The neurological basis for fear and memory

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Fear conditioning using sound and taste aversion, as applied to mice, have revealed interesting information on the basis of memory allocation.

European 'Cellular mechanisms underlying formation of the fear memory trace in the mouse amygdala' (FEAR Memory TRACE) project is investigating memory allocation and the recruitment of certain neurons to encode a memory. By studying conditioned fear memory in response to an auditory stimulus, the researchers have delved into pathological emotional states and neural mechanisms involved in memory allocation, retrieval and extinction.

Prior research has revealed that the conditioned fear response in mice is located in a specific bundle of neurons in the amygdala. Memory allocation modulation is due to expression of the transcription factor, cyclic adenosine 3', 5'-monophosphate response element binding protein (CREB) and possibly neuronal excitability.

FEAR Memory TRACE focused on the electrophysiological properties of neurons encoding the same memory. The project also aimed to ascertain the biophysical mechanisms in the plasticity changes recorded in the specific set of neurons in the fear memory trace.

Recording information on auditory fear conditioning and conditioned taste aversion, the scientists used intra-amygdala surgery using viral vectors and electrophysiological experiments to detect neuronal excitability.

Transfected by virus, CREB tagged with green fluorescent protein together with the gene for channelrhodopsin2 were used in neural control experiments. Combined, these two elements caused neuron firing in specific nerve cells. Molecular techniques included western blot for protein detection, genotyping and viral DNA preparation.

Behavioural tests on long- and short-term memory of mice involving fear conditioning and taste aversion showed increased memory performance at the three-hour point rather than the five-hour point. The intrinsic excitability of the mice receiving both shock and the tone was increased at three hours, not five, compared to mice that only received the tone.

As the project continues to its close in two years, the aim is to identify biophysical mechanisms involved in recruiting neurons that compete with each other for a specific memory. FEAR Memory TRACE will also develop computational models to assess the role of these mechanisms in memory performance.

Information on biochemical processes in neural mechanisms has wide application in many clinical situations including patients suffering memory loss, such as stroke victims. Fear response manipulation can be applied in treatment of neuroses and phobias.

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