Are you genetically predisposed to antisocial behavior?

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Both positive and negative experiences influence how genetic variants affect the brain and thereby behaviour, according to a new study. "Evidence is accumulating to show that the effects of variants of many genes that are common in the population depend on environmental factors. Further, these genetic variants affect each other," explained Sheilagh Hodgins of the University of Montreal and its affiliated Institut

Universitaire en Santé Mentale de Montréal. "We conducted a study to determine whether juvenile offending was associated with interactions between three common genetic variants and positive and negative experiences." Hodgins and her colleagues published the study on December 11, 2014 in the *International Journal of Neuropsychopharmacology*.

Every single high school student aged 17 to 18 years old in Västmanland, a Swedish county, was invited to participate in the study, and 1,337 agreed to do so. They anonymously completed questionnaires reporting on delinquency, family conflict, experiences of sexual abuse, and the quality of their relationship with their parents. They also provided a sample of saliva from which the researchers extracted DNA.

The Monoamine oxidase A (MAOA) gene is a key enzyme in the catabolism of brain neurotransmitters, monoamines, especially serotonin. Catabolism is the breaking down of complex materials and the releasing of energy within an organism. "About 25% of Caucasian men carry the less active variant of MAOA. Among them, those who experience physical abuse in childhood are more likely than those who are not abused to display serious antisocial behaviour from childhood through adulthood," Hodgins explained. "Among females it is the high activity variant of the MAOA gene that interacts with adversity in childhood to increase the likelihood of antisocial behaviour."

The brain-derived neurotrophic factor (BDNF) gene modulates neuronal plasticity. The term neuronal plasticity refers to our brain cells' ability to reorganize pathways and connections throughout our lives. "The low expressing variants of BDNF are carried by approximately 30% of individuals and some previous studies had shown that this variant was associated with aggressive behaviour if carriers were exposed to aggressive peers. The third gene we studied was the serotonin transporter 5-HTTLPR," Hodgins said. "The low activity variant of this gene is

carried by approximately 20% of individuals. Among carriers of this low activity variant, those exposed to adversity in childhood are more likely than those who are not to display antisocial and aggressive behaviour."

"We found that the three genetic variants interacted with each other and with family conflict and sexual abuse to increase the likelihood of delinquency, and with a positive parent-child relationship to decrease the risk of delinquency," Hodgins explained. "Among carriers of the low activity variants of all three genes, those exposed to family conflict or sexual abuse or both reported high levels of delinquency while those who reported a positive and warm relationship with their parents reported little or no delinquency." Thus, the same genetic variants were associated with high and low levels of delinquency depending on exposure to negative or positive environments.

In conclusion, variants of three common genes, MAOA, BDNF, and 5-HTTLPR, interacted with each other and with negative environmental factors to increase the risk of delinquency and with a positive environmental factor to decrease the risk of delinquency in a large sample of teenagers. "These findings add to those from other studies to show that genes affect the brain, and thereby behaviour, by altering sensitivity to the environment," Hodgins said.

More information: Kent W Nilsson, Erika Comasco, Sheilagh Hodgins, Lars Oreland, Cecilia Åslund published "Genotypes do not confer risk for delinquency but rather alter susceptibility to positive and negative environmental factors: Gene-environment interactions of BDNF Val66Met, 5-HTTLPR, and MAOA-uVNTR" in the *International Journal of Neuropsychopharmacology* on December 11, 2014.

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