Genes and brain plasticity

Can we change how experiences are encoded and thereby modify our behavior?

by Ami Citri
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William James, the famous physician-scientist, once commented that since children grow into “walking bundles of habits,” they should be very careful which habits they acquire.

As we go about our lives, our brain continuously records interactions with the surrounding world, in the process creating behavioral patterns that can become deeply ingrained and semi-automatic. Sometimes these patterns may even develop into phobias and restricted behaviors that limit our interaction with the world. This, I believe, is what William James was warning us about.

For decades, neuroscientists have been investigating the brain’s mechanisms for encoding information. They have made tremendous progress in understanding how memories are formed, and have deepened our understanding of the development of behavioral patterns. The hope is that once we understand the codes used by the brain, we can modify them to correct situations in which this plasticity has gone awry and forms patterns that are harmful to us.

Recent experiments in my lab have led to the surprising observation that there is a “genetic code” for experiences in the brain. We have found that all important experiences (the ones we will remember tomorrow and next week, and perhaps even next year) have a unique fingerprint in the patterns of the genes that are turned on as the experience is encoded in the brain.

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The identification of this gene expression code may allow us to correct the encoding of experiences that define how we interact with the world around us.

In our current research on mice, we are finding that by manipulating the gene expression code for an aversive memory in the brain, we can cause mice to change their strategies for coping with an aversive experience. We achieve this by manipulating the activity of a single gene (Egr2) in the amygdala, a region of the brain that is associated with the processing of aversive experiences.
Egr2 is the gene we have found to be most prominently affected by negative experiences within the amygdala of mice, providing a “hallmark” of aversive experience. We hypothesize that the expression of Egr2 is induced in the amygdala to mediate aspects of the encoding of aversive experiences, defining future behavior in response to similar circumstances.

We made use of special viruses to manipulate the activity of Egr2 in the amygdala prior to exposing the mice to high doses of lithium chloride, a compound that causes malaise and nausea and forms a potent memory of aversion to the context in which the compound was applied.

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Mice naturally respond to aversive experiences by means of a passive coping mechanism that leads them to freeze when confronted with a fear-inducing or aversive experience. Our manipulation of Egr2 expression caused their behavior to shift from freezing to active coping; they then responded by fleeing when confronted with a context associated with an aversive experience, actively avoiding that situation.

We interpret these results to mean that the expression of Egr2 is induced by aversive experiences to enhance the expression of passive coping mechanisms (which might be evolutionarily advantageous to mice under certain circumstances). When the expression of the gene is disrupted, we therefore observe a transition to active coping mechanisms.

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We believe that similar mechanisms are found in humans, supporting the development of habitual, semi-automatic behavioral patterns. Consider the last time someone behaved aggressively towards you, for example by cutting you off on the road. Perhaps your automatic response was to become angry and behave in a similarly aggressive way; in any event, your response is a product of the past encoding of similar situations, which likely utilized patterns of gene expression very similar to those we observe in mice.

In the future, it may be possible to tease apart the building blocks that encode a traumatic memory in the brain, helping people overcome deeply ingrained anxieties and phobias.

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