A hippocampal subfield model for the modulation of theta dynamics

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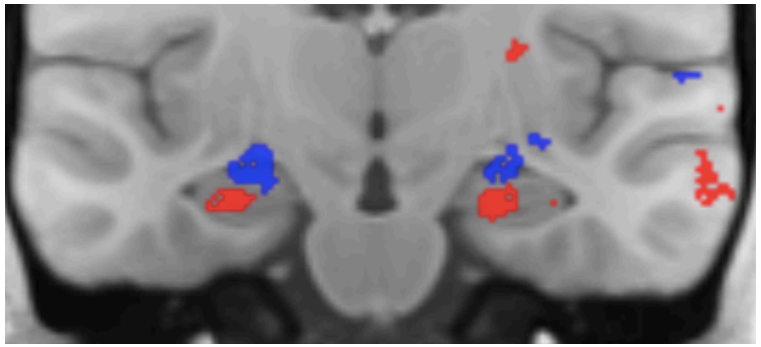
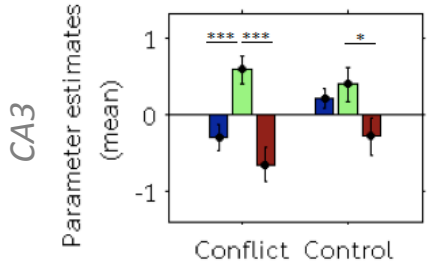
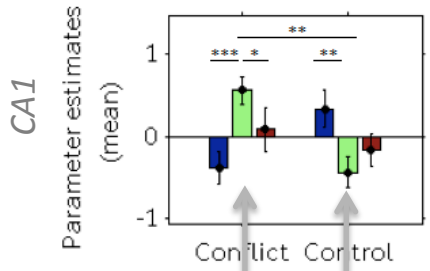
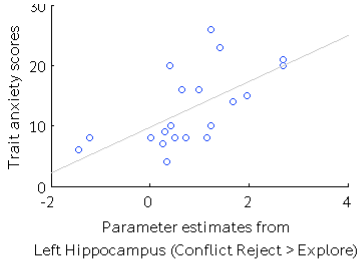
Starting with phenomenological data:
Emergence of aversive/behavioural inhibition signal moving from CA3 to CA1

Hippocampal activation associated with choice during a gambling task







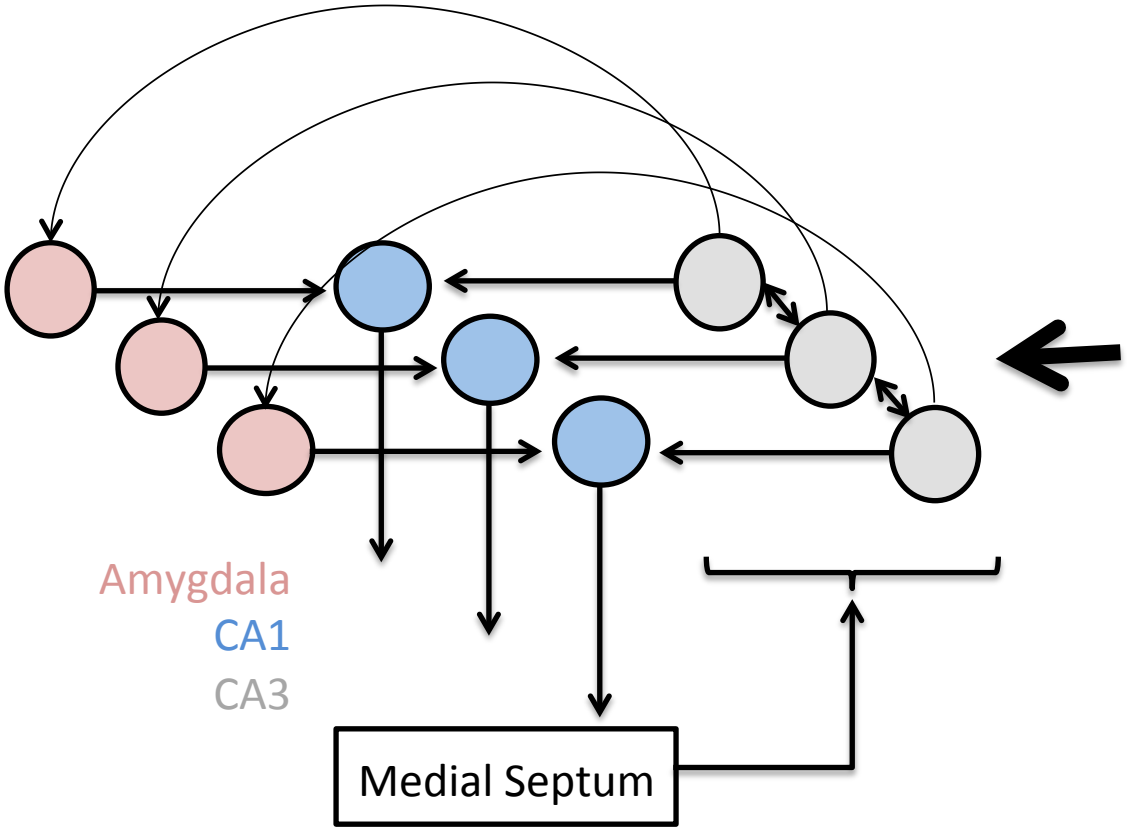


Rejecting an instrumentally aversive gamble

Rejecting an gamble that is aversive in another context, but *not currently so*

General hypothesis: Hippocampal mechanisms (pattern completion/separation) should be able to modulate medial septum activation/theta oscillations (=substrate of anxiety)

Project hypothesis: Dynamics within a circuit consisting of hippocampal subfields CA3, CA1 (comparator), amygdala and medial septum (theta generator) should capable of bi-directionally modulating theta (amplification & damping down)



CA3 neurons spike spontaneously (constant p(spike) per timepoint), or are driven by direct inputs

CA3 neurons are recurrently connected, and project *weakly* to Amygdala neurons

CA1 activation requires temporal integration of CA3 and Amygdala inputs

MS unit is activated by CA1, and increases spontaneous p(CA3 spike) with a theta frequency (i.e. makes CA3 neurons more likely to spike in certain time windows). MS unit activity persists, then decays.

Hypotheses

- Strengthening of recurrent CA3 connections should reduce the input necessary to cause MS activation. Beyond a threshold, MS should be strongly active, and circuit produces positive feedback to itself.

- When network is in a state of positive feedback resulting in strong/sustained MS activation, active weakening of CA3 recurrent weights should then reduce MS activation