

Rwandan genocide survivors provide new insights into resilience and PTSD

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The 1994 genocide in Rwanda resulted in the mass killing of up to one million people over the course of about 100 days. Although the exact death toll is unknown, experts estimate that as much as 20% of the country's entire population was murdered. There can be no doubt or surprise then that some of the survivors developed posttraumatic stress disorder, or PTSD, an anxiety disorder that can develop after witnessing or experiencing a traumatizing event, such as abuse, war, or natural disaster.

However, even under stress as extreme as genocide, not all individuals develop PTSD. Why is it that some do and some don't? A new study published in [Biological Psychiatry](#), by Elsevier was designed to address that question.

The clues may come from molecular genetics. Scientists already know that, in general, the more traumatic events a person experiences, called 'traumatic load', the higher their likelihood of developing PTSD. In this new study, Iris-Tatjana Kolassa and her colleagues show that genetic factors influence this relationship.

They studied 424 Rwandan Genocide survivors, some with and some without PTSD. As expected, they found that those survivors with higher traumatic load had a higher prevalence of lifetime PTSD, a dose-response relationship. But importantly, they also found that the Val158Met polymorphism of the gene encoding the enzyme catechol-O-methyltransferase (COMT) plays a role in this relationship.

Individuals homozygous for the Met allele of this COMT [polymorphism](#) have substantially lower activity of this enzyme. Lower COMT activity would be expected to produce higher levels of [norepinephrine](#) and [dopamine](#), neurotransmitters that are released during stress. Rwandan survivors with at least one Val allele in this gene showed the typical dose-response relationship between trauma

severity and PTSD risk, but those homozygous for the Met allele exhibited a high risk for PTSD independently of the severity of traumatic load.

In other words, people who, due to their genotype, were more likely to inactivate the stress neurotransmitters were somewhat protected from developing stress-related problems relative to people who were less able to metabolize the transmitters.

Dr. John Krystal, Editor of *Biological Psychiatry* commented: "We hope that [molecular genetics](#) will help us to identify those who are most resilient so that we can learn about ways that people cope with stress at a psychological, behavioral, and biological level. We also would like a biological test to help us to identify people who are most vulnerable to the negative effects of stress so that we could target supportive services to these people." This study is another step in that direction.

Dr. Iris-Tatjana Kolassa cautions that many technical and clinical questions remain open in attempting to develop molecular genetic tests that predict patterns of stress response. But she acknowledged that human genetics could someday play a role in the prevention and treatment of PTSD.

Provided by Elsevier

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