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Original article

Family Economic Hardship, Corticotropin-Releasing Hormone Receptor Polymorphisms, and Depressive Symptoms in Rural African-American Youths

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A B S T R A C T

Purpose: The purpose of this study was to use pooled data from two independent studies of rural African-American youths to test the moderation effect of the corticotropin-releasing hormone receptor 1 gene (*CRHR1*) on the link between family economic hardship and trajectories of depressive symptoms.
Methods: Two longitudinal studies were conducted involving African-Americans, aged 16 (N = 474) and 18 (N = 419) years, who were randomly recruited in rural Georgia. Family economic hardship and youths' depressive symptoms were assessed four times across 2.5 years. Genetic data also were collected. Haplotype analysis was performed on single-nucleotide polymorphisms of *CRHR1*; two haplotypes were aggregated to form a *CRHR1* index. Growth curve models were executed to determine whether *CRHR1* moderated the link between Wave 1 family economic hardship and youths' development of depression.
Results: *CRHR1* × family economic hardship interactions significantly predicted youths' depressive symptoms. When exposed to family economic hardship 1 standard deviation above the mean at Wave 1, youths who scored 0 on the *CRHR1* index showed high and increasing depressive symptoms across time, whereas those who scored 2 on the index showed a decrease in depressive symptoms.
Conclusions: The *CRHR1* gene reduces the risk for depressive symptoms among youths living in families undergoing high levels of economic hardship.
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IMPLICATIONS AND CONTRIBUTION

The present study extends the literature on the buffering effects of *CRHR1* on the association between family economic hardship and trajectories of depressive symptoms among rural African-American youths. Finding a protective role for *CRHR1* in the presence of family economic hardship contributes to genetically informed resilience research.

African-American adolescents and young adults have historically lived with high and chronic levels of economic hardship, a situation that continues today. A consistent result is a heightened risk for mental health impairments, particularly elevated depressive symptoms [1,2]. Contextual pathways from economic hardship to the development of depressive symptoms have been identified among African-American youths. A "parental" pathway chronicles the ways in which the stressors associated with coping with chronic economic hardship induce depressive symptoms in African-American youths' caregivers. These symptoms spill over to compromise the quality of parental investment and nurturance the children receive. Depression associated declines in nurturant-involved caregiving and increases in harsh-inconsistent parenting presage the development of depressive symptoms among African-American youths [3–5]. Outside the

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family, youths living with economic hardship are likely to encounter crime, violence, and drug use, along with a lack of safe recreational areas. Such "neighborhood effects" contribute uniquely to the development of depressive symptoms [6–8]. Although economic hardship heightens the risk for elevated depressive symptoms, not all youths who experience hardship develop symptoms [9]. Recent studies have shown buffering effects of social support [10] and racial identity [1]; however, studies have not examined the possibility that genetic variation may moderate the impact of economic hardship on the development of depressive symptoms among African-American youths. In this study, we examined the moderational role of the corticotropin-releasing hormone receptor 1 gene (*CRHR1*) on the longitudinal association between economic hardship and the development of depressive symptoms.

Research on physiological responses to stress using both animal and human models provides a foundation for understanding individual variation in depressive symptoms in response to acute and chronic stress [11–14]. One potential biological process involves overactivation of the hypothalamic–pituitary–adrenal (HPA) axis [15,16]. Genes associated with regulating HPA activity have been examined for a role in modulating reactivity to stressful environments. An accumulating body of evidence points to the involvement of the receptor for *CRHR1* in this process. In a typical stress response, elevations of cortisol occasioned by CRH on mineralocorticoid and glucocorticoid receptors in the hippocampus, paraventricular nucleus, and pituitary reduce activation of the HPA axis and stabilize glucocorticoid, creating a negative feedback system that modulates the stress response. Repeated activation of the HPA axis in response to chronic or recurring stress can compromise its functioning, as evidenced by a protracted cortisol response to a stressful event or, alternatively, no cortisol response at all [17]. Alterations in HPA axis functioning have been associated with depressive symptoms among a high-risk sample of Caucasian adolescents [18]. Other research has shown that carrying the homozygous *CRHR1* single-nucleotide polymorphism (SNP), rs110402, is associated with a higher cortisol response to the Dex/CRH test only when the subjects experienced childhood maltreatment [19] and have high trait anxiety [20].

CRH and CRHR1 are also present in high levels in the amygdala, hippocampus, and frontal cortex [21,22]. They mediate physiological aspects of the stress response [23–25]. Overactivity of CRH and CRHR1 is found in animal studies with rhesus monkeys [26] and rodents [27] when exposed to early life stress and trauma. For example, CRHR1 antagonists reduce behavioral fear responses to maternal separation in rhesus monkeys [26]. Although these antagonists have been examined as possible treatments for depression with mixed results [28], it does underline its role in further understanding individual variation in the effect of life stress on depression [24].

Past studies have demonstrated a role for variation in the *CRHR1* gene in moderating the effects of childhood abuse, reported retrospectively, on depression-related phenotypes in adulthood [29–31]. Each of these studies found several SNPs of *CRHR1* to be protective from elevations in depressive symptoms when individuals experienced high levels of stress. Before definitive conclusions about this effect can be drawn, however, two questions must be answered. The first involves change across time. Existing findings are mainly based on depressive symptoms assessed at a single point of data collection, making it difficult to determine the temporal ordering of a predictor and the depressive symptoms. Longitudinal studies with repeated assessments of depressive symptoms are needed to clarify the direction of the association. The second question concerns the specificity of childhood adversity. Previous research has focused on maltreatment, but other kinds of childhood adversity, such as economic hardship, contribute to depression. The purpose of this study was to examine the moderational role of *CRHR1* in the longitudinal association between economic hardship and the development of depressive symptoms.

To address these questions, we analyzed data from two longitudinal studies of rural African-American adolescents in which family economic hardship and depressive symptoms were assessed on four occasions across 2.5 years. Participants were genotyped for the *CRHR1* SNPs in the aforementioned studies that protected youths from depression. We predicted that African-American youths carrying more of these SNPs will evince lower levels of depressive symptoms across 2.5 years when they live in families with high levels of economic hardship.

Methods

Overview

This study is unique in combining data from two longitudinal samples of rural African-American adolescents and young adults involving almost 900 participants to test hypotheses about *CRHR1* × family economic hardship interactions. The longitudinal design included parent ratings of economic hardship, youths' reports of depressive symptoms at each wave of data collection, and genotyping of youths for *CRHR1* polymorphisms.

Participants

Strong African-American families–teen. We recruited 502 rural African-American families (51% with daughters) to participate in the Strong African-American families–teen (SAAF–T) randomized prevention trial. Random assignment to the prevention or control condition was controlled in all data analyses. In each family, an adolescent (*M* age = 16.00 years, standard deviation [*SD*] = .57) and his or her primary caregiver (in most cases, the biological mother) provided pretest data. Data were collected on three additional occasions 5 months, 18 months, and 22 months after the pretest. The retention rate was 95% (478/502) at the fourth data collection. At the third data collection, researchers gathered deoxyribonucleic acid (DNA) from 94% (n = 474) of the original sample of youths. These 474 adolescents constituted the SAAF–T segment of the analytic sample for this study. At pretest, families' mean monthly income was $1,482.50, and 63.8% of them lived below federal poverty standards. The primary caregivers worked an average of 41.5 hours per week (*SD* = 20.36); they can be classified as working poor.

Adults in Making. Youths (*M* age = 17.02 years, *SD* = .75; 58.5% female) and primary caregivers from 494 families participated in the Adults in Making (AIM) prevention trial. Again, assignment to the prevention or control condition was controlled in all data analyses. Three visits were made to the original AIM sample 6, 17, and 28 months after the pretest data collection. The retention rate at Wave 4 was 86% (424/494). At the second data collection, researchers gathered DNA from 83.4% (n = 419) of the youths; they constituted the AIM segment of the analytic sample. At pretest, the primary caregivers reported working an average of

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