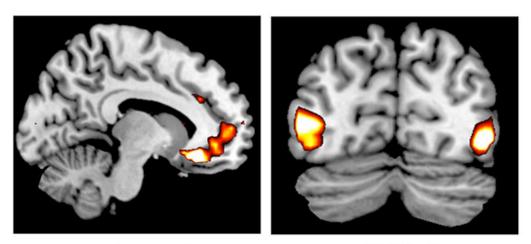


How your brain reacts to emotional information is influenced by your genes

May 7 2015



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Image shows increased activity in the brains of ADRA2b deletion carriers.

Your genes may influence how sensitive you are to emotional information, according to new research by a UBC neuroscientist. The study, recently published in The *Journal of Neuroscience*, found that carriers of a certain genetic variation perceived positive and negative images more vividly, and had heightened activity in certain brain regions.

"People really do see the world differently," says lead author Rebecca



Todd, a professor in UBC's Department of Psychology. "For people with this <u>gene variation</u>, the emotionally relevant things in the world stand out much more."

The gene in question is ADRA2b, which influences the neurotransmitter norepinephrine. Previous research by Todd found that carriers of a deletion variant of this gene showed greater attention to negative words. Her latest research is the first to use brain imaging to find out how the gene affects how vividly people perceive the world around them, and the results were startling, even to Todd.

"We thought, from our previous research, that people with the deletion variant would probably show this emotionally enhanced vividness, and they did more than we would even have predicted," says Todd, who scanned the brains of 39 participants, 21 of whom were carriers of the <u>genetic variation</u>.

Carriers of the gene variation showed significantly more activity in a region of the brain responsible for regulating emotions and evaluating both pleasure and threat. Todd believes this may help explain why some people are more susceptible to PTSD and intrusive memories following trauma.

"Emotions are not only about how feel about the world, but how our brains influence our perception of it," says Adam Anderson, professor of human development at Cornell University and senior author of the study. "As our <u>genes</u> influence how we literally see the positive and negative aspects of our world more clearly, we may come to believe the world has more rewards or threats."

Todd points out there are also benefits to carrying the gene variant. "People who have the deletion variant are drawing on an additional network in their brains important for calculating the emotional relevance



of things in the world," she says. "In any situation where noticing what's relevant in the environment is important, this gene variation would be a positive."

Todd says a prime example of a carrier of this variation was French novelist Marcel Proust: "He bit into the Madeleine cookie and then wrote seven volumes of memoirs," she says. "He probably was emotionally sensitive too and he was certainly creative. He's a classic deletion carrier.

Background

About the study:

Study participants were asked to estimate the amount of "noise", or pixelation, applied to images that had either positive, negative or neutral emotional content.

Compared to non-carriers, carriers of the ADRA2b deletion variant gene estimated lower levels of noise on positive and negative images, relative to neutral images, indicating emotionally enhanced vividness, or EEV.

Carriers of the deletion variation also showed significantly more brain activity reflecting EEV in key regions of the brain sensitive to emotional relevance.

About the gene:

The ADRA2b deletion variant appears in varying degrees across different ethnicities. Although roughly 50 per cent of the Caucasian population studied by these researchers in Canada carry the genetic variation, it has been found to be prevalent in other ethnicities. For



example, one study found that just 10 per cent of Rwandans carried the ADRA2b gene variant.

Further research is planned to explore emotionally enhanced vividness, or EEV in other ethnic groups, and how ADRA2b influences emotional associations related to anxiety, post-traumatic stress disorder and addiction.

More information: "Neurogenetic Variations in Norepinephrine Availability Enhance Perceptual Vividness" *Journal of Neuroscience*, 22 April 2015, 35(16): 6506-6516; <u>DOI:</u> <u>10.1523/JNEUROSCI.4489-14.2015</u>

Provided by University of British Columbia

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