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The role of the basolateral amygdala in gaze avoidance behaviour

Background

Our behaviours arise from the coordinated activity of interconnected networks of neurons in the brain. A major challenge of neuroscience is unravelling this connectivity between networks. However, with more than 10^{14} anatomical connections, across different spatial scales in the brain and dynamically changing during behaviour, this is not an easy task!

One approach to this problem is from the perspective of circuit theory. That is, let us assume the connections in the brain follow the pattern of a circuit – sensory inputs are connected to central processing functions which are able to activate muscle effectors to generate behaviour. We can then explore the various ‘components’ to understand their function within the brain and its circuits. Unlike a true circuit, however, isolating these circuit components has itself been a challenge – methods to activate/inactivate them have previously been invasive and non-specific.

Recently, some of the techniques have undergone considerable refinement - optogenetics is one example. It is a biological technique that uses light to influence, control and monitor neurons, which have been genetically modified for light-sensitivity. This is a very precise technique that enables us to monitor right down to individual neurons in real time, even in freely-moving animal models.

The Problem

We are researching the neural circuits involved in human social interactions and how they are affected during mental disease, in particular the circuits involved in the pattern of eye movements (known as gaze behaviour) in social settings.

Making eye contact is crucial for human interactions – parents connect socially with their babies by looking at them and it is foundational for the behavioural development that follows, such as learning by imitation. Gaze avoidance in early years usually leads to developmental disabilities and isolation, as seen in most children with Autism Spectrum Disorder (ASD) and other psychiatric disorders like Schizophrenia. If we understood the mechanisms underlying gaze avoidance, we might find new strategies for helping patients.

One hypothesis is that neurons in the amygdala (a part of the brain that processes memory, decision-making and emotional responses) are responsible for motivating us to make eye contact. If that’s the case, we might expect inhibition of those neurons to produce gaze avoidance, but this has not yet been tested.

The Project

We will conduct experiments to test this hypothesis. We will also undertake measurements of the gaze behaviour in patients with ASD and Schizophrenia to validate the behaviour that results from the neuron inhibition experiments. We will use a combination of behavioural measurements, electrophysiology and optogenetics to reverse engineer brain circuits involved in gaze avoidance and provide causal evidence in favor of this hypothesis. We will probe specific components of brain circuits involved in gaze orienting, face recognition, social interactions and autonomic response that lead to control of emotions during social behaviour. Using this approach will help to pinpoint specific deficits in circuit components function observed in diseases such as Schizophrenia and Autism Spectrum Disorder. Finally, our circuit approach will potentially lead to innovative interventions to treat core symptoms of mental disease using technologies such as deep brain stimulation and local drug delivery.

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