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Research report

Children's risk and resilience following a natural disaster: Genetic vulnerability, posttraumatic stress, and depression

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ABSTRACT

Objective: We examined children's risk and resilience following a natural disaster, evaluating the role of stress, social support, and two genetic markers: the short allele of the serotonin transporter gene (5-HTTLPR), and the met allele of the Brain-Derived Neurotrophic Factor (BDNF).Under high levels of hurricane exposure or hurricane-related stressors, we expected children displaying the markers would report greater symptoms of posttraumatic stress disorder (PTSD) and depression than children without these markers. Social support was explored as an additional moderating variable.

Method: Eight months after Hurricane Ike, 116 children (*M* age=8.85 years, *SD*=.89; 54% girls) residing in Galveston, Texas, provided saliva samples and completed measures of hurricane exposure and stress, and symptoms of PTSD and depression; 80 also completed a social support measure.

Results: For BDNF, analyses revealed several Gene by Environment interactions; greater stress was related to more symptoms of PTSD and depression, and this effect was stronger for children with the met allele. No findings emerged for 5-HTTLPR. Stressors and social support also were associated with children's PTSD and depressive symptoms.

Limitations: Findings should be tempered by the relatively small sample, especially for analysis that included social support.

Conclusions: The met allele (BDNF) may play a role in children's disaster reactions. Further research should consider the complex interplay between genes, stressors, support, and psychological outcomes over time.

1. Introduction

Disasters occur worldwide, affecting over 66 million children annually (Pronczuk and Surdu, 2008). Both population growth and climate change are expected to contribute to an increased prevalence and severity of disasters in the future, with an anticipated 175 million children to be affected annually by disasters over the next decade (Borenstein, 2011; Seballos et al., 2011). Further, children appear to be a psychologically vulnerable population in the aftermath of disasters (Bonanno et al., 2010), likely due to the disruption and deterioration of key support systems postdisaster (Norris and Kaniasty, 1996).

Natural disasters are especially important for understanding the contributions of traumatic stress to the development of psychopathology in children. Disasters affect individuals in a given geographic area without consideration of their prior psychological functioning (La Greca and Silverman, 2012). Many other stressors (e.g., divorce, abuse) occur disproportionately to individuals with psychological difficulties (e.g., Davies et al., 1997) making it difficult to directly evaluate the effects of stressors on psychological functioning.

In September 2008, Hurricane Ike struck the Texas coastline, causing widespread damage and life disruption in Galveston, where 75% of all homes sustained damage or were destroyed. Ike was a strong Category 2 hurricane, causing $25 billion in damages and taking 103 lives (Berg, 2008). This event provided an opportunity to evaluate factors that predict children's risk and resilience in the aftermath of natural disasters.

Specifically, in the context of Hurricane Ike, we evaluated the role of genetic markers as potential risk factors for children's postdisaster internalized distress. To our knowledge, no studies have examined this issue in youth. However, recent studies with adults indicate that certain genetic markers, in conjunction with disaster exposure, may be implicated in the expression of posttraumatic stress disorder (PTSD) and depressive symptoms among disaster-exposed adults (Kilpatrick et al., 2007). Accumulating

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evidence on children also reveals that symptoms of PTSD and depression are common in the aftermath of natural disasters, including hurricanes (Goenjian et al., 2001; La Greca et al., 2010, 2013). Further, children's psychological reactions are strongly influenced by their disaster exposure and by stressors occurring during the recovery period (La Greca, et al., 1996, 2010; Yell and et al., 2010). Thus, the current study evaluated whether genetic factors, interacting with high levels of hurricane exposure and stress, might also play a role in children's postdisaster PTSD and depressive symptoms.

Two genetic markers implicated in the development of mood and/or anxiety disorders were evaluated in this study. One was the short (s) allele of the serotonin transporter gene, 5-HTTLPR. This marker, in interaction with environmental adversity, has been linked to a variety of psychological outcomes and especially to affective disorders and PTSD (Caspi et al., 2003, 2010; Goenjian et al., 2012). For example, Kilpatrick et al. (2007) found that the presence of the s allele increased adults' risk for PTSD and major depression under the conditions of high hurricane exposure and low social support. Similarly, the presence of the s allele of the 5-HTTLPR polymorphism has been shown to moderate the relationship between life stress and depression among adolescent girls (Gotlib et al., 2008). Thus, we expected that the presence of the s allele of the 5-HTTLPR polymorphism would be associated with symptoms of PTSD and depression among children reporting high levels of disaster exposure or other disaster-related stressors. Based on Kilpatrick et al. (2007), we additionally explored whether children's levels of social support played a role as a moderating variable. Specifically, in a subset of our sample, we examined the three-way interaction between environmental factors (hurricane-related stressors, social support) and 5-HTTLPR, expecting that children with high stress, low support, and the presence of the s allele would be the ones most vulnerable to experiencing high levels of PTSD and depressive symptoms postdisaster.

These same gene by environment (G × E) interactions also were examined using a second genetic marker, the met allele (on the Val66Met polymorphism) of the Brain-Derived Neurotrophic Factor (BDNF). Similar to work on 5-HTTLPR, evidence indicates that the presence of the met allele of the BDNF gene modulates the effect of adversity on adult depression (e.g., Aguilera et al., 2009). Thus, we examined the same G × E interactions (described above), using the presence of the met allele as our genetic marker.

In order to evaluate G × E interactions, we assessed children's exposure to hurricane-related stressors in several ways. First, we examined hurricane exposure, as assessed by children's *perceived life threat* during the hurricane. This measure of exposure appears to be the best predictor of children's disaster-related PTSD reactions (Goenjian et al., 2001; La Greca et al., 2010; see Furr et al. 2010). Next, we evaluated G × E interactions with hurricane-related stressors occurring during the postdisaster recovery period, specifically immediate and ongoing *hurricane-related loss and disruption*; prior studies indicate that such stressors are important contributors to children's PTSD reactions (La Greca et al., 1996, 2010; Vernberg et al., 1996) and depressive symptoms (Lai et al., 2013; Pina et al., 2008; Scheeringa and Zeanah, 2008).

Finally, this study provided an opportunity to replicate and extend earlier findings on risk and resilience factors that play a role in children's postdisaster distress. Based on a conceptual model of risk and resilience (see La Greca et al., 1996), studies of Hurricanes Andrew, Charley, and Katrina have found that girls, minority youth, and children experiencing more stressors typically report more PTSD symptoms postdisasters (Kronenberg et al., 2010; La Greca et al., 1996, 2010; Weems et al., 2010). In contrast, social support appears to be a resilience factor, contributing to fewer PTSD symptoms among children exposed to traumatic events (Kelley et al., 2010;La Greca et al., 1996; Llabre and Hadi, 1997). The present study attempted to replicate these findings on risk and resilience, and extend them to a second indicator of children's distress: symptoms of depression.

2. Method

2.1. Participants

Participants were 116 children (M age = 8.85 years, SD = .89; 54% girls; 38% White; 31% Hispanic, 22% Black, 11% Asian/Other), who represented 35% of the full sample of children who participated in our Time 1 assessment of children's reactions to Hurricane Ike. Analyses compared the 116 children with saliva samples for DNA analyses with the 212 who did not participate in genetic testing. Findings revealed no differences between the groups with respect to age, gender, perceived life threat, stressors, or symptoms of PTSD and depression. However, non-Hispanic White children were overrepresented (38%) among children who participated in genetic testing compared to those who did not participate (22%), $\chi^2 = 12.90$, $p < .01$. Also, children who participated in genetic testing reported greater overall social support (M = 3.33, SD = .48; t (199) = −3.57, $p < .001$) compared to those not participating (M = 3.06, SD = .57). These differences should be kept in mind when interpreting the study results. The final sample for genetic analyses was n = 115, as laboratory errors resulted in missing data for one child.

2.2. Procedures

The study protocol was reviewed and approved by the Internal Review Boards for the University of Miami, the University of Texas Medical Branch, and the Galveston Independent School District (GISD). Active informed parental consent in parents' preferred language (i.e., English or Spanish) was obtained for all participants. Via homeroom teachers, parental consent forms and letters describing the study were distributed to all second through fourth graders enrolled in the GISD in April of 2009. Approximately 35% returned consent forms with permission to participate. Written child assent was also obtained for all children prior to participation. At the time of the survey testing (May 2009), a second letter describing the ancillary study for genetic testing and a parental consent form were distributed to children who were enrolled in the overall study; children were asked to return the forms within a week. We received 67 parental consents for child participation; these children provided assent to participate and their saliva samples were collected for genetic testing in May 2009. One saliva sample spilled and was not able to be analyzed, yielding 66 useable samples. In December 2009, parents were re-contacted to request permission for their child's participation in genetic testing. This resulted in 50 additional child participants.

Children's saliva samples were collected using Oragene DNA collection kits (http://www.dnagenotek.com/DNA_Genotek_Product_Oragene_DNA_A_Overview.html). Children spit a small saliva sample into a clear plastic tube. The tubes were sealed tightly, shaken gently, placed into individual zip-lock bags, and transported to the Biorepository and Labs at the Institute for Human Genomics at the University of Miami for analysis.

The child report measures described below were obtained in May 2009 and were used in current study analyses. All survey data were collected in the schools, during group sessions of 25–40 students. Six to eight research assistants and two study investigators were present at all times to oversee testing and assist children needing individual help. All questions were read aloud to the children as they followed along and marked their answers. Individual