# How the emotions of others capture our attention: A human amygdala disruption study

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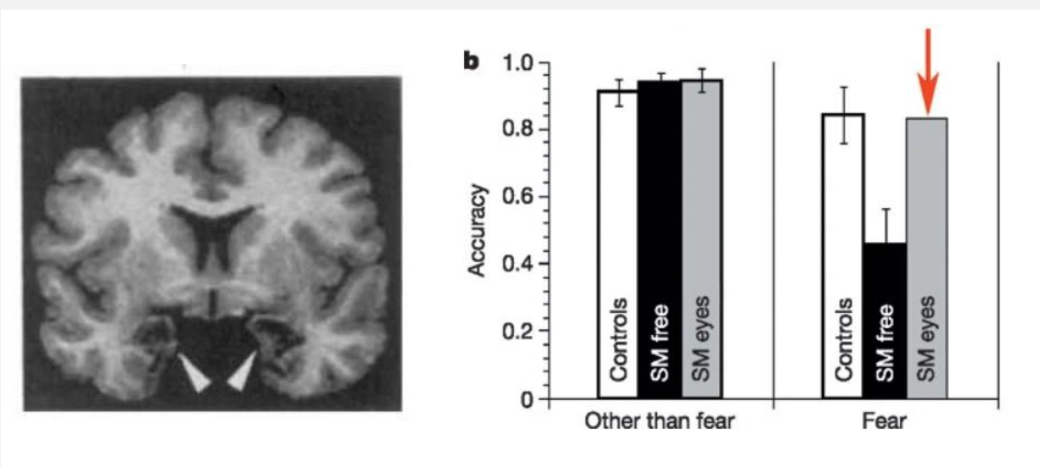
## 1. Introduction

We instinctively look towards faces expressing strong emotions such as fear. This survival-critical response – potentially alerting us to danger – requires rapid detection (sensory) and orientation (motor) towards a face *in advance of* conscious recognition of the emotion.

The neural basis of this response may involve the amygdala, but rare studies of humans with chronic amygdala damage conflict reporting impaired, unaffected or heightened *detection* of fear. How the amygdala contributes to a spatially specific *oculomotor* response remains unexplained.




***Unexplained phenomena.** SM with bilateral amygdala damage improved her ability to recognise fear when instructed to look at the eyes of others (Adolphs R et al, Nature, 2005).*

## 2. Aim

We aimed to investigate & clarify the neural mechanisms underlying this instinctive response in humans.

Hypothesis: *The amygdala is necessary for rapid detection and spatially-targeted orientation towards faces expressing fear.*

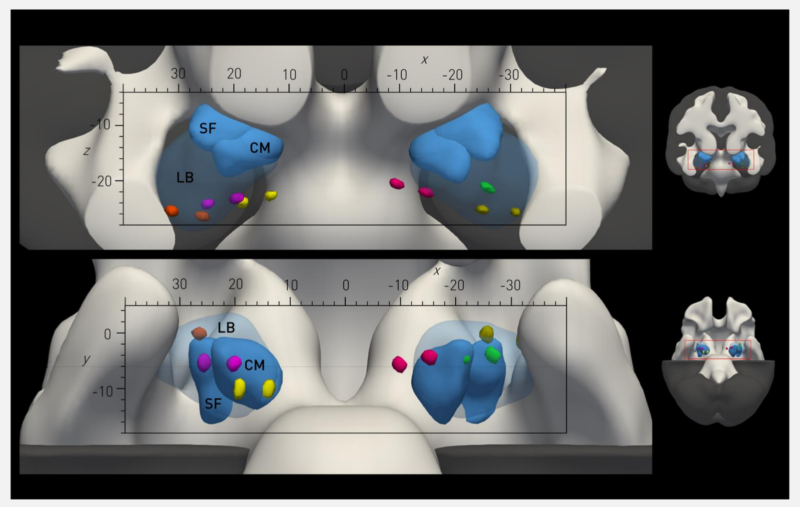
The underlying neurocircuitry is proposed to be parallel (fast pathways via the amygdala, slower pathways via the cortex) and not easily interrogated with correlative methods (fMRI, EEG). Studies of humans with chronic amygdala lesions are potentially confounded by chronic neural and behavioural compensation. In order to overcome the limitations of previous studies we:

- Applied transient focal unilateral electrical disruption of the human amygdala allowing within-subject comparison on a timescale too short for confounding neural plasticity to occur .
- Developed a behavioural paradigm that parameterised instinctive gaze shifts towards fearful faces as a function of saccadic latency (acquired by 1kHz eye-tracking)
- Developed variants of the paradigm to dissociate face detection (sensory) and orientation (motor) roles by dissociating location of face from direction of orientation.

## 3. Method

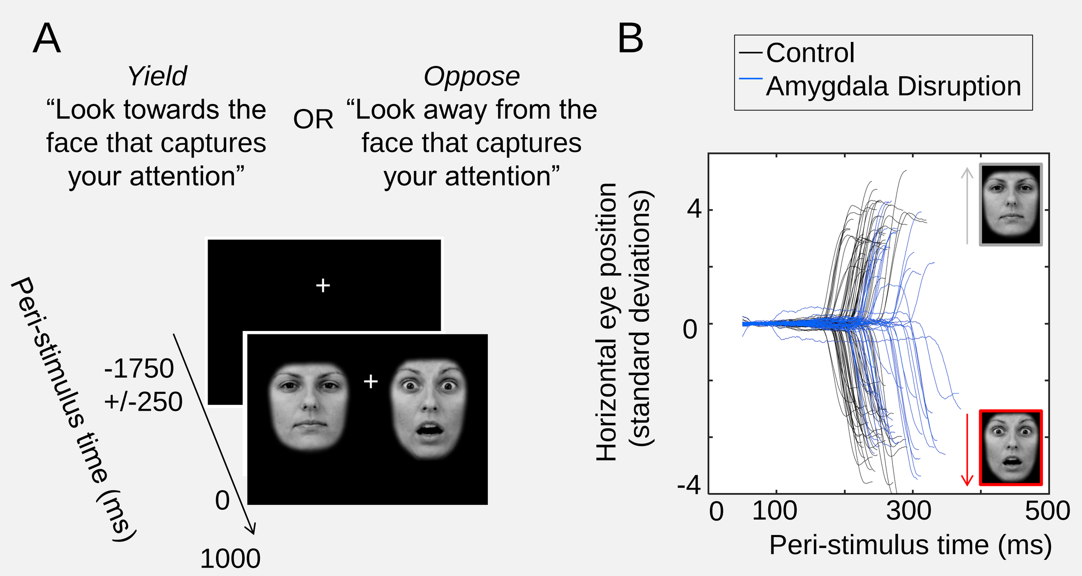
### 3.1 Cohort undergoing amygdala disruption

Six patients with unilateral intracranial amygdala electrodes temporarily implanted for the clinical evaluation of focal epilepsy performed a behavioural task.




***Electrical Disruption.** Behaviourally disruptive trains of 50Hz direct electrical stimulation were administered via bipolar electrodes located within the laterobasal (LB) amygdala. Each patient represented by a different colour. Superficial (SF) and centeromedial (CM) nuclei amygdala subnuclei also shown.*

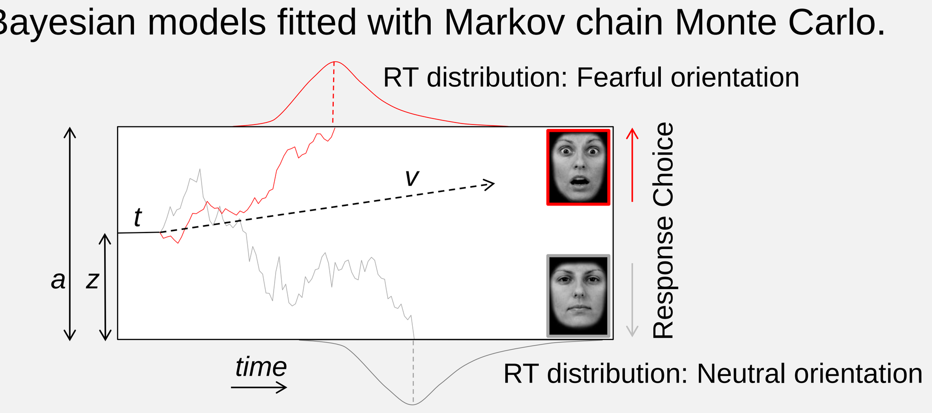
### 3.2 Behaviour task




***The task.** We designed a paradigm to (**A**) capture instinctive orientations to fearful faces, with variants to dissociate the location of fearful face presentation (sensory) from the direction of the orientation response (motor). We recorded eye-movements (**B**) with and without focal, transient, unilateral direct electrical disruption of the amygdala.*

### 3.3 Behavioural Analysis

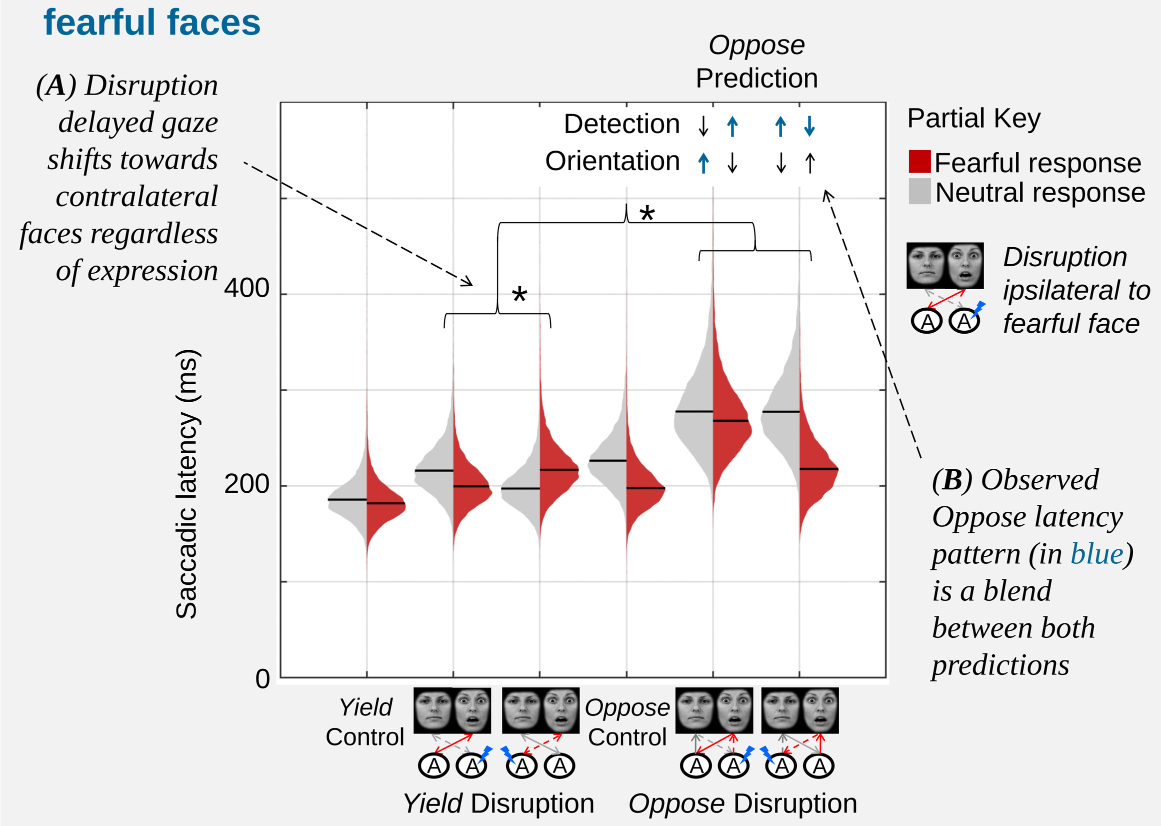
Response Choice (neutral, fearful) and saccadic latency were modelled individually and jointly (within a Drift-diffusion model) as a function of Instruction (Yield, Oppose) and Disruption (none, ipsilateral to fearful face, contralateral to fearful face) within multi-level Bayesian models fitted with Markov chain Monte Carlo.




***The Drift-diffusion model of action selection.** This conceives orientation choice as the outcome of a latent neural competition parameterised by prior preference (z, bias), the rate of evidence accumulation (v, drift-rate), response caution (a, decision threshold) and non-decision processes (t, non-decision time).*

## 4. Results

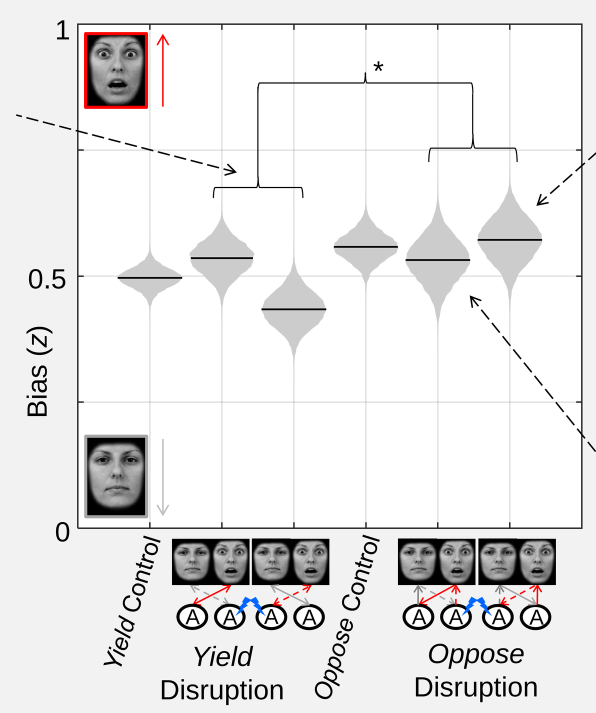
### 4.1 The amygdala mediates rapid orientation to contralateral fearful faces




***Saccadic latency over 1064 trials.** Disruption delayed instinctive gaze shifts to the contralateral hemifield(**A**) establishing the functional lateralisation of the human amygdala. Observed latencies in the Oppose condition were mixed (**B**) suggesting that the amygdala modulates detection of all contralateral faces, but facilitates rapid automatic orienting specifically towards fearful ones. Arrows in abscissa: show direction of proposed detection (face to amygdala) and orientation (amygdala to face) involving fearful (red) and neutral (grey) faces; dashed = interrupted.*

### 4.2 Within a drift-diffusion model, this role is best explained as an orientation bias towards the fearful face

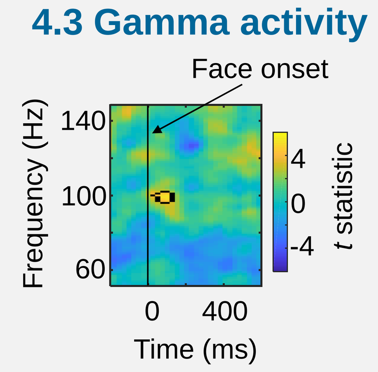
***Drift-diffusion bias.** Disruption of the amygdala biases instinctive gaze shifts away from the contralateral hemifield in the Yield condition. The Oppose condition reverses this in a salience-specific manner*




*When a fearful face is contralateral the intact amygdala mediates early detection only of a neutral face and subsequent gaze shifts away (↑bias to fearful face).*

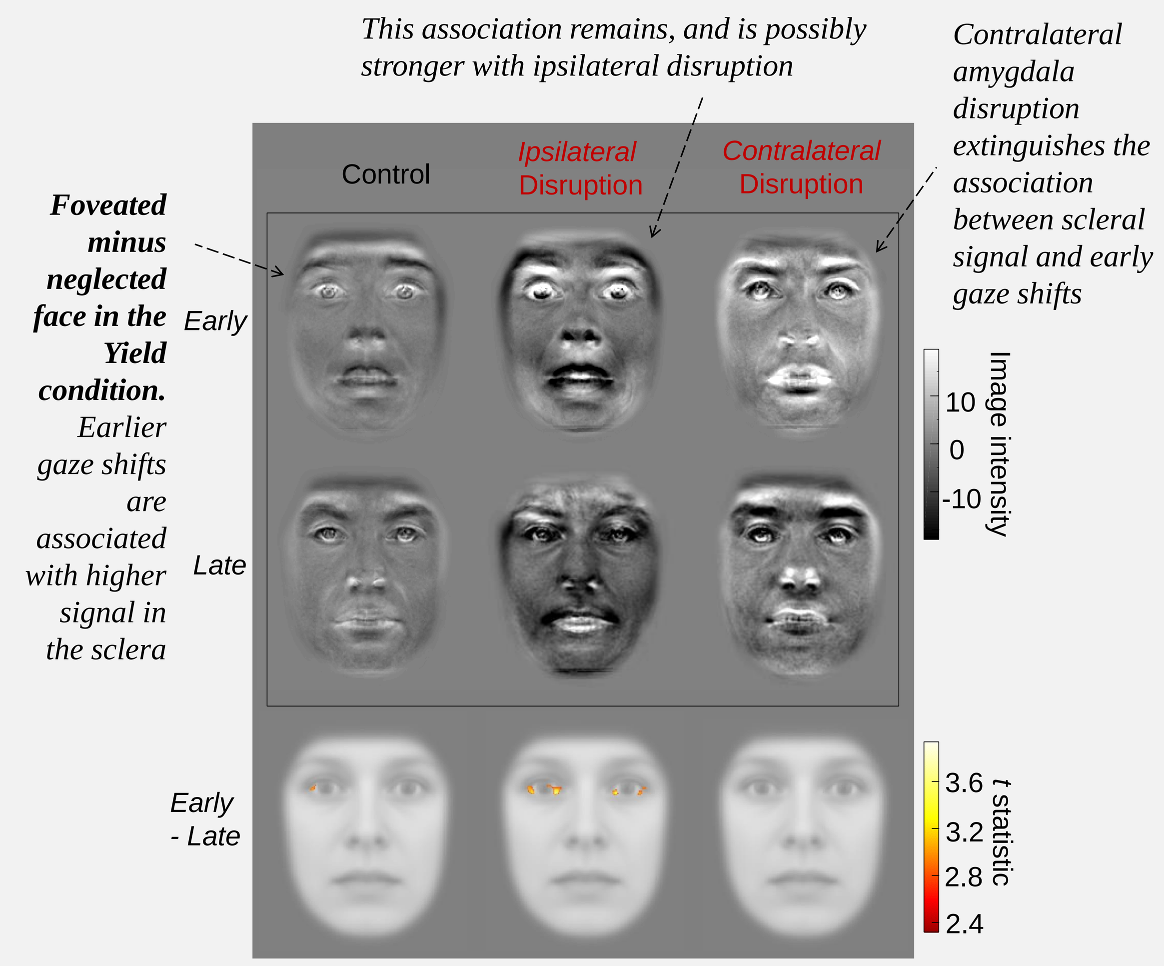
*When a neutral face is contralateral the intact amygdala mediates automatic orienting towards the fearful face so gaze shifts away towards the neutral face are delayed (↑ bias to fearful face).*

### 4.3 Gamma activity predicts orientation to a contralateral face




***Amygdala local field potentials (Yield condition only).** High gamma oscillatory activity 29-131ms after face onset preceded orientation to the contralateral hemifield (relative to orientation to the ipsilateral one) regardless of facial expression. This is a potential analogue of the prior bias to orient contralaterally*

### 4.4 Pixel-wise analysis of target facial morphology revealed scleral exposure as its primary driver




## Conclusion

The amygdala is here re-conceptualised as a functionally lateralised instrument of early action, reconciling previous conflicting accounts confined to face detection. Here, we demonstrate:

- The amygdala targets faces in the contralateral hemifield establishing its functional lateralisation.
- The amygdala mediates detection of faces and automatic orientation towards fearful ones.

We reveal an underlying neural organisation analogous to the superior colliculus. Greater clarity on its role has the potential to guide therapeutic resection, and inform novel focal stimulation techniques for the management of neuropsychiatric conditions.

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