The neural circuitry necessary for decision making by evidence accumulation

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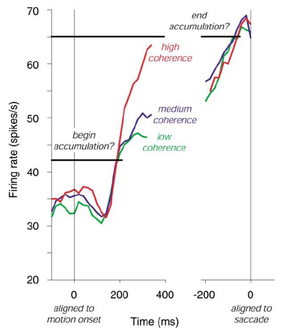
Summary

Growing neurophysiological evidence indicates that during decision making single neurons can integrate the sensory evidence in favour of a particular response. Mathematical models can describe the dynamics of this evidence accumulation process (Ratcliff et al, 2003; Reddi & Carpenter, 2000) and promise to connect the behavioural and the neurophysiological levels of description, but they do not address the neural mechanisms which could implement such accumulation. Our analysis demonstrates that it is implausible that evidence accumulation is the result of endogenous neural processes alone, but instead is probably the result of modulated positive feedback in neural circuits. We present an established model of the circuits between the cortex, thalamus and basal ganglia (Gurney et al, 2001) and show how it is capable of supporting cortical evidence accumulation. The model also deals with previously unaddressed problems of how accumulation is initiated, modulated and terminated and/or overridden. Our model provides an established circuitry that can implement and control the accumulation dynamic, something not provided by descriptive models.

1. Decision making by evidence accumulation

The accumulation of sensory evidence appears to subserve decision making. *Right*, from Gold & Shadlen (2002) mean firing rates of lateral intraparietal (LIP) area neurons during a motion discrimination task. A ramp like increase in firing is time-locked to stimulus onset. Rate of increase is dependent on stimulus strength. Response initiation is time-locked to threshold crossing

How can a signal accumulation dynamic be created in neural circuits?



2. An endogenous neuronal process?

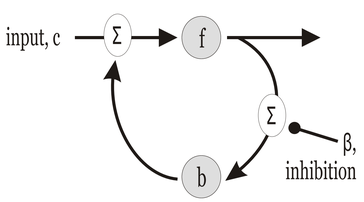
It is unlikely that signal accumulation is the consequences of direct integration, endogenous to the single neuron:

- Adaptation to constant input is the norm in cortical neurons.
- The membrane time constant that would be required to evince accumulation over the durations observed is implausibly large (full analysis in Humphries et al, submitted).
- Fast activity decay rates post-decision are incompatible with a slow integration
- Accumulation rates vary according to task, requiring additional control input even if accumulation *per se* is endogenous.

3. Can positive feedback create appropriate accumulation?

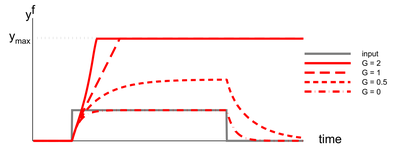
In order to investigate the minimum plausible circuitry of simple model neurons that can generate signal accumulation we define the following circuit, *shown right:*

Let **f** and **b** be populations of simple leaky integrator neurons which produce outputs y_f and y_b respectively. Let each output be weighted by w_f and w_b respectively and passed and summed as shown. **f** receives input *c* and the feedback relay between **b** and **f** is subject to inhibition β.



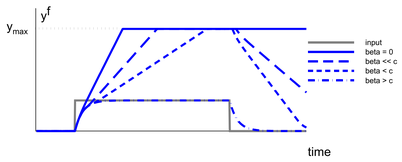
For this simple feedback system the output, y_f is defined by: time *t*, the input *c*, the inhibition on the feedback β - if any (see section 4) - and the weights on the outputs. The closed loop gain is given by $G = w_f . w_b$

Positive feedback alone, without inhibition, can create signal accumulation but introduces the control problem of how the positive feedback circuit is broken. For G < 1 accumulation is not guaranteed to reach threshold. For G ≥ 1 the signal does not reset once input has been withdrawn. G = 1 creates linear growth, making the circuit a perfect integrator



4. Positive feedback requires control via inhibition

Tuning inhibition, for G = 1, allows the control of the rate of accumulation. β >0 allows decay of signal accumulation with the disappearance of the input. β > c prevents accumulation to a level greater than c.



5. Thalamo-cortico loops provide this generic circuit, the basal ganglia provide inhibition control

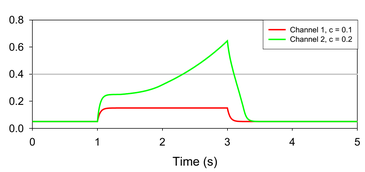
Having established that a modulated positive feedback loop is the minimum plausible generic circuit for initiation and control of signal accumulation, are there any candidate neural circuits for the generation of cortical signal accumulation in decision tasks? We propose that thalamo-cortical loops are the cause of signal accumulation in cortical cells implemented in decision making:

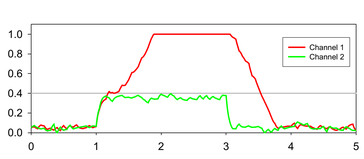
- Reciprocal excitatory relays between thalamus and cortex are well established.
- The basal ganglia provides a source of inhibition, which can be modulated by cortical inputs.
- The basal ganglia is implicated in resolution between competing behaviours (Redgrave et al, 1999).

We have proposed a model of behaviour switching by the basal ganglia, embedded within thalamo-cortical circuits and based on the known functional anatomy of the connections between the nuclei. The details are presented in Gurney et al, 2001 & Humphries et al, 2002. The essentials are represented schematically in the figure *far right.*

This model provides the circuitry for modulated positive feedback, with additional provision for the resolution of conflicts. Model properties that make it suitable for generating cortical signal accumulation in support of decision making including:

Selection between two competing inputs generates cortical signal accumulation (*right*). Inputs are both step inputs of onset t = 1 and offset t = 3.





The addition of noise results in error selections on some trials (*shown left*). In these cases, the channel with the lower average input provokes selection because of positive feedback on a transient rise in the signal.

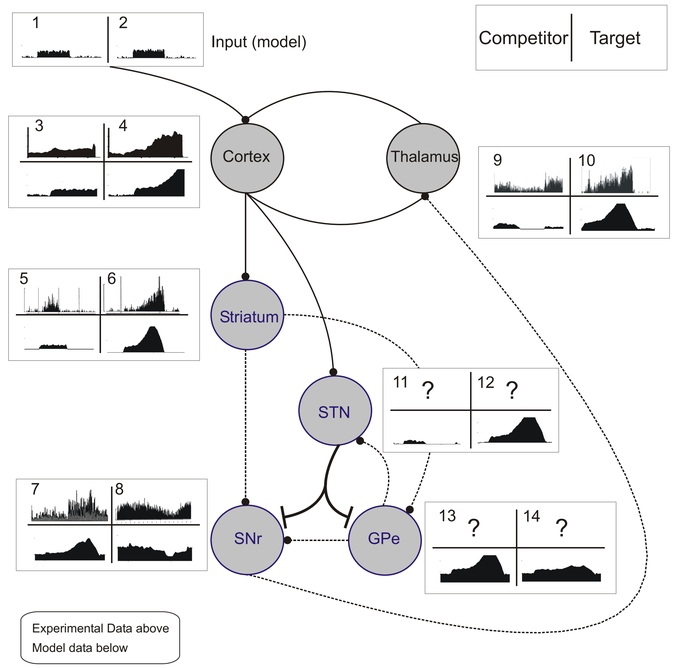
Additional properties:

- Noise allows selection between matched inputs.
- Inhibition from basal ganglia means arbitrarily small inputs do not cause selection.

6. Model matches activity data and makes predictions

The addition of noise allows simulation activity to be matched to recorded activity in neural loci involved in decision making. Where recording data is not available the model makes predictions about the patterns of activity that it should be possible to find in those nuclei.

Two noisy input signals and characteristic simulated activities in each of the model loci are shown, with matched recording data where available (*below*):



Predictions:

- Cells which demonstrate the activity predicted should be found in the subthalamic nucleus (STN) and globus pallidus external segment (GPe).
- Disruption of normal basal ganglia function should affect cortical choice-related activity.

Sources for data fitting

- (3,4) Adapted with permission from Sato et al. (2001).
- (5,6) Hikosaka, Sakamoto, & Usui (1989b) and Hikosaka, Sakamoto, & Usui (1989a).
- (7,8) Handel & Glimcher (1999) and Hikosaka & Wurtz (1983)
- (9,10) Watanabe and Funahashi (2004).

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