) architecture. Syntax:

A: exc(5) =1> B: inh(5)

The use of NMDA connections allows the desired contextual coding, as opposed to the more direct coding of phase-locked firing (figure below; the bottom population does not have internal NMDA connections). The e2i NMDA weights are 0.25 of default strength.



In the INei architecture, the absence firing is coded as low activation of the inhibitory population, low-frequency oscillations are coded as increased excitability but not by firing and constant firing is coded as inhibitory spikes. This leads to two interesting cases of critical-weight behaviour.

First, in the case of recurrent inhibition, $i2e$ weight determines whether oscillation or complete quiescence occurs after constant firing. At the default weight, oscillation occurs. For parsimony, this should be the expected case if oscillations can occur at all. Otherwise, a higher $i2e$ weight for the NMDA-activated inhibitory population would have to be assumed. Furthermore, this structure explains the (re-)occurrence of ERS after ERD.

Second, if the weights are such that they code the pass-oscillation / stop constant (po/sc) behaviour, an interesting hypothesis on the role of preparatory ERD is generated. If oscillatory behaviour can, without causing spikes, push the mean membrane potential of the po/sc inhibitory population towards its threshold. This makes it more sensitive to further input, and hence quicker to respond to constant firing or another form of increased input. Thus, reducing oscillatory activity prior to movement might be used to allow a longer period of constant firing before self-regulation (which can be identified, in this case, with ERS) sets in. This offers an explanation to the paradoxical situation in which increased activity at a frequency band related to brain-muscle communication has a negative relation to preparation. A second paradox, that of ERD itself, suggests a class of hypotheses described below.

oscCon6.txt attempts to simulate such a differential inhibitory coding scheme (coding for competition via AMPA, coding for constant firing via NMDA) with multiple excitatory subsets, but this reveals an interesting problem. It was difficult to find an NMDA weight

such that multiple subsets spiking did not evoke a response, while constant firing of a single subset did. This suggests (xxx) that an enforcing architecture / control system (e.g 2i) should be looked for, as opposed to a very specific combination of weights with, as yet, no existence proof.

3. Global - local oscillation paradoxes

Relevance: Occurrence of both ERD and EEG-EMG coherence / sensitivity at 20 Hz.

As yet less systematic simulations:

Theta and competition resolution

Files:

test\manyWang1.txt

Originally to test normalization. Now subsets of mods seem to survive resolution.

What laws does that process follow ***?

With 10 mods, multiple mods (2, 3) can survive to 2 seconds.

H1: delays and neuronal self-regulation will increase noise and loss of osc.

H2: external disturbance causing noise can induce collapse to "choice": control by noise?

Competition biasing / Preparation by relaxation

Files: Frankie

Frankie1:

exc lat inh: works as comp and as prep.

Osc act in prepared pop.

Files: motorPrep1

mp2:

common inhibition: while both activated rhythmic, then noisy-rhythmic till one wins, which then goes high rate while the other is dead. Hence: excitation of the subsystem is translated into a lack of competition and low firing rates.

Desinhibition

Files: DesInhERD

desInhERD1:

osc -> high-rate -> osc; high rate at desinh.

desinh required high i-i weight (x10)

Motor ERD and prep

Files:

Control: initiation of competition in interaction with changing external / memory circumstances

Alpha: blocking function (thalamus). Intracortical function? Prevention of capture.

If-then / S-R phase coding. Generalization to multiple pairs, matching-mechanism for decoding: this should work!. Empirical testability. Compositionality.