Association study of trauma load and SLC6A4 promoter polymorphism in posttraumatic stress disorder : evidence from survivors of the Rwandan genocide

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As exposure to different types of traumatic stressors increases, the occurrence of posttraumatic stress disorder (PTSD) increases. However, because some people exhibit either surprising resilience or high vulnerability, further influencing factors have been conjectured, such as gene-environment interactions. The SLC6A4 gene, which encodes serotonin transporter, has been identified as predisposing toward differential emotional processing between genotypes of its promoter polymorphism.; We investigated 408 refugees from the Rwandan genocide and assessed lifetime exposure to traumatic events, PTSD (according to DSM-IV) status, and genotype of the SLC6A4 promoter polymorphism. The study was conducted from March 2006 to February 2007.; The prevalence of PTSD approached 100% when traumatic exposure reached extreme levels. However, persons homozygous for the short allele of the SLC6A4 promoter polymorphism showed no dose-response relationship but were at high risk for developing PTSD after very few traumatic events. This genotype influence vanished with increasing exposure to traumatic stressors.; We find evidence for a gene-environment interplay for PTSD and show that genetic influences lose importance when environmental factors cause an extremely high trauma burden to an individual. In the future, it may be important to determine whether the effectiveness of therapeutic interventions in PTSD is also modulated by the SLC6A4 genotype.
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