The Association Between Exposure to Poverty and Anxiety in Middle Childhood: Examination of the Modulating Roles of Coping, Responses to Stress, and Threat Bias Neural Activity

Hannah Bianco
University of Denver

Follow this and additional works at: https://digitalcommons.du.edu/etd

Part of the Psychology Commons

Recommended Citation
Bianco, Hannah, "The Association Between Exposure to Poverty and Anxiety in Middle Childhood: Examination of the Modulating Roles of Coping, Responses to Stress, and Threat Bias Neural Activity" (2016). Electronic Theses and Dissertations. 1199.
https://digitalcommons.du.edu/etd/1199

This Dissertation is brought to you for free and open access by the Graduate Studies at Digital Commons @ DU. It has been accepted for inclusion in Electronic Theses and Dissertations by an authorized administrator of Digital Commons @ DU. For more information, please contact jennifer.cox@du.edu,dig-commons@du.edu.
THE ASSOCIATION BETWEEN EXPOSURE TO POVERTY AND ANXIETY IN MIDDLE CHILDHOOD: EXAMINATION OF THE MODULATING ROLES OF COPING, RESPONSES TO STRESS, AND THREAT BIAS NEURAL ACTIVITY

A Dissertation

Presented to

the Faculty of Arts and Humanities

University of Denver

In Partial Fulfillment

of the Requirements for the Degree

Doctor of Philosophy

by

Hannah Christine Bianco

August 2016

Advisor: Pilyoung Kim, Ph.D.
Abstract

This study examined the relationship between the amount of time spent living in poverty since birth and self-reported symptoms of anxiety in middle childhood. Several models were tested with consideration to the potential modulating roles of coping strategies, responses to stress, and threat bias neural functioning. Exposure to poverty is associated with increased risk for anxiety throughout childhood, adolescence, and into young adulthood (McLoyd, 1998, Najman et al., 2010). Individual factors such as use of various coping strategies and responses to stress, as well as neural processes related to attentional bias toward threat, have been shown to differentially impact risk for anxiety (Bar-Haim et al., 2007; Grant et al., 2003; Wadsworth & Berger, 2006). However, the relationships between exposure to poverty, use of specific coping strategies and responses to stress, threat bias neural functioning, and anxiety symptoms is unknown. In the current study, exposure to poverty, coping and responses to stress, and symptoms of anxiety were measured by child and parent report during a visit to the family’s home. Child brain activation during an attentional threat-bias task was then assessed using functional magnetic resonance imaging (fMRI). The amount of time spent in poverty since birth was correlated with anxiety symptoms. The effect of time spent in poverty since birth was strengthened by endorsement of low levels of involuntary disengagement to result in increased levels of anxiety symptoms. Lastly, the amount of time spent in poverty since
birth was related to ventrolateral prefrontal cortex activation in response to an emotional threat-bias task. These findings support the differential impact of stress responses and neural correlates of threat bias on the relationship between childhood exposure to poverty and anxiety symptoms.
# Table of Contents

Chapter One: Introduction ........................................................................................................ 1  
1.1 Exposure to Poverty ........................................................................................................... 2  
1.2 Influence of Coping and Responses to Stress ................................................................. 5  
1.3 Influence of Threat Bias Neural Activation ..................................................................... 9  
1.4 The current study ............................................................................................................... 11  

Chapter Two: Methods ............................................................................................................. 15  
2.1 Participants ....................................................................................................................... 15  
2.2 Procedure ......................................................................................................................... 18  
2.3 Measures .......................................................................................................................... 18  
2.4 fMRI Paradigm ................................................................................................................ 20  
2.5 Data Analysis .................................................................................................................. 22  
   2.5.1 Behavioral Data Analysis .......................................................................................... 22  
   2.5.2 fMRI Data Acquisition and Imaging Analysis ......................................................... 22  
   2.5.3 Data Analysis Pertaining to Research Questions ...................................................... 23  

Chapter Three: Results ............................................................................................................. 25  
3.1 Behavioral Results ........................................................................................................... 25  
   3.1.1 Correlations among Variables of Interest ................................................................ 25  
   3.1.2 Results related to hypothesis 1: Greater proportions of life spent in poverty will be associated with higher levels of overall anxiety symptoms. .................................................. 28  
   3.1.3 Results related to hypothesis 2: Coping and responses to stress will act as either a route through which exposure to poverty acts on the development of anxiety or as a buffer against or agonist to the influence of poverty exposure on anxiety. ......................................................................................... 28  
3.2 fMRI results .................................................................................................................... 31  
   3.2.1 Incongruent vs. Congruent contrast ....................................................................... 31  
   3.2.2 Results related to hypothesis 3: Exposure to poverty impacts anxiety via threat-bias neural processes in the vlPFC region and amygdala or the effects of poverty on anxiety are amplified by disruptions in these regions of interest. .................................................................................................................. 34  

Chapter Four: Discussion ......................................................................................................... 37  

References ................................................................................................................................ 49
List of Figures

Figure 1. Hypotheses a-c ........................................................................................................ 12
Figure 2. Participant Distribution of Percent of Life Spent in Poverty .............................. 16
Figure 3. Dot-probe Task Design ....................................................................................... 21
Figure 4. Interaction Between Percent of Life Spent in Poverty and Anxiety Symptoms at Low and High Levels of Involuntary Disengagement .................................................. 31
Figure 5. Ventrolateral Prefrontal Cortex and Amygdala Activation During Incongruent > Congruent Contrast ............................................................................................................ 32
Figure 6. Ventrolateral Prefrontal Cortex Activation During Incongruent > Congruent Contrast, Regressed on Percent of Life Spent in Poverty ................................................. 35
Figure 7. Decomposition of the Interaction Between Condition (Incongruent Trials vs. Congruent Trials) and Percent of Life Spent in Poverty on Ventrolateral Prefrontal Cortex Neural Activity ........................................................................................................... 36
Figure 8. Threat Bias Scores Correlated with Ventrolateral Prefrontal Cortex Neural Activation During Incongruent > Congruent Regressed on Percent of Life Spent In Poverty ......................................................................................................................... 36
List of Tables
Table 1. Demographic Characteristics and Descriptive Statistics ........................................ 17
Table 2. Bivariate Correlations for Study Variables (Time Point = Home Visit, n = 47) 26
Table 3. Bivariate Correlations for Study Variables (Time Point = fMRI Scan, n = 30). 27
Table 4. Direct and Indirect Effects of Independent Variables on Total Anxiety
Symptoms (Y) and Significance of Mediation Models. ......................................................... 29
Table 5. Moderated Multiple Regression of Percent of Life Spent in Poverty on Total
Anxiety Symptoms...................................................................................................................... 30
Table 6. Neural Activation Clusters During Incongruent > Congruent Contrast. ........... 33
Chapter One: Introduction

Anxiety is a serious mental health condition that has life-long and society-wide consequences. Children and adolescents are at the greatest risk for developing anxiety disorders, with the median age of onset being 11 years old (Kessler et al., 2005). The onset of anxiety prior to age 13 is a predictor of subsequent anxiety in addition to a range of other mental disorders (e.g. depression, ADHD, CD, etc.) in adolescence (Bittner et al., 2007) and adulthood (Pine, Cohen, Gurley, Brook, & Ma, 1998; Woodward & Fergusson, 2001). Even at subclinical levels, number of reported anxiety symptoms is associated with perceived lower quality of life and well-being and lower academic functioning in middle childhood and adolescence (Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1995; Muris, 2006; Stevanovic, 2013). Anxiety disorders have long-term effects on physical health and mortality as they are associated with increased risk for cardiovascular disease, substance abuse, and suicide in adults (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Further, within the United States, anxiety disorders accounted for approximately $42.3 billion in economic burden in 1990 alone (Greenberg et al., 1999). There is strong support for the deleterious impact of early and chronic exposure to poverty on child brain development and behavior (Shonkoff & Phillips, 2000) as well as the buffering role of adaptive coping and exacerbating role of involuntary stress responses on the development of anxiety and internalizing problems among children exposed to poverty (Wadsworth, Santiago, Wolff, & Reinhard, 2008).
Given the early age at which anxiety disorders typically develop and the far-reaching impact they assert, there is a critical need for increased understanding of vulnerability factors contributing to anxiety in middle childhood.

1.1 Exposure to Poverty

Children raised in low-income environments are at high risk for the development of anxiety disorders (McLoyd, 1998; Spence, Najman, Bor, O'Callaghan, & Williams, 2002). The heightened levels of stress experienced by children in poverty result in increased risk for anxiety and other internalizing disorders (Najman et al., 2010; Wadsworth & Achenbach, 2005; Wadsworth & Berger, 2006; Wadsworth et al., 2008). There are numerous routes by which poverty influences development, as well as many individual and family-level factors that can exacerbate the negative impact of exposure to poverty on children’s socioemotional functioning. Conger et al. (1994) found that adverse economic conditions can be expressed within a family as economic pressure, leading to increased marital conflict, parent depressed mood, and parent hostility toward children. Parent hostility, in turn, was directly related to increased risk for adolescent internalizing and externalizing symptoms. Low maternal responsiveness serves to exacerbate the effects of cumulative risk (i.e., poverty, family turmoil, poor housing quality, violence, etc.) on measures of child allostatic load (physiological “wear and tear” on the body related to experience of chronic stress; Evans, Kim, Ting, Tesher, Shannis, 2007). These examples highlight the Family Stress Model route (Conger & Elder, 1994) by which economic strain affects parental distress which, in turn, disrupts parenting practices, the parent-child relationship, and the parents’ relationship, ultimately having a negative impact on children’s well-being.
But what are the neurobiological implications of associations between financial
disadvantage and increased risk for mental health problems? A growing field that
combines neural and social sciences aims to address this type of question by investigating
the relationship between childhood exposure to poverty and brain development and
functioning. An increasing base of literature supports the deleterious effect of poverty,
and the reduced access to resources and increased stressors that accompany financial
hardship, on neurobiological processes. Recent studies have linked family income to
children’s neurocognitive development across multiple domains including language,
memory, emotion processing, and behavioral problems (Farah et al., 2006; Gianaros et
al., 2008; Hanson et al., 2015; Noble, Wolmetz, Ochs, Farah, & McCandliss, 2006;
Javanbakht et al., 2015). Hanson et al. (2013) found that while children from lower-
income families had similar gray matter brain volumes in infancy, they demonstrated
reduced total gray matter compared with children from middle- and high-income
households by toddlerhood. Additionally, smaller volumes of gray matter were related to
greater behavior problems by pre-school age. Luby (2013) found that early childhood
exposure to poverty was related to smaller white and cortical gray matter and
hippocampal and amygdala volumes 5-10 years later. Hair, Hanson, Wolfe, and Pollak
(2015) additionally found reductions in grey matter volume for children and adolescents
from low-income households (below 150% of the poverty line) relative to developmental
norms matched on sex and age. Further, these anatomical differences significantly
mediated the relationship between exposure to poverty and academic achievement scores.
Tottenham, Hare, Millner, Gilhooly, Zevin, and Casey (2011) found that previously
institutionalized children who were exposed to early adverse rearing conditions
demonstrated elevated amygdala activation and disrupted activation of brain areas associated with support of emotional learning and reactivity processes which, in turn, mediated the link between early rearing environment and eye contact, a marker of social competence and skill. This evidence base for the impact of early exposure to adverse and low-resource environments on detrimental neural development, and subsequent negative effect on cognitive and behavioral functioning, supports the current study’s aim to explore the role of neural processes in the relationship between exposure to poverty and anxiety. As referred to here, previous research examining the relationship between low-income and adverse environments on brain development with children has largely focused on structural differences and their association with indirect measures of functioning (e.g., academic achievement as an indicator of cognitive functioning, eye contact as a marker for social competence skill). The current study aims to look at the direct relationship between exposure to poverty and functional neural processes related to bias for threats in the environment and, in turn, how neural functioning relates to self-reported anxiety symptoms among a sample of youth in middle childhood. It is hoped that elucidation of these direct relationships will help inform interventions for youth from diverse economic backgrounds as well as treatments that target changes in neural processing (e.g., attention bias modification treatment) versus cognitive processes (e.g., cognitive therapy).

There are few studies of the effects of income using functional neuroimaging. Gianaros et al., 2008 found that young adults who reported lower perceived parental social standing (an indicator of socioeconomic disadvantage) exhibited greater amygdala activation in response to threatening facial expressions. Kim et al. (2013) found that
childhood exposure to poverty before age 9 was associated with altered amygdala and vIPFC activation during emotion regulation at age 24 even when controlling for current income level. Additionally, McEwen (2012) found that exposure to the numerous stressors associated with being raised in low-income environments impacts glucocorticoid levels which, in turn, impair PFC and amygdala development and functioning in adulthood. These findings highlight the role of family income during childhood in predicting changes in brain development, specifically in the amygdala and vIPFC regions, in adulthood. However, because the existing literature on the effects of income largely examines neural functioning in adulthood, it is unknown whether individuals from diverse economic backgrounds, and who experience a range of anxiety symptomatology, exhibit altered amygdala and vIPFC activation in middle childhood. The current research focuses on 8- to 10-year-old youth in order to observe possible changes in brain functioning during a sensitive developmental period as well as to gain a better understanding of the proximal causes of early exposure to poverty. Contributions to the extant literature regarding the effects of early-life economically-adverse experiences serves the important role of informing not only research but also the development of public policy and funding of important prevention and intervention programs. Consequently, this research focuses on children from economically diverse backgrounds in order to advance our understanding of the impact of environmental factors on neural development and mental health.

1.2 Influence of Coping and Responses to Stress

The current study also assesses the impact of children’s coping and responses to stress on the relationship between exposure to poverty and anxiety symptoms. Working
within Compas’ and colleagues’ (2001) model, both voluntary (coping) and involuntary responses to stress are examined. Compas et al. (2001) define coping as “conscious volitional efforts to regulate emotion, cognition, behavior, physiology, and the environment in response to stressful events or circumstances” (p. 89). That is, coping involves dealing with a stressor through voluntary attempts at regulating one’s emotions, thoughts, actions, and physiological responses. The ability to effortfully regulate responses such as emotional and physiological arousal affects the strategies one chooses to employ as well as the efficacy with which an individual can carry out a chosen strategy. Because of this, involuntary responses to stress that develop, in part, as a result of conditioning via environmental factors, can have a strong impact on the coping process by limiting the ability to employ subsequent effortful strategies. The model proposed by Compas et al. (2001) includes further categorization of both voluntary and involuntary stress responses to include engagement (e.g., problem-solving or intrusive thoughts) with or disengagement (e.g., denial or emotional numbing) from a stressor or one’s response to a stressor.

Coping strategies involving voluntary disengagement from stressors (e.g., avoidance) as well as involuntary engagement and disengagement responses (e.g., intrusive thoughts and fleeing, respectively), have generally been considered to be maladaptive because they are associated with emotion regulation difficulties (McHugh, Reynolds, Leyro, & Otto, 2013; Sandler, Tein, & West, 1994) and do little to resolve the stressor or allow for children to adapt to the stressor. However, when facing a potentially dangerous stressor (e.g., family conflict), use of disengagement coping strategies such as avoidance may be beneficial, at least in the short term (see Wadsworth, Santiago, Wolff,
& Reinhard, 2008). On the other hand, long-term use of disengagement coping does not help to resolve the stressor or help youth develop active coping strategies. Thus, while disengagement coping may protect youth from dangerous stressors in the moment, its practice hinders the growth of more generalizable and effective coping strategies in the long run.

Coping strategies involving voluntary behavioral and cognitive engagement with stressors, that is primary and secondary control coping strategies (e.g., problem solving and cognitive restructuring, respectively), have generally been considered to be adaptive because they are more effective in resolving the stressor and in regulating negative responses to the stressor (Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000). As is the case with use of disengagement coping, the type of stressor may impact the effectiveness of primary and secondary control coping, even making them maladaptive in some circumstances (Gonzales, Tein, Sandler, & Friedman, 2001; Landis et al., 2007). Carothers, Arizaga, Carter, Taylor, and Grant (2016) found that for urban, predominantly low-income females, voluntary problem-focused coping exacerbated the relationship between exposure to violence and internalizing and externalizing symptoms. The authors suggest that as levels of violence exposure increases, personal controllability over the stressor decreases, thus reducing the effectiveness of active coping and causing more frustration and externalizing problems. This moderating effect of active coping with poverty-related stress highlights the importance of studying the interaction between specific stressors as well as specified responses to stress in order to gain a greater understanding of under what circumstances certain coping strategies are likely to be the most effective.
Financial strain and poverty-related stress have been shown to negatively impact coping which, in turn, is associated with poorer functioning (Poetz, Eyles, Elliot, Wilson, & Keller-Olaman, 2007; Wadsworth, Raviv, Compas, & Connor-Smith, 2005). For example, Kim, Neuendorf, Bianco, and Evans (in press) found that chronic exposure to poverty from birth to early adolescence is associated with increases in the proportion of disengagement coping endorsed in adolescence. Increased use of disengagement coping in adolescence mediated the relationship between exposure to poverty prior to age 13 and both externalizing and internalizing symptoms at age 17. Additionally, disengagement styles of coping with stress have been associated with heightened anxiety symptoms among low-income individuals (Wadsworth & Berger, 2006). It has also been shown that anxiety-disordered children tend to use more disengagement coping strategies and less primary and secondary control coping strategies compared to non-anxious children (Legerstee, Garnefski, Jellesma, Verhulst, & Utens, 2010). Further, experiencing heightened levels of involuntary responses to stress has been shown to amplify the effects of poverty-related stress on anxiety symptoms (Wolff, Santiago, & Wadsworth, 2009). Given the importance of coping and responses to stress for predicting anxiety symptoms, an important component of Cognitive Behavioral Therapy (CBT) interventions for child and adolescent anxiety is the teaching and practice of cognitive and behavioral coping skills. Increases in awareness of automatic cognitions and physiological responses in combination with use of primary and secondary control coping (Kendall, 1994) as well as reductions in the use of disengagement coping strategies such as cognitive and behavioral avoidance (Essau, Conradt, Sasagawa, & Ollendick, 2012) correlate with reduced levels of anxiety.
The environment in which children are raised plays a key role in shaping the development of coping strategies and responses to stress (Kilewer, 2013). Further, use of various effortful coping and involuntary stress responses impacts children’s functioning and has consequences for adjustment in later life (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001). Despite this, we know relatively little about the potential for coping and stress responses to modulate the effect of exposure to poverty on mental health outcomes. Use of adaptive coping may be a pathway by which the challenge of growing up in an economically adverse environment is attenuated, resulting in reductions in worry and hypervigilance (hallmark characteristics of anxiety) or active coping, considered adaptive in many situations, may exacerbate the effects of uncontrollable stressors. Alternatively, involuntary responses to stress may interact with exposure to poverty to exacerbate the influence of poverty on physiological and cognitive processes and, in turn, heighten anxiety symptoms. Gaining a better understanding of how coping and stress responses influence the relationship between exposure to economically adverse environments and anxiety outcomes can inform prevention and intervention efforts targeting youth from low-income backgrounds with the potential to assist such youth in learning coping skills or addressing stress responses to be adaptive rather than potentially damaging (Wadsworth, 2012).

1.3 Influence of Threat Bias Neural Activation

A strong body of research supports the association between anxiety and attentional bias toward threat – relegating attention and cognitive resources toward negative emotional stimuli (e.g., angry expressions) or threats in the environment (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenberg, van Ijzendoorn, 2007; Mogg &
Bradley, 2003; Shechner et al., 2012). While threat-biased attention facilitates detection of potential danger, and may be adaptive in certain contexts (Robinson, Charney, Overstreet, Vytal, & Grillon, 2012), oversensitive attention toward threat (hypervigilance) and difficulties regulating attention away from perceived threats is associated with the etiology and maintenance of anxiety (Britton et al., 2012; Mogg & Bradley, 2003). Additionally, attentional threat bias is associated with anxiety in typically-developing child and adolescent populations (Hadwin et al., 2003; Heim-Dreger, Kohlmann, Eschenbeck, & Burkhardt, 2006) as well as clinically-anxious compared to non-anxious children and adolescents (Roy et al., 2008).

Neuroimaging studies have implicated aberrant brain activity of limbic and ventrolateral prefrontal cortex (vlPFC) regions during attentional threat bias tasks in relation to anxiety among both adolescents and adults (Bishop, Duncan, Brett, & Lawrence, 2004; Fani et al., 2012; Monk et al., 2006; Telzer et al., 2008). The amygdala plays a central role in the detection and appraisal of emotionally salient stimuli in the environment (Ochsner & Gross, 2005) while the vlPFC is implicated in extinction of conditioned fear and more generally in regulation of negative emotions (Phan et al., 2005; Quirk & Beer, 2006). Interestingly, research conducted within adolescent samples has found differences in directionality of vlPFC activation when compared to adults. Heightened vlPFC activation in response to negative stimuli is found in anxiety-disordered children and adolescents while reduced vlPFC activation is found in anxiety-disordered adults (Beesdo et al., 2009; McClure et al., 2007). For example, Monk et al. (2006) found increased right vlPFC activation in response to masked angry facial expressions among adolescents with Generalized Anxiety Disorder (GAD) compared to
their healthy counterparts. Thus, it has been postulated that, among anxious youths, greater vPFC activation may be serving a compensatory role for perturbations of the amygdala whereas, among anxious adults, reduced vPFC activation is associated with failure to suppress amygdala reactivity. Existing neuroimaging studies have focused on clinical adolescent and adult populations, limiting our understanding of neural functioning during the middle childhood developmental stage, before the typical age of onset of anxiety disorders. Understanding how environmental resources, or lack thereof, may be related to the development of anxiety symptoms via neural mechanisms during emotional information processing is critical in order to design efficacious prevention and intervention programs before anxiety symptoms deepen. Identifying altered amygdala and vPFC functions related to varying levels of exposure to poverty as well as a range of anxiety levels can precipitate much needed intervention before the onset of an anxiety disorder (Blackford & Pine, 2012).

1.4 The current study

Coping strategies, involuntary responses to stress, and disruptions to neural processes involved in threat-related attentional bias could reasonably serve as pathways between, or have a moderating influence on, the percent of life a child spends in poverty since birth and anxiety symptoms. The present study extends prior work examining direct links between these various factors by examining potential behavioral and neural routes by which poverty influences mental health in middle childhood. The key research questions include: 1) Does a direct association exist between percent of life spent in poverty and self-report of anxiety symptoms? 2) Do effortful coping and involuntary responses to stress mediate or moderate the association between exposure to poverty and
anxiety symptoms? 3) Does vIPFC and amygdala activation related to attention to threat bias mediate the association between exposure to poverty and anxiety symptoms? (see Figure 1)

**Figure 1. Hypotheses a-c**
Percent of life in poverty is associated with anxiety. Coping and responses to stress mediate/moderate the relationship between percent of life in poverty and anxiety. Threat bias neural activity mediates percent of life in poverty and anxiety. Hypothesis a: Greater proportions of life spent in poverty will be associated with higher levels of overall anxiety symptoms. Hypothesis b: Coping and responses to stress act as either a route through which exposure to poverty acts on the development of anxiety or act as a buffer or agonist on the influence of poverty exposure on anxiety. Primary and secondary control coping contribute to reduced anxiety and disengagement coping and involuntary engagement and disengagement responses to stress contribute to increases in anxiety symptoms. Hypothesis c: Exposure to poverty is associated with increases in amygdala and vIPFC attentional threat bias neural activity which, in turn, is associated with increased anxiety symptoms.
It is hypothesized that greater proportions of life spent in poverty will be associated with higher levels of overall anxiety symptoms as is consistent with previous research supporting a link between being raised in low-income environments and risk for anxiety (McLoyd, 1998; Spence et al., 2002). While many existing studies focus solely on family income or parental education, the current study specifically assesses the percent of a child’s life spent in poverty using an in-depth maternal interview format that captures parental income and family needs on a monthly basis for the entirety of the child’s life. The duration of exposure to poverty is associated with higher levels of both internalizing and externalizing behavior problems (see Brooks-Gunn & Duncan, 1997; Flouri & Midouhas, 2016). This focus on duration of exposure to poverty is based in a strong body of research showing that experiencing persistent poverty, especially during childhood, can have adverse effects on multiple domains of development and throughout the lifespan (Evans & Kim, 2012).

Previous research supports an environmental impact on development of coping strategies and stress responses as well as links between coping, stress responses, and anxiety. Based on the protective role that adaptive coping has been shown to play when children are faced with poverty-related stress (Wadsworth & Santiago, 2008), as well as the negative effect of involuntary engagement for families faced with financial strain (Wolff et al., 2009), coping and responses to stress could feasibly act as either a route through which exposure to poverty acts on the development of anxiety or act as a buffer or agonist on the influence of poverty exposure on anxiety. It is hypothesized that greater percent of life spent in poverty will be associated with higher proportions of disengagement coping and involuntary engagement and disengagement stress responses,
as well as lower proportions of primary and secondary control coping. Additionally, primary and secondary control coping will contribute to reduced anxiety and disengagement coping and involuntary engagement and disengagement responses to stress will contribute to increases in anxiety symptoms.

Lastly, there is strong evidence that childhood exposure to poverty impacts brain development and is related to neural disturbances in functioning (Gianaros et al., 2008; McEwen & Gianaros, 2010; Kim et al., 2013). Additionally, perturbations of attentional bias to threat neural activity in the vlPFC and amygdala have been found to be related to anxiety in children, adolescents, and adults (Bishop et al., 2004; Fani et al., 2012; Monk et al., 2006; Telzer et al., 2008). Given these established relationships, it is hypothesized that exposure to poverty will correlate with increased vlPFC and amygdala threat bias reactivity which will, in turn, be associated with increased anxiety symptoms.
Chapter Two: Methods

2.1 Participants

Study subjects included 47 children (Table 1) who completed all phases of a larger study of socioeconomic and parenting influences on emotional and cognitive development. Subjects were recruited from the wider Denver and Boulder, Colorado areas through a university database of families interested in participating in research and community outreach. Mothers of the children were initially contacted by phone. 146 consented to participate in the telephone screening. During the screening, research assistants interviewed mothers for demographic information, including family income to assess their eligibility for the study. Inclusion criteria included reported family income-to-needs ratio for the past year falling between 0 and 500% (see Figure 2) of the federal poverty line, the participating child was between 8 and 10 years old at the time of entering the study, and the child’s mother lived with the child at least 50% of the time. If eligible, children and their mothers were invited to participate in two additional stages of the study, and a home visit was scheduled. Subjects were excluded prior to participation in the home visit if the child was claustrophobic or if the child had fMRI contraindications (e.g., non-removable metal in body other than metal fillings). Forty-seven families participated in the home visit and 35 children completed an fMRI scan. Subjects were excluded from participation in the fMRI scan if the child had an IQ of less than 80, if the child was currently being treated for a mental health problem, was taking
medication that influenced brain activation or hormone regulation, was receiving psychiatric treatment, or had been diagnosed with a neurological disorder. The primary reason for children not completing the fMRI scan was due to loss of contact with the family (n=5). Other reasons included having orthodontia or dental work after the home visit that included fMRI contraindications (e.g., spacers or braces; n=2), scheduling conflicts (n=2), and child attending fMRI visit but being unable to complete the scan due to being uncomfortable with the scanning procedure (e.g., claustrophobia; n=3). The children who completed the fMRI scan were significantly older (M = 9.26, SD = .59) at the time of the home visit than those who did not complete the fMRI scan (M = 8.80, SD = .77; t(45) = -2.13, p = .039). No other significant demographic differences existed.

Figure 2. Participant Distribution of Percent of Life Spent in Poverty
While the number of children who spent 10%-89% of their lives in poverty was fairly evenly distributed, a greater proportion of children spent either less than 10% (n = 21) or greater than 90% of their lives in poverty (n = 10).
Table 1. Demographic Characteristics and Descriptive Statistics

<table>
<thead>
<tr>
<th></th>
<th>Completed Home Visit (n = 47)</th>
<th>Completed fMRI scan (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>26</td>
<td>15</td>
</tr>
<tr>
<td>Females</td>
<td>21</td>
<td>15</td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>25 (53.2%)</td>
<td>17 (56.7%)</td>
</tr>
<tr>
<td>Black/African American</td>
<td>13 (27.7%)</td>
<td>8 (26.7%)</td>
</tr>
<tr>
<td>Multi-racial/Other</td>
<td>9 (19.1%)</td>
<td>5 (16.6%)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>9.14 (0.66)</td>
<td>9.62 (0.56)</td>
</tr>
<tr>
<td>IQ</td>
<td>99.4 (12.47)</td>
<td>101.47 (13.63)</td>
</tr>
<tr>
<td><strong>Behavioral Self- and Parent-Report</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of life spent in poverty since birth</td>
<td>0.40 (0.40)</td>
<td>0.35 (0.41)</td>
</tr>
<tr>
<td>Anxiety Symptoms (SCARED Total Score)</td>
<td>36.08 (17.14)</td>
<td>31.40 (15.94)</td>
</tr>
<tr>
<td>Primary Control Coping (RSQ Ratio Factor 1)</td>
<td>.17 (.02)</td>
<td>.18 (.02)</td>
</tr>
<tr>
<td>Secondary Control Coping (RSQ Ratio Factor 2)</td>
<td>.24 (.04)</td>
<td>.25 (9.04)</td>
</tr>
<tr>
<td>Disengagement Coping (RSQ Ratio Factor 3)</td>
<td>.15 (.02)</td>
<td>.15 (.02)</td>
</tr>
<tr>
<td>Involuntary Engagement (RSQ Ratio Factor 4)</td>
<td>.25 (.04)</td>
<td>.24 (.03)</td>
</tr>
<tr>
<td>Involuntary Disengagement (RSQ Ratio Factor 5)</td>
<td>.19 (.02)</td>
<td>.18 (.03)</td>
</tr>
<tr>
<td><strong>Dot-probe Task Performance</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Accuracy</td>
<td>-</td>
<td>84.18 (12.06)</td>
</tr>
<tr>
<td>Threat bias score</td>
<td>-</td>
<td>2.9 (22.28)</td>
</tr>
<tr>
<td>Reaction time (ms)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Angry-congruent correct trials</td>
<td>-</td>
<td>691.75 (81.60)</td>
</tr>
<tr>
<td>Angry-incongruent correct trials</td>
<td>-</td>
<td>694.65 (79.26)</td>
</tr>
</tbody>
</table>

Data presented as mean (SD) unless otherwise noted.
2.2 Procedure

Data collection occurred across two stages: a visit by a research team to the family’s home and a visit by the child and her/his parent to an fMRI scanning center. 

Home visit: During the home visit, trained research assistants completed behavioral questionnaires and the Wechsler Abbreviated Scale of Intelligence – Second Edition (WASI-II; Wechsler, 2011) for IQ screening with children. Mothers completed questionnaires on child’s anxiety and family income across the child’s life. fMRI visit: For the fMRI scan, children visited the Center for Innovation and Creativity (CINC) at the University of Colorado, Boulder with their parent. A car service and childcare stipend was available for participants who did not have access to transportation or childcare. Prior to the fMRI scan, child participants completed practice trials of the computer tasks on a laptop and practiced reducing body movement in a mock scanner. During the fMRI scan, participants completed several tasks, including the dot-probe, in a randomized order. The total duration of home visit and fMRI visit were up to 3 hours each. The average length of time between home visits and fMRI scans was 4.43 months (SD=4.01).

2.3 Measures

Percent of life spent in poverty since birth was assessed in an interview format with the child’s mother. Family income and number of family members was assessed on a monthly basis from child’s birth to time of the fMRI scan (Evans & Kim, 2012). The amount of time spent in poverty was determined by number of months in which the family fell below 150% of the federal poverty line as determined by the US Census.
Bureau on an annual basis as an adjusted, per capita index of income (see Table 1 for means and standard deviations).

Anxiety symptoms were measured using the Screen for Child Anxiety Related Emotional Disorders (SCARED) questionnaire (see Table 1 for means and standard deviations). The SCARED is a self-report instrument consisting of 41 statements related to anxiety symptoms which can parallel the Diagnostic and Statistical Manual Mental Disorders, 4th edition (DSM-IV; American Psychiatric Association, 1994) classification of anxiety disorders. The SCARED includes five factors (panic/somatic, generalized anxiety, separation anxiety, social phobia, and school phobia) which were summed to create a total score. The child version of the SCARED demonstrates good internal consistency, test-retest reliability, and discriminant validity (Birmaher et al., 1999) and strong reliability cross culturally (Hale, Crocetti, Raaijmakers, & Meeus, 2011).

Coping strategies and responses to stress were assessed using the Responses to Stress Questionnaire (RSQ; Connor-Smith et al., 2000; see Table 1 for means and standard deviations), which is a 57-item measure that assesses 10 types of coping. The current study used three coping factors: Primary Control, Secondary Control, and Voluntary Disengagement and two responses to stress factors: Involuntary Engagement and Involuntary Disengagement. Narrow-band subscales for each category of coping are: for primary control coping: problem solving, emotional expression, emotion regulation; for secondary control coping: cognitive restructuring, positive thinking, acceptance, and distraction; for voluntary disengagement: denial, wishful thinking, and avoidance; for involuntary engagement: emotional arousal, impulsive action, intrusive thoughts,
physiological arousal, and rumination; for involuntary disengagement: emotional
numbing, cognitive interference, escape, and inaction. Participants’ coping and
involuntary stress responses were anchored to family-related stressors (e.g., “arguing
with your mother/father,” “competing with your sibling,” “having a hard time talking
with your parents,” “your parents hassling or nagging you”). Family-related stressors
were selected as anchors because children from diverse backgrounds (e.g., low- and high-
income households) commonly experience some level of familial conflict or stress which
they make attempts to cope with or to which they experience involuntary responses.
Factor scores were computed as proportion of the total score for all items (i.e., sum of
scores for Primary Control divided by sum of all item responses). The psychometric
characteristics of the RSQ have been well established for a variety of samples, including
low-income families (Connor-Smith et al., 2000).

2.4 fMRI Paradigm

A dot-probe paradigm (see Monk et al., 2006) was used to measure neural
activation in response to threat-bias. In the current study, participants first viewed a
fixation cross for 500msec, followed by a pair of faces with either angry/neutral or
neutral/neutral expressions for 500 msec (face pairs always consisted of the same
individual though the expression varied). An arrow pointing either to the left or right (i.e.
< or >) then appeared for 1100 msec, in the location of either the top or bottom face
image (see Figure 3). Participants indicated which direction the arrow was pointing by
button press. All participants completed 8 practice trials outside of the scanner directly
before the experiment. The task included two runs with 120 trials each. Inter-trial
intervals averaged 400msec with 200-600msec min/max. Three trial types were presented: 1) angry/neutral face pair followed by arrow probe in the same location as angry face (angry-congruent trial) 2) angry/neutral face pair followed by arrow probe in same location as neutral face (angry-incongruent trial) and 3) neutral/neutral face pair followed by arrow probe in same location as one of the neutral faces (neutral trial). There were 80 angry-congruent trials and 80 angry-incongruent trials across both runs. Neutral trials occurred 80 times across both runs and provided a comparison for the emotion conditions. Emotional faces and arrow probes were displayed an equal number of times to each of the top and bottom sections of the screen.

Figure 3. Dot-probe Task Design
Trials are 33% angry-congruent, 33% angry-incongruent, and 33% neutral. Each stimulus appears for 500msec and the order of the trial presentation is randomized across runs. An average jitter of 400ms is used. The total duration of the task is 15 minutes.
2.5 Data Analysis

2.5.1 Behavioral Data Analysis

Behavioral data included response times (RT) which were used to calculate behavioral bias scores by subtracting the mean RT for angry-congruent trials from the mean time for angry-incongruent trials. Positive values indicate attention bias for threat while negative values indicate attentional orienting toward neutral stimuli. Additionally, RTs were used as a covariate during functional neuroimaging analyses to account for individual differences in reaction speed. Behavioral accuracy was calculated in order to determine child performance and attention to the task (i.e., that child was awake and making good effort to complete the task). Behavioral accuracy was calculated as correct trials (participant indicated correct side of the screen that the dot appeared on) divided by total trials. Incorrect trials included participant indicating incorrect location of the dot and responses that occurred outside of the time limit (i.e. RT 800msec> or <200msec; Monk et al., 2006).

2.5.2 fMRI Data Acquisition and Imaging Analysis

Images were acquired using a 3T Siemens Trio scanner with 36 contiguous 3.0mm axial slices. Structural images used T1-weighted anatomical images acquired for spatial registration (3D magnetization prepared rapid acquisition GRE (MPRAGE); 192 1mm axial slices, 256 × 256 matrix, 256 mm FOV, flip angle = 7°). Functional data were acquired (537 T2*-weighted echo-planar-imaging (EPI) volumes; TR = 2,300 ms; TE = 27 ms; flip angle = 73°; field of view = 192 mm; 64 × 64 matrix; 36 axial slices; voxels = 3mm³). fMRI data was analyzed using Analysis of Functional NeuroImages software.
Of the 35 participants who completed the fMRI scanning procedure, 4 were excluded due to more than 15% of TRs being removed because of excessive movement classified as more than 1.0mm in any direction and 1 was excluded due to low behavioral accuracy on the dot-probe task (<60% accuracy). Incorrect trials were excluded from analyses.

### 2.5.3 Data Analysis Pertaining to Research Questions

The direct effect of percent of life spent in poverty on anxiety symptoms was tested using a regression analysis, controlling for covariates, in SPSS 23. Effortful coping (primary control coping, secondary control coping, and disengagement coping) and involuntary responses to stress (involuntary engagement and involuntary disengagement) were tested as potential mediators/moderators of the relationship between percent of life spent in poverty and anxiety, controlling for covariates. Mediation and moderation models were run separately for each of the five RSQ factors due to the small sample size and the dependency of proportional scores (Wadsworth et al., 2005). Mediation and moderation analyses were conducted using a macro developed for SPSS called PROCESS with 95% bias-corrected Confidence Intervals and bootstrapping procedures (10,000 bootstrap resamples; Hayes, 2012; Hayes 2008).

In order to assess whether threat bias neural activity in the hypothesized ROIs (vIPFC and amygdala) was associated with percent of life spent in poverty at the group level, a linear mixed-effects model was conducted using the 3dLME program in AFNI with the contrast of interest including condition (angry-congruent versus angry-incongruent) as a within-subject factor, individual percent of life spent in poverty as a
variable of interest, and with covariates. The contrast of interest, angry-incongruent versus angry-congruent trials, was selected due to its reflection of the difference in participants’ neural response to angry-incongruent trials and angry-congruent trials which is representative of threat bias neural activity, an approach used in previous publications (Price et al., 2014; Telzer, 2008). A voxel-wise p < .005 threshold, followed by cluster thresholds set through Monte Carlo simulations (10,000 iterations, dimensions: 54x64x50, 3x3x3 voxels, 7x7x6mm smoothness) with 3dClustSim in AFNI was used to identify suprathreshold clusters of activation at the whole-brain level (p < .05, corrected). Twenty-four voxels for whole brain were indicated for a p-value of .005 uncorrected, .05 corrected. The amygdala and vlPFC were identified as regions of interest (ROI) based on supporting evidence, thus clusters of activation in these areas were identified using a p-value of .05 uncorrected (Lieberman & Cunningham, 2009). The vlPFC region was defined by Brodmann areas 44, 45, and 47.

Mediation analyses were performed using PROCESS (Hayes, 2012) to assess the indirect effect of threat bias neural activity on the relationship between percent of life spent in poverty and anxiety. Post-hoc analyses were performed to test whether the results were driven by fMRI task condition (angry-congruent vs angry-incongruent) or differed between youth who had spent no portion of their lives in poverty compared to those who had spent some to all of their lives in poverty.
Chapter Three: Results

3.1 Behavioral Results

3.1.1 Correlations among Variables of Interest

As shown in Table 2, percent of life spent in poverty was significantly, negatively correlated with child race \((r(47) = -0.57, p < .001)\) and child IQ \((r(47) = -0.67, p < .001)\) with more time spent in poverty being associated with greater likelihood that the child is a racial minority. Child race and child IQ were also significantly, negatively correlated with total SCARED scores \((r(47) = -0.51, p < .001\) and \(r(47) = -0.45, p = .001\), respectively; see Table 2). Thus, these demographic variables have been included as covariates in all following analyses. Percent of life in poverty was also positively correlated with total SCARED scores \((r(47) = 0.48, p = .001)\), with greater amount of time spent in poverty being related to higher amounts of self-reported symptoms of anxiety. Primary control coping and secondary control coping were negatively associated with anxiety symptoms \((r(47) = -0.46, p = .001\) and \(r(47) = -0.55, p < .001\), respectively). Disengagement coping, involuntary engagement, and involuntary disengagement were positively associated with anxiety symptoms \((r(47) = 0.35, p = .017, r(47) = 0.51, p < .001,\) and \(r(47) = 0.34, p = .021,\) respectively). Threat bias did not correlate with total SCARED scores, \(r(30) = -0.15, p = .438\), or percent of life spent in poverty, \(r(30) = 0.24, p = .204\) (see Table 3).
Table 2. Bivariate Correlations for Study Variables (Time Point = Home Visit, n = 47)

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Child Race</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Child IQ</td>
<td>0.47**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. % of Life in Poverty</td>
<td>-0.57***</td>
<td>-0.67***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Anxiety Symptoms</td>
<td>-0.51***</td>
<td>-0.45**</td>
<td>0.48**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Primary Control Coping</td>
<td>0.34*</td>
<td>0.31*</td>
<td>-0.23</td>
<td>-0.46**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Secondary Control Coping</td>
<td>0.19</td>
<td>0.49**</td>
<td>-0.37*</td>
<td>-0.55***</td>
<td>0.48**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Disengagement Coping</td>
<td>-0.28</td>
<td>-0.35*</td>
<td>0.23</td>
<td>0.35*</td>
<td>-0.58**</td>
<td>-0.35*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Involuntary Engagement</td>
<td>-0.28</td>
<td>-0.42**</td>
<td>0.32*</td>
<td>0.51***</td>
<td>-0.49***</td>
<td>-0.84***</td>
<td>0.14</td>
<td></td>
</tr>
<tr>
<td>9. Involuntary Disengagement</td>
<td>-0.05</td>
<td>-0.23</td>
<td>0.20</td>
<td>0.34*</td>
<td>-0.64***</td>
<td>-0.67***</td>
<td>0.13</td>
<td>0.39**</td>
</tr>
</tbody>
</table>
Table 3. Bivariate Correlations for Study Variables (Time Point = fMRI Scan, n = 30)

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Threat Bias</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. % of Life in Poverty</td>
<td></td>
<td>0.24</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Anxiety symptoms</td>
<td>-0.15</td>
<td></td>
<td>0.41*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Primary Control Coping</td>
<td>-0.15</td>
<td>-0.01</td>
<td></td>
<td>-0.52**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Secondary Control Coping</td>
<td>0.23</td>
<td>-0.30</td>
<td>-0.60**</td>
<td>0.452*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Disengagement Coping</td>
<td>0.00</td>
<td>0.12</td>
<td>0.49**</td>
<td>-0.49**</td>
<td>-0.40*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Involuntary Engagement</td>
<td>-0.03</td>
<td>0.25</td>
<td>0.60**</td>
<td>-0.47*</td>
<td>-0.83***</td>
<td>0.21</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Involuntary Disengagement</td>
<td>-0.17</td>
<td>0.08</td>
<td>0.27</td>
<td>-0.69***</td>
<td>-0.64***</td>
<td>0.10</td>
<td>0.33</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Incongruent &gt; Congruent vlPFC-cluster activation</td>
<td>-0.16</td>
<td>-0.42*</td>
<td>-0.23</td>
<td>0.15</td>
<td>0.27</td>
<td>-0.25</td>
<td>-0.28</td>
<td>-0.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Incongruent &gt; Congruent Amygdala-cluster activation</td>
<td>0.03</td>
<td>-0.06</td>
<td>0.15</td>
<td>-0.02</td>
<td>-0.19</td>
<td>0.00</td>
<td>0.17</td>
<td>0.10</td>
<td>0.28</td>
<td></td>
</tr>
<tr>
<td>11. Percent of Life in Poverty on Incongruent &gt; Congruent vlPFC-cluster activation</td>
<td>-0.37*</td>
<td>-0.66***</td>
<td>-0.24</td>
<td>0.14</td>
<td>0.23</td>
<td>-0.31</td>
<td>-0.19</td>
<td>-0.03</td>
<td>0.64**</td>
<td>0.34</td>
</tr>
</tbody>
</table>
3.1.2 Results related to hypothesis 1: Greater proportions of life spent in poverty will be associated with higher levels of overall anxiety symptoms.

Percent of life spent in poverty significantly predicted anxiety symptoms, $\beta = .48$, $t(45) = 3.71$, $p = .001$, when race and IQ were included as covariates, the effect of percent of life spent in poverty on anxiety became non-significant.

3.1.3 Results related to hypothesis 2: Coping and responses to stress will act as either a route through which exposure to poverty acts on the development of anxiety or as a buffer against or agonist to the influence of poverty exposure on anxiety.

Percent of life spent in poverty was not significantly associated with any of the Responses to Stress Questionnaire (RSQ) scales. As a result, none of the RSQ scales mediated the relationship between percent of life spent in poverty and anxiety (see Table 4). Moderation analyses found that, of the RSQ scales, Involuntary Disengagement moderated the relationship between percent of life spent in poverty and anxiety symptoms ($B = -385.86$, $SE = 181.23$, $p = .039$, see Table 5) whereby reports of lower proportions of involuntary disengagement were associated with higher than average anxiety symptoms for youth who had spent more than half their lives in poverty and lower than average symptoms of anxiety for youth who spent less than half their lives in poverty. For youth who reported higher proportions of involuntary disengagement, percent of life spent in poverty was not significantly associated with anxiety symptoms (see Figure 4).
### Table 4. Direct and Indirect Effects of Independent Variables on Total Anxiety Symptoms (Y) and Significance of Mediation Models.

<table>
<thead>
<tr>
<th>Predictor (X)</th>
<th>Mediator</th>
<th>B</th>
<th>SE</th>
<th>t</th>
<th>p</th>
<th>LLCI</th>
<th>ULCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percent of life in poverty</td>
<td>Covariates</td>
<td>7.00</td>
<td>6.86</td>
<td>1.02</td>
<td>0.313</td>
<td>-6.85</td>
<td>20.85</td>
</tr>
<tr>
<td>Primary Control Coping</td>
<td></td>
<td>-200.54</td>
<td>86.52</td>
<td>-2.32</td>
<td>0.026</td>
<td>-375.27</td>
<td>-25.81</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>-0.13</td>
<td>0.21</td>
<td>-0.61</td>
<td>0.545</td>
<td>-0.55</td>
<td>0.30</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td>-7.22</td>
<td>4.74</td>
<td>-1.52</td>
<td>0.135</td>
<td>-16.79</td>
<td>2.35</td>
</tr>
<tr>
<td>Direct effect of X on Y</td>
<td></td>
<td>7.00</td>
<td>6.86</td>
<td>1.02</td>
<td>0.313</td>
<td>-6.85</td>
<td>20.85</td>
</tr>
<tr>
<td>Indirect effect of X on Y</td>
<td></td>
<td>-1.09</td>
<td>2.90</td>
<td></td>
<td></td>
<td>-10.19</td>
<td>2.80</td>
</tr>
<tr>
<td>(F(4, 42)=6.28, p&lt;.001, R^2=0.380)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of life in poverty</td>
<td>Secondary Control Coping</td>
<td>3.72</td>
<td>6.43</td>
<td>0.58</td>
<td>0.566</td>
<td>-9.27</td>
<td>16.71</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>-188.66</td>
<td>54.49</td>
<td>-3.46</td>
<td>0.001</td>
<td>-298.71</td>
<td>-78.61</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td>0.03</td>
<td>0.21</td>
<td>0.21</td>
<td>0.17</td>
<td>0.868</td>
<td>-0.38</td>
</tr>
<tr>
<td>Direct effect of X on Y</td>
<td></td>
<td>-11.04</td>
<td>4.31</td>
<td>-2.56</td>
<td>0.014</td>
<td>-19.75</td>
<td>-2.33</td>
</tr>
<tr>
<td>Indirect effect of X on Y</td>
<td></td>
<td>3.72</td>
<td>6.43</td>
<td>0.58</td>
<td>0.570</td>
<td>-9.27</td>
<td>16.71</td>
</tr>
<tr>
<td>(F(4, 42)=8.64, p&lt;.001, R^2=0.457)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of life in poverty</td>
<td>Disengagement Coping</td>
<td>7.77</td>
<td>7.25</td>
<td>1.07</td>
<td>0.291</td>
<td>-6.87</td>
<td>22.41</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>149.92</td>
<td>112.64</td>
<td>1.33</td>
<td>0.190</td>
<td>-77.39</td>
<td>377.24</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td>-0.17</td>
<td>0.23</td>
<td>-0.76</td>
<td>0.454</td>
<td>-0.62</td>
<td>0.28</td>
</tr>
<tr>
<td>Direct effect of X on Y</td>
<td></td>
<td>-9.10</td>
<td>4.95</td>
<td>-1.84</td>
<td>0.073</td>
<td>-19.09</td>
<td>0.88</td>
</tr>
<tr>
<td>Indirect effect of X on Y</td>
<td></td>
<td>7.77</td>
<td>7.25</td>
<td>1.07</td>
<td>0.290</td>
<td>-6.87</td>
<td>22.41</td>
</tr>
<tr>
<td>(F(4, 42)=8.56, p&lt;.001, R^2=0.358)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of life in poverty</td>
<td>Involuntary Engagement</td>
<td>5.48</td>
<td>6.69</td>
<td>0.82</td>
<td>0.418</td>
<td>-8.04</td>
<td>19.00</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>181.41</td>
<td>66.29</td>
<td>2.74</td>
<td>0.009</td>
<td>47.53</td>
<td>315.29</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td>-0.06</td>
<td>0.21</td>
<td>-0.28</td>
<td>0.784</td>
<td>-0.48</td>
<td>0.37</td>
</tr>
<tr>
<td>Direct effect of X on Y</td>
<td></td>
<td>-8.79</td>
<td>4.51</td>
<td>-1.95</td>
<td>0.058</td>
<td>-17.91</td>
<td>0.33</td>
</tr>
<tr>
<td>Indirect effect of X on Y</td>
<td></td>
<td>5.48</td>
<td>6.69</td>
<td>0.82</td>
<td>0.420</td>
<td>-8.04</td>
<td>19.00</td>
</tr>
<tr>
<td>(F(4, 42)=7.04, p&lt;.001, R^2=0.407)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent of life in poverty</td>
<td>Involuntary Disengagement</td>
<td>5.74</td>
<td>7.07</td>
<td>0.81</td>
<td>0.421</td>
<td>-8.52</td>
<td>20.00</td>
</tr>
<tr>
<td>IQ</td>
<td></td>
<td>172.26</td>
<td>82.50</td>
<td>2.09</td>
<td>0.043</td>
<td>5.76</td>
<td>338.76</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td>-0.18</td>
<td>0.21</td>
<td>-0.84</td>
<td>0.407</td>
<td>-0.61</td>
<td>0.25</td>
</tr>
<tr>
<td>Direct effect of X on Y</td>
<td></td>
<td>-11.11</td>
<td>4.77</td>
<td>-2.33</td>
<td>0.025</td>
<td>-20.75</td>
<td>-1.48</td>
</tr>
<tr>
<td>Indirect effect of X on Y</td>
<td></td>
<td>5.74</td>
<td>7.07</td>
<td>0.81</td>
<td>0.420</td>
<td>-8.52</td>
<td>20.00</td>
</tr>
<tr>
<td>(F(4, 42)=8.64, p&lt;.001, R^2=0.457)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** 95% bias-corrected CIs with the inclusion of variables on Total Anxiety Symptoms (Y) and significant indirect relationship at \(p < 0.05\).
Table 5. Moderated Multiple Regression of Percent of Life Spent in Poverty on Total Anxiety Symptoms.

<table>
<thead>
<tr>
<th>Model</th>
<th>B</th>
<th>SE</th>
<th>t</th>
<th>p</th>
<th>LLCI</th>
<th>ULCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratio of Primary Control Coping</td>
<td>-191.85</td>
<td>130.51</td>
<td>-1.47</td>
<td>.149</td>
<td>-455.63</td>
<td>71.92</td>
</tr>
<tr>
<td>Percent of Life in poverty</td>
<td>10.78</td>
<td>42.65</td>
<td>0.25</td>
<td>.802</td>
<td>-75.42</td>
<td>96.98</td>
</tr>
<tr>
<td>Primary Control Coping x Percent of Life in Poverty</td>
<td>-21.50</td>
<td>239.50</td>
<td>-0.09</td>
<td>.929</td>
<td>-505.54</td>
<td>462.55</td>
</tr>
<tr>
<td>IQ</td>
<td>-0.13</td>
<td>0.21</td>
<td>-0.61</td>
<td>.546</td>
<td>-.056</td>
<td>0.30</td>
</tr>
<tr>
<td>Race</td>
<td>-7.27</td>
<td>4.83</td>
<td>-1.50</td>
<td>.140</td>
<td>-17.03</td>
<td>2.49</td>
</tr>
<tr>
<td>$F(5, 41)=4.91, p = .001, R^2=0.380$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of Secondary Control Coping</td>
<td>-211.25</td>
<td>74.86</td>
<td>-2.82</td>
<td>.007</td>
<td>-362.55</td>
<td>59.94</td>
</tr>
<tr>
<td>Percent of Life in poverty</td>
<td>-12.84</td>
<td>37.75</td>
<td>-0.34</td>
<td>.736</td>
<td>-89.14</td>
<td>63.47</td>
</tr>
<tr>
<td>Secondary Control Coping x Percent of Life in Poverty</td>
<td>71.26</td>
<td>160.10</td>
<td>0.45</td>
<td>.659</td>
<td>-252.31</td>
<td>394.83</td>
</tr>
<tr>
<td>IQ</td>
<td>0.04</td>
<td>0.21</td>
<td>0.21</td>
<td>.831</td>
<td>-0.38</td>
<td>0.47</td>
</tr>
<tr>
<td>Race</td>
<td>-10.78</td>
<td>4.40</td>
<td>-2.45</td>
<td>.019</td>
<td>-19.66</td>
<td>-1.89</td>
</tr>
<tr>
<td>$F(5, 41)=.82, p &lt; .001, R^2=0.460$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of Disengagement Coping</td>
<td>-33.67</td>
<td>235.01</td>
<td>-0.14</td>
<td>.887</td>
<td>-508.28</td>
<td>440.94</td>
</tr>
<tr>
<td>Percent of Life in poverty</td>
<td>-37.34</td>
<td>51.16</td>
<td>-0.73</td>
<td>.470</td>
<td>-140.66</td>
<td>65.98</td>
</tr>
<tr>
<td>Disengagement Coping x Percent of Life in Poverty</td>
<td>289.17</td>
<td>324.62</td>
<td>0.89</td>
<td>.378</td>
<td>-366.43</td>
<td>944.77</td>
</tr>
<tr>
<td>IQ</td>
<td>-0.24</td>
<td>0.24</td>
<td>-1.00</td>
<td>.324</td>
<td>-0.72</td>
<td>0.24</td>
</tr>
<tr>
<td>Race</td>
<td>-9.56</td>
<td>4.99</td>
<td>-1.92</td>
<td>.062</td>
<td>-19.63</td>
<td>0.51</td>
</tr>
<tr>
<td>$F(5, 41)=4.82, p = .002, R^2=0.370$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of Involuntary Engagement</td>
<td>152.79</td>
<td>93.67</td>
<td>1.63</td>
<td>.111</td>
<td>-36.53</td>
<td>342.11</td>
</tr>
<tr>
<td>Percent of Life in poverty</td>
<td>-15.50</td>
<td>48.50</td>
<td>-0.32</td>
<td>.751</td>
<td>-113.51</td>
<td>82.51</td>
</tr>
<tr>
<td>Involuntary Engagement x Percent of Life in Poverty</td>
<td>83.45</td>
<td>191.01</td>
<td>0.44</td>
<td>.664</td>
<td>-302.60</td>
<td>469.50</td>
</tr>
<tr>
<td>IQ</td>
<td>-0.07</td>
<td>0.21</td>
<td>-0.33</td>
<td>.740</td>
<td>-0.50</td>
<td>0.36</td>
</tr>
<tr>
<td>Race</td>
<td>-8.98</td>
<td>4.58</td>
<td>-1.96</td>
<td>.057</td>
<td>-18.24</td>
<td>0.28</td>
</tr>
<tr>
<td>$F(5, 41)=5.56, p &lt; .001, R^2=0.410$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ratio of Involuntary Disengagement</td>
<td>320.34</td>
<td>105.43</td>
<td>3.04</td>
<td>.004</td>
<td>107.42</td>
<td>533.26</td>
</tr>
<tr>
<td>Percent of Life in poverty</td>
<td>77.03</td>
<td>34.17</td>
<td>2.25</td>
<td>.030</td>
<td>8.03</td>
<td>146.03</td>
</tr>
<tr>
<td>Involuntary Disengagement x Percent of Life in Poverty</td>
<td>-385.86</td>
<td>181.23</td>
<td>2.13</td>
<td>.039</td>
<td>-751.86</td>
<td>19.86</td>
</tr>
<tr>
<td>IQ</td>
<td>-0.16</td>
<td>0.21</td>
<td>-0.78</td>
<td>.440</td>
<td>-0.58</td>
<td>0.26</td>
</tr>
<tr>
<td>Race</td>
<td>-10.02</td>
<td>4.61</td>
<td>-2.17</td>
<td>.036</td>
<td>-19.34</td>
<td>0.70</td>
</tr>
<tr>
<td>$F(5, 41)=6.83, p &lt; .001, R^2=0.454$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: 95% bias-corrected CIs with the inclusion of 0 indicate statistically non-significant indirect relationship at $p < 0.05$. 


3.2 fMRI results

3.2.1 Incongruent vs. Congruent contrast

The whole-brain one-sample t-test identified two significant clusters in a contrast of incongruent versus congruent trials (see Table 6). Additionally, as hypothesized, ROIs of vIPFC ($t(29) = 3.57$, peak TLRC coordinates $x, y, z, = 41, 23, 20, 8$ voxels, $p < .05$ uncorrected, Brodmann areas 45 and 46, extending into the right insula) and amygdala ($t(29) = 4.27$, peak TLRC coordinates $x, y, z, = -16, -7, -13, 7$ voxels, $p < .05$ uncorrected, Brodmann areas 28 and 34, including left parahippocampal gyrus and extending into left uncus, lentiform nucleus, and left medial globus pallidus) showed significant activation for the incongruent-versus-congruent contrast (see Figure 5). Post hoc paired sample t-test analyses were conducted using percent signal-change values.
extracted from the identified ROI regions. vlPFC activation was significantly greater for the congruent condition (M = 0.02, SD = .12) than for the incongruent condition (M = -0.02, SD = 0.13; t(29) = 3.59, p = .001). Amygdala activation was also significantly greater for the congruent condition (M = 0.11, SD = .12) than the incongruent condition (M = 0.02, SD = .09; t(29) = 3.95, p < .001).

(a) (b)

Figure 5. Ventrolateral Prefrontal Cortex and Amygdala Activation During Incongruent > Congruent Contrast
Whole sample Incongruent > Congruent. The whole-brain analysis comparing the incongruent to congruent trials revealed activation significantly greater than zero across all participants in the hypothesized regions: (a) right vlPFC (peak TLRC coordinates x, y, z, = 41, 23, 20, 8 voxels, p < .05 uncorrected, Brodmann areas 45 and 46) and (b) left amygdala (peak TLRC coordinates x, y, z, = −16, −7, −13, 7 voxels, p < .05 uncorrected, Brodmann areas 28 and 34). Figure presented at p = .005.
Table 6. Neural Activation Clusters During Incongruent > Congruent Contrast.
Activation in response to incongruent relative to congruent trials that survived the $p < .05$ cluster-corrected thresholds at voxel-wise threshold of $p = .005$ identified in the one-sample $t$-test (Figure 2) and linear mixed-effects modeling analysis (AFNI 3dLME) as regressed on percent of life in poverty (Figure 3).

<table>
<thead>
<tr>
<th>Area of activation</th>
<th>Brodmann area</th>
<th>Side</th>
<th>Cluster Size (voxels)</th>
<th>x</th>
<th>y</th>
<th>z</th>
<th>t(29)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No regressor variable</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole-brain analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superior Temporal Gyrus</td>
<td>41, 42, 21</td>
<td>R</td>
<td>86</td>
<td>47</td>
<td>-37</td>
<td>8</td>
<td>5.99</td>
</tr>
<tr>
<td>Middle Temporal Gyrus</td>
<td>22, 21, 39</td>
<td>L</td>
<td>26</td>
<td>-55</td>
<td>-49</td>
<td>8</td>
<td>4.40</td>
</tr>
<tr>
<td>ROI analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Middle Frontal Gyrus (vIPFC)</td>
<td>45, 46</td>
<td>R</td>
<td>8</td>
<td>41</td>
<td>23</td>
<td>20</td>
<td>3.57</td>
</tr>
<tr>
<td>Amygdala, Parahippocampal Gyrus</td>
<td>28, 34</td>
<td>L</td>
<td>7</td>
<td>-16</td>
<td>-7</td>
<td>-13</td>
<td>4.27</td>
</tr>
<tr>
<td>Regressed on % of Life in Poverty</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No significant clusters identified at the whole-brain level</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ROI analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insula (vIPFC)</td>
<td>13, 45, 44</td>
<td>R</td>
<td>6</td>
<td>38</td>
<td>20</td>
<td>17</td>
<td>16.99</td>
</tr>
</tbody>
</table>

$F(1, 28)$
3.2.2 Results related to hypothesis 3: Exposure to poverty impacts anxiety via threat-bias neural processes in the vIPFC region and amygdala or the effects of poverty on anxiety are amplified by disruptions in these regions of interest.

Linear mixed-effects modeling analyses (AFNI 3dLME) examining the interaction of condition (incongruent trials versus congruent trials) and percent of life child spent in poverty indicated significant activation in the hypothesized vIPFC region \((F(1, 28) = 16.99, \text{peak TLRC coordinates } x, y, z, = 38, 20, 17, 6 \text{ voxels, } p < .05\) uncorrected, Brodmann areas 13, 45, and 44, including right insula, and extending in to right inferior frontal gyrus; see Figure 6). Post hoc regression analyses demonstrated a significant association between percent of life spent in poverty and vIPFC activity during incongruent vs. congruent contrast \((\beta = -.66, t(29) = -4.63, p < .001)\). Percent of life spent in poverty was associated with vIPFC activity during incongruent trials at the trend level \((\beta = -.33, t(29) = -1.84, p = .077)\). Percent of life spent in poverty was not associated with vIPFC activity during congruent trials \((\beta = .21, t(29) = 1.14, p = .266; \text{see Figure 7})\).

Behavioral threat bias (response time on incongruent trials minus response time on congruent trials) was found to correlate with vIPFC activity regressed on percent of life spent in poverty \(r(30) = 0.38, p = .045\) (see Figure 8). No associations were found between time spent in poverty since birth and threat-biased amygdala activation.

The test of threat bias neural activity as a mediator of percent of life spent in poverty on anxiety symptoms used percent signal-change values extracted from the identified vIPFC region. The mediation model was found to be non-significant due to
threat bias neural activity not being significantly related to anxiety symptoms ($B = 24.42, SE = 48.06, p = .62$).

Figure 6. Ventrolateral Prefrontal Cortex Activation During Incongruent > Congruent Contrast, Regressed on Percent of Life Spent in Poverty
Whole sample Incongruent > Congruent regressed on percent of life spent in poverty. In response to incongruent versus congruent trials, activation in a priori ventrolateral prefrontal cortex ROI ($F(1, 28) = 16.99$, peak TLRC coordinates $x, y, z = 38, 20, 17, 6$ voxels, $p < .05$ uncorrected, Brodmann areas 13, 45, and 44). Figure presented at $p = .005$. 
Overall regression analysis demonstrated a significant association between percent of life spent in poverty and vlPFC activity during incongruent vs. congruent contrast ($\beta = -0.66, t(29) = -4.63, p < .001$). Percent of life spent in poverty and vlPFC activity during incongruent trials was significant at the trend level ($\beta = -0.33, t(29) = -1.84, p = .077$). The association between percent of life spent in poverty and vlPFC activity during congruent trials was non-significant ($\beta = 0.21, t(29) = 1.14, p = .266$).

**Figure 7. Decomposition of the Interaction Between Condition (Incongruent Trials vs. Congruent Trials) and Percent of Life Spent in Poverty on Ventrolateral Prefrontal Cortex Neural Activity**

Negative correlation between Threat Bias Score (average RT on incongruent trials minus average RT on congruent trials) and vlPFC ROI neural activation for incongruent > congruent regressed on Percent of life in Poverty.

**Figure 8. Threat Bias Scores Correlated with Ventrolateral Prefrontal Cortex Neural Activation During Incongruent > Congruent Regressed on Percent of Life Spent In Poverty**
Chapter Four: Discussion

This study examined the associations among exposure to poverty, coping and stress responses, neural correlates of attentional threat bias, and anxiety symptomatology among youth in middle childhood. The amount of time a child spent in poverty since birth was associated with anxiety symptoms only when covariates (child race and IQ) were not included in the model. Involuntary disengagement responses to stress interacted with time spent in poverty since birth to influence self-reported anxiety symptoms. Percent of life spent in poverty was negatively related to ventrolateral prefrontal cortex (vlPFC) activation during an attentional threat bias task. Finally, behavioral threat bias was significantly correlated with vlPFC activation related to percent of life spent in poverty.

The first aim of the current study was to test the association between exposure to poverty and reported symptoms of anxiety. The percent of life a child spent living in poverty was positively associated with self-report of anxiety symptoms; however this effect was significant only when covariates were not included in the analysis. Inclusion of the covariates did not significantly increase the variance explained above and beyond that of percent of life in poverty alone but did reduce the effect of exposure to poverty to non-significant levels. The reduced effect of exposure to poverty with the inclusion of covariates may be a result of a lack of power due to a small sample size. Research with large samples (i.e., N > 1000) has shown a direct effect of childhood exposure to poverty
on increased anxiety symptoms with the inclusion of demographic variables as covariates (Najman et al., 2010; Spence, 2002; Wadsworth & Achenbach, 2005). The current study’s smaller sample size (N = 47) does not allow for additional analyses (e.g., ANOVA) with categorical variables like race or sex due to the cell sizes being too small (e.g., n ≤ 12) and thus precludes an accurate assessment of violations of test assumptions. An additional reason for why a direct effect of exposure to poverty on anxiety symptoms may not be present is related to the developmental timing of anxiety disorders. The median age of onset for anxiety disorders is 11 years old (Kessler et al., 2005). It is possible that a sufficient amount of variance in anxiety symptoms had not emerged among the younger participants in the current study (mean age = 9.62 years old, standard deviation = 0.56) to detect a significant association with exposure to poverty. Future research would benefit from larger sample sizes and inclusion of a wider age-range, such as middle-childhood through adolescence, to detect more complex associations between poverty, anxiety, and demographic factors.

The second aim of the current study explored the role of coping and responses to stress in modulating the relationship between exposure to poverty and anxiety symptoms. Within mediation models, primary and secondary control coping were significantly, negatively related to anxiety symptoms and involuntary engagement and involuntary disengagement responses to stress were significantly, positively related to anxiety symptoms. These findings are consistent with prior research supporting the use of effortful problem-solving and cognitive restructuring coping skills to reduce symptoms of anxiety (Kendall, 1994) as well as evidence showing that maladaptive coping such as
disengagement and involuntary responses to stress are associated with increases in internalizing problems (Compas et al., 2001). No mediation effects of coping or responses to stress were found due to a lack of significant relationship within mediation models between the percent of life youth spent living in poverty and the RSQ subscales. While environmental factors play a clear role in development of cognitive and behavioral mechanisms associated with adaptive coping (Adler & Snibbe, 2003; Lachman & Weaver, 1998), the current study does not support a direct link between exposure to poverty and coping or responses to stress within a mediation framework. This suggests that the influence of poverty on development of coping and stress responses may be more complex, requiring the inclusion of additional, mediating factors (e.g., double mediation or multiple mediation). Previous research supports the role of multiple mediating factors affected by financial strain and influencing coping such as family conflict (DeCarlo Santiago & Wadsworth, 2009), parental socialization of coping (Kilewer, 2013), and ethnicity and cultural background (Brantley, O'Hea, Jones, & Mehan, 2002).

The current study offers supporting evidence for the moderating effect of involuntary responses to stress and exposure to poverty on anxiety symptoms. Youth who reported higher proportions of involuntary disengagement were found to experience moderate levels of anxiety symptoms regardless of the percent of their lives spent in poverty. Children who reported lower proportions of involuntary disengagement and have spent little to none of their lives in poverty appear to be buffered against anxiety. However, greater amounts of time spent in poverty exacerbates the detrimental effect of even low proportions of involuntary disengagement. Involuntary disengagement in
response to stress, such as emotional numbing, cognitive interference, inaction, and escape are common correlates of anxiety symptoms. It is not surprising then, that these stress responses interact with poverty exposure to heighten anxiety. However, the finding that low proportions (in contrast to high proportions) of involuntary disengagement interact with exposure to poverty to affect anxiety symptoms appears contrary in nature. One consideration is that on average participants who reported a high proportion of involuntary disengagement experienced greater symptoms of anxiety (n = 24, M = 36.38, SD = 14.43). In contrast, those who reported both a low proportion of involuntary disengagement and spent less time in poverty experienced significantly lower levels of anxiety (n = 12, M = 18.58, SD = 8.20). This suggests that it is the unique contribution of increased time spent in poverty that amplifies the influence of automatic stress responses to result in anxiety. Given the automatic nature of involuntary stress responses, they may be below children’s awareness, especially at low levels. Thus, there is an important need for interventions to first focus on building awareness and identification of thoughts and behaviors related to involuntary stress responses prior to practice and use of effortful coping skills.

Grant et al.’s (2003) review of research assessing the relationship between stressors and child and adolescent psychopathology highlights the dearth of studies incorporating mediating and moderating factors into their models. The authors recommend that future work focus not only on potential indirect pathways by which stressors impact child and adolescent mental health, but also on not “lumping” potential mediating or moderating constructs together, but rather to be specific about the variables.
being examined and their unique relationships. The current study attempts to address this call for specificity by closely examining the potential for mediating and moderating roles of three specific types of coping (primary control, secondary control, and disengagement coping) and two types of responses to stress (involuntary engagement and involuntary disengagement). In finding the specific moderating effect of involuntary disengagement on the relationship between exposure to poverty and anxiety, we gain insight into a specific area of risk that may be addressed through prevention, intervention, and treatment efforts. Grant et al. (2003) also discuss the importance of defining the stressor (e.g., an event or condition) when testing models of risk and resilience. In the current study, anxiety symptoms, coping, and responses to stress were assessed relative to exposure to poverty. Additionally, coping and responses to stress were anchored to experience of family-related stressors (e.g., “arguing with your mother/father,” “competing with your sibling,” “having a hard time talking with your parents,” “your parents hassling or nagging you”). Family-related stressors were chosen as anchors on the Responses to Stress Questionnaire because children from households of a wide range of economic levels experience negative interactions and conflict with family members. In the present study, the minimum number of stressors endorsed was 7 out of a possible 12 and, on average, participants endorsed experiencing 11 family-related stressors. While family-related stressors were an ideal anchor for this study’s sample, it is possible that an anchor referencing financial strain as a stressor would have resulted in a stronger association between percent of life spent in poverty and coping and responses to stress. Given that a large proportion of the sample had not experienced economic hardships, use
of financial strain-related anchors would not have applied to all individuals. Additionally, there is a strong base of evidence that financial strain works through familial disruptions to impact child and adolescent development (Conger et al., 1992; Conger & Elder, 1994; Conger et al., 2002), further supporting the use and relevance of family-related stressors as a reference point for children’s report of coping and responses to stress in the current study.

The third aim of the current study examined the potential modulating role of neural correlates of attentional threat bias in the relationship between exposure to poverty and anxiety symptoms. Specifically, neural activation corresponding to attention bias for threat was measured as a contrast of the two main experimental conditions: a probe appears on the opposite side of the screen to where an angry face appeared (angry-incongruent) and a probe appears on the same side of the screen as an angry face (angry-congruent). Contrary to hypothesis, it was found that spending a greater proportion of life in poverty was associated with decreased, as opposed to increased, right vlPFC activation. This finding is consistent with Kim et al.’s (2013) finding that young adults who had lower family income when they were 9 years old, demonstrated decreased vlPFC activation during effortful regulation of negative affect at age 24. The ventral prefrontal cortex, in concert with subcortical regions such as the hippocampus and amygdala, is involved in emotion and attentional regulatory processes (Milad et al., 2007; Monk et al., 2008; Strawn et al., 2012), exerting a top-down control effect on subcortical regions during the process of determining allocation of attention to threat (e.g., Bishop et al., 2004). In the current study, vlPFC activation during the angry-congruent condition
was not associated with exposure to poverty. The overall negative association appears to be driven by the trend-level finding that increased exposure to poverty is associated with decreased vIPFC activation on the angry-incongruent condition. One possible explanation for this finding is that children who experience greater amounts of poverty demonstrate an attentional bias for angry faces as well as difficulty regulating attention away from threatening stimuli (i.e., in order to locate the probe on the opposite side of the screen) due to, or because of, reduced recruitment of the vIPFC. The finding that higher behavioral threat bias scores are associated with lower poverty exposure-related threat biased vIPFC activation (and more specifically with vIPFC activation during incongruent trials, \( r(30) = -0.41, p = .024 \)) is in line with this interpretation. The increased response time to angry-incongruent trials compared to angry-congruent trials suggests that children exposed to greater amounts of poverty, who also demonstrate lower threat-bias vIPFC activation, have an attentional preference toward threatening stimuli.

Exposure to low-income environments and their associated heightened levels of stress, increases risk for development of negative socioemotional outcomes (McLoyd, 1998; Spence et al., 2002, Wadsworth & Achenbach, 2005). Individuals raised in lower socioeconomic status environments are at greater risk for health problems in adulthood, even when controlling for adult levels of income (Evans and Kim, 2007, Poulton et al., 2002). Evidence exists for the mediating role of chronic stress exposure in the relationship between being raised in environments of poverty and disrupted neural emotion regulation processes in early adulthood (Kim et al., 2013). This evidence provides support for a process of embedding, whereby exposure to stressful early life...
experiences “gets under the skin,” imparting increased risk for both physical and mental health-related problems later in life (Repetti et al., 2002; Sapolsky, 2004). In line with this, the current findings suggest that exposure to greater amounts of poverty may be expressed as disrupted vIPFC activation during attention for threat.

Behavioral threat bias (response time to incongruent-angry trials minus response time to congruent-angry trials) was negatively correlated with poverty-related vIPFC activity. Children who spend more time in poverty are more likely to be exposed to adverse experiences such as witnessing violence, harsher and more punitive parenting, and family conflict and separation (see Evans, 2004) and as a result may develop increased sensitivity to potential threats in their environment. Additionally, it has been suggested that the key adaptive component underlying anxiety is the ability to detect and activate defense mechanisms to potentially threatening stimuli in the environment (Robinson et al., 2011). One conceptualization that lends itself to development of adaptive threat bias as a result of living in adverse environments is the Adaptive Calibration Model (ACM; Del Giudice, Ellis, & Shirtcliff, 2011). The ACM is founded in the observation that there is extensive individual variation in the functioning of the stress response system, such that there exists a large degree of external events that trigger stress responsivity as well as a wide range of levels of response and duration of the stress response system’s recovery to baseline. The ACM primarily aims to provide a framework for understanding the development of individual differences in stress responsivity through what are considered adaptive mechanisms, taking into account multiple life history-related traits (e.g., opportunities in the environment, parenting, uncontrollable
events) as well as the stress response system’s own adaptive calibration processes.

Working within the ACM framework, a bias toward detecting threats in the environment may develop as a result of exposure to increased real-life threats (e.g., neighborhood violence, inter-parental conflict, crime, etc.) and serve an important function of detecting threats and preparing the stress response system for action.

That a behavioral threat bias associated with anxiety correlates with poverty-related alterations in threat attention neural processes is, thus, not surprising. It is, however, an important connection to draw given prior efforts to reduce anxiety via cognitive and attentional interventions focused on re-training behavioral threat bias and neural correlates of threat bias (Britton et al., 2013; Foa & McNally, 1986; Mathews, Mogg, Kentish, & Eysenck, 1995; O’Toole, & Dennis, 2012). Increasing our understanding of associations among environmental factors, behavioral and neural processes, and mental health outcomes allows for informed intervention and treatment efforts.

The current study found decreased activation of the amygdala and vlPFC associated with attentional threat bias (i.e., incongruent > congruent contrast) averaged across the whole sample. Both the amygdala and vlPFC regions demonstrated greater reactivity during the congruent condition than during the incongruent condition. There is limited research examining neural functioning during dot-probe tasks without relation to other individual factors (e.g., threat bias neural activity related to anxiety). Fu, Taber-Thomas, & Pérez-Edgar (2015), found that among a sample of 9-12 year olds, consisting of half behaviorally inhibited and half healthy controls, amygdala activation was greater
for incongruent versus congruent conditions. Price et al., (2014) found that among a sample of 9 to 13 year olds, 75% of which had an anxiety diagnosis and 25% of which were healthy controls, the rostrodorsal ACC (rdACC) demonstrated greater downward deflection during incongruent trials than during congruent trials. The rdACC has been implicated in both threat reactivity and control over emotional processes. These mixed findings suggest that a cautious approach be taken to interpretation of the current study’s results. That said, increased amygdala and vlPFC reactivity during the congruent condition may suggest continued recruitment of these areas in emotion attentional control processes related to sustaining attention toward threat as opposed to reduced activity observed during the incongruent condition which may indicate successful disengagement from threatening stimuli.

The finding that spending a greater proportion of life in poverty is related to increases in anxiety symptoms, whether adaptive or maladaptive, has implications for prevention and intervention programs. Youth from low-income families, while at higher risk for developing an anxiety disorder (McLoyd, 1998; Spence et al., 2002), also face greater barriers to accessing intervention and treatment services (DeVoe et al., 2007). Universal prevention programs, such as school-based programming, are a promising approach to reaching youth from diverse backgrounds (Herzig-Anderson, Colognori, Fox, Stewart, & Masia Warner, 2012). Also, increased efforts in recent years to expand integrated health care, for example co-location and full integration of mental and medical health care, supports early identification of mental health problems such as anxiety as well as facilitates access to intervention and treatment. Recognition of the early and
significant impact of low-resource environments on children’s well-being, highlights the need for universal and integrated programs and acts to increase awareness of this important health issue.

One limitation of the current study is that the cross-sectional nature of the data does not allow for conclusions regarding directionality and causality. However, although collected retrospectively, data pertaining to exposure to poverty is temporally prior to current assessment of anxiety, coping, and neural functioning, adding to existing literature implicating the deleterious effects of poverty on a multitude of developmental processes. Future research will benefit from examining the associations among these constructs using a prospective design.

A second limitation is that the current study was based on a small sample with high variability among all variables of interest. This prevented the comparison of groups with high (clinical) or low (non-clinical) anxious symptomatology or from categorically different financial backgrounds (i.e., low-income vs. high-income). Additionally, the current findings may have benefited from testing of more complex models (e.g., multiple mediation) but additional analyses were restricted by sample size. Future studies that select for group differences and allow for larger group sample sizes will have greater potential to detect interactions and test complex models.

The present findings revealed distinct associations between exposure to poverty, coping and responses to stress, threat-bias neural activation, and anxiety symptomatology in middle childhood. The impact of exposure to poverty on anxiety, its interaction with involuntary disengagement responses to stress, and its association with neural processes
related to the development and maintenance of anxiety has far reaching implications for interventions targeting youth from economically adverse backgrounds. The translational nature of the current findings lends support for interventions that involve the bolstering of both cognitive (e.g., re-training of threat bias, use of cognitive coping strategies) and behavioral (e.g., awareness of automatic physiological responses to stress, use of behavioral coping strategies) skills. Additionally, the current study provides evidence in line with recent calls for interventions that give special attention to the social context in which mental and physical health problems develop among at-risk youth (Wadsworth, 2012).
References


pediatric generalized anxiety disorder. *Archives of General Psychiatry, 64*(1), 97-106. doi:10.1001/archpsyc.64.1.97


56


