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University of Denver

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MITIGATING RISK FOR ANXIETY AMONG PRESCHOOL-AGE
CHILDREN LIVING IN POVERTY: EVALUATING THE IMPACT OF
ADULT-PROVIDED SOCIAL SUPPORT ON AUTONOMIC STRESS
REACTIVITY

A Dissertation

Presented to
the Faculty of Social Sciences
University of Denver

In Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

by
Brian C. Wolff
August 2009

Advisor: Martha E. Wadsworth, Ph.D.

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Author: Brian C. Wolff

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Advisor: Martha E. Wadsworth, Ph.D.

Degree Date: August 2009

ABSTRACT

Poverty increases children's exposure to stress, elevating their risk for developing patterns of heightened sympathetic and parasympathetic stress reactivity. Repeated patterns of high sympathetic activation and parasympathetic withdrawal place children at risk for anxiety disorders. This study evaluated whether providing social support to preschool-age children during mildly stressful situations helps reduce reactivity, and whether this effect partly depends on children's previously assessed baseline reactivity patterns. The Biological Sensitivity to Context (BSC) theory proposes that highly reactive children may be more sensitive than less reactive children to *all* environmental influences, including social support. In contrast, conventional physiological reactivity (CPR) theory contends that highly reactive children are more vulnerable to the impact of stress but are less receptive to the potential benefits present within their social environments. In this study, baseline autonomic reactivity patterns were measured. Children were then randomly assigned to a high-support or neutral control condition, and the effect of social support on autonomic response patterns was assessed. Results revealed an interaction between baseline reactivity profiles and experimental condition. Children with patterns of high-reactivity reaped more benefits from the social support in the experimental condition than did their less reactive peers. Highly reactive children experienced relatively less reactivity reduction in the neutral condition while experiencing relatively greater reactivity reduction in the support condition. Despite their

demonstrated stability over time, reactivity patterns are also quite susceptible to change at this age; therefore understanding how social support ameliorates reactivity will further efforts to avert stable patterns of high-reactivity among children with high levels of stress, ultimately reducing risk for anxiety disorders.

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Mitigating Risk for Anxiety Among Preschool-age Children Living in Poverty:
Evaluating the Impact of Adult-Provided Social Support on Autonomic Stress Reactivity

Anxiety disorders are the most common mental health disorders diagnosed among preschool-age children (Costello, Egger, & Angold, 2005; Egger & Angold, 2006), affecting almost 10% of children aged two to five (Angold, Egger, Erkanli, & Keeler, in press). Risk for developing anxiety disorders is particularly high among the nearly one in five children currently living in poverty in the United States (U.S. Census Bureau, 2007). With its limited resources, the stressful environment of poverty poses unique challenges to young children's healthy psychological and emotional development. Poor children experience substantially greater exposure to chronic and intense stress than their more affluent peers, such as substandard housing, exposure to violence, and family conflict (Attar, Guerra, & Tolan, 1994; DuRant, Cadenhead, Pendergrast, Slavens, & Linder, 1994; Evans & English, 2002; Wadsworth et al., 2008). Such heightened exposure to early stress elevates risk for developing patterns of intense and prolonged physiological stress responses, which in turn are associated with anxiety (Chen, Matthews, & Boyce, 2002; Evans, 2003). In addition to the distress caused directly by anxiety disorders, such as excessive worrying and irritability, children with anxiety also experience impairment in social relations, behavior, mood, attention, and academic performance (Angold et al., in press; Costello et al., 2005). The pervasiveness of anxiety disorders, along with the negative impact they have on day-to-day functioning, strongly supports the public health importance of investigating early risk factors for anxiety, such as patterns of heightened

physiological stress reactivity, and evaluating methods of ameliorating such risk, particularly among young children living in poverty.

Beyond reducing children's overall stress levels, systematic efforts to reduce young children's risk for anxiety involve identifying the pathways through which early stress leads to psychological impairment. One mediating pathway that has received increasing attention in the psychology literature is the development of patterns of exaggerated stress reactivity within the autonomic nervous system. Evidence from this emerging literature reveals the following pathway through which early stress exposure places children at risk for later autonomic reactivity-mediated mental health problems. (1) Early exposure to chronic and intense stress sensitizes the autonomic nervous system to the presence of new environmental stressors (Repetti, Taylor, & Seeman, 2002). (2) This sensitization leads children to become hyper-vigilant to perceived and real threats in their environment, causing more frequent activation of autonomic responses (Sanchez, Ladd, & Plotsky, 2001). Autonomic over-activation not only amplifies the magnitude and duration of children's reactions to stress, but also reduces their physiological capacity to respond flexibly to changing and novel environmental conditions (Heim & Nemeroff, 2001). (3) Individuals who have experienced repeated, exaggerated stress responses are at risk for developing moderately stable patterns of high-reactivity (Matthews, Saloman, Kenyon, & Allen, 2002). (4) High-reactivity patterns are associated with the development of psychopathology (Pine, Cohen, Gurley, Brook, & Ma, 1998), particularly anxiety disorders (Weems, Zakem, Costa, Cannon, & Watts, 2005). The present study investigated whether a brief, adult-provided social support intervention could temporarily disrupt the pathway from mild stress exposure to heightened reactivity. Further, we

examined whether this intervention was differentially more or less effective as a function of individual differences in children's baseline reactivity patterns. Finding ways to reduce autonomic reactivity during stressful situations has significant implications for reducing risk for childhood anxiety disorders.

The Effects of Early Stress and Poverty on Autonomic Stress Reactivity

Autonomic influences on the heart during times of stress are adaptive, evolutionarily developed responses that typically protect human health from external threats. Under normal circumstances, these quick, coordinated responses help individuals remain alert, prepared, and action-oriented when encountering relatively infrequent stressors. However, for many individuals in the modern world, environmental threats, both perceived and real, are more frequent than that for which humans have been prepared by evolution, leading to over-activation of autonomic response systems. Unfortunately, chronic autonomic over-activation can have the opposite effect of health protection, instead drastically wearing down and damaging both physical and mental health over time, a process known as allostatic load (McEwen, 1998). Allostatic load can occur when any or all physiological stress response systems incur heavy internal or external demands through heightened stress exposure, become less flexible in their ability to respond to new demands, and/or have difficulty recovering when the perceived threats subside (McEwen, 1998). The accumulated effect of repeated autonomic reactivity is a prime example of allostatic load, in that it depletes physiological resources over time, leading initially to states of chronic hyperarousal and dysregulation, which then elevate risk for physical and mental illness across the lifespan (Evans & Kim, 2003; Johnston-Brooks, Lewis, Evans, & Whalen, 1998; McEwen, 1998).

If exaggerated autonomic reactivity and allostatic load are, in part, byproducts of heightened stress exposure, then researchers and practitioners seeking to prevent these outcomes would benefit from knowing who in our society is at greatest risk for such exposure. Though heightened stress exposure is a reality for many individuals in the modern world, stress is not evenly distributed to all segments of the population. Individuals of lower socioeconomic status (SES) experience a disproportionate share of society's stress (Adler et al., 1994). Compared with their more affluent peers, poor youth and adults experience more chronically chaotic and stressful living conditions, including, but not limited to, poorer housing quality, greater crowding and noise levels, more frenetic daily activity, less structure and routine in the home, and less overall predictability (Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005). Further, poor families experience relatively high levels of general economic strain, family and parental conflict, exposure to neighborhood and family violence, and discrimination (Wadsworth et al., 2008). While exposure to one or two of these stressors in isolation may not begin the cascade toward physiological risk and psychopathology, multiple stressors tend to exacerbate one another creating a cumulative risk for both physiological and psychological deterioration (Evans, 2003, Wadsworth et al., 2008). In a study examining cumulative stress exposure and allostatic load among poor children, Evans (2003) found that children with more cumulative physical, psychosocial, and structural risks in their home environments demonstrated greater allostatic load, as indexed by multiple markers of high autonomic and neuroendocrine reactivity. Clearly, the environment of poverty and the experience of poverty-related stress create compounding risks for young children,

who often have limited resources to prevent the onset of psychological and behavioral problems.

Autonomic Stress Reactivity, Poverty, and Children's Mental Health

Over time, as heightened physiological reactivity and allostatic load co-occur with the chronic stress of living in poverty, children from low-income families often develop mental health problems. Although both internalizing and externalizing disorders occur more frequently in lower SES families, reactivity is more specifically associated with internalizing disorders (e.g., Boyce et al., 2001). To test this claim, Boyce et al. recruited a clinical sample of individuals with a range of internalizing and externalizing disorders and assessed their patterns of autonomic stress reactivity. They found that more highly reactive individuals were more likely to be high internalizers, while less reactive individuals were more likely to be high externalizers. This finding coincided with earlier work conducted by Iaboni, Douglas, and Ditto (1998) suggesting the presence of reduced heart rate reactivity among children with Attention Deficit/Hyperactivity Disorder (ADHD). Additionally, Wolff and colleagues (in press) recently found evidence across multiple reporters that heightened involuntary stress response levels exacerbated the longitudinal association between poverty-related stress and anxiety, with only marginal or non-significant evidence predicting aggression.

Further specifying the association between reactivity and mental health, there is increasing evidence from research examining the tripartite model of anxiety and depression that heightened physiological stress reactivity is specific and central to the development of anxiety disorders (e.g., Chorpita, 2002; Weems et al., 2005), whereas low positive affect is a specific hallmark of depressive disorders. A number of past studies

demonstrate the association between heightened reactivity and anxiety. Beidel (1991) found that children with high levels of test anxiety had significantly faster heart rates and higher systolic blood pressure than children with low levels of test anxiety. Similarly, children with Post-Traumatic Stress Disorder (PTSD) demonstrated significantly lower heart rate variability, a type of high-reactivity, than children without PTSD who also experienced a traumatic event (Scheeringa, Zeanah, Myers, & Putnam, 2004). Further, high physiological reactivity is more consistently associated with anxiety as compared to depression. Examining youth participants' heart rate responses to an anxiety-provoking video of a German shepherd running toward the camera, Weems et al. (2005) found physiological reactivity to be uniquely associated with symptoms of anxiety versus depression, controlling for participants' specific fears of animals. Another recent study found that among a community sample of young adolescents, higher levels of parasympathetic withdrawal, an indicator of high stress reactivity, were associated specifically with parent- and self-reported anxiety, not depression. Finally, in a large community sample of children from 3rd through 12th grades, Chorpita (2002) found physiological hyperarousal to be predictive specifically of symptoms of panic disorder in the overall sample and separation anxiety among older participants, but not predictive of depression.

In sum, although early stress and heightened physiological reactivity patterns broadly predict both internalizing and externalizing symptoms, evidence for the association between reactivity and anxiety disorders, *per se*, is most consistent in the literature at this time. As such, findings from the present study may be most relevant for efforts to prevent childhood anxiety, particularly among youth living in poverty.

Autonomic Stress Reactivity

“Physiological reactivity” encompasses several types of biological response to stress. One class of physiological stress response involves activation of the sympathetic branch and withdrawal of the parasympathetic branch of the autonomic nervous system. Sympathetic activation mobilizes physiological resources to assist in an individual’s fight or flight response to environmental threats (Kemeny, 2003). Parasympathetic withdrawal shifts internal resources away from the body’s homeostatic regulatory processes, providing more available energy to sustain active sympathetic responses to stress (Porges, Doussard-Roosevelt., Portales, & Greenspan, 1996). Flexible, coordinated responses from both the sympathetic and parasympathetic systems are central to a healthy physiological response profile.

As such, research examining causes and consequences of individual differences in autonomic stress reactivity benefits not only from measuring both types of autonomic stress response, but also from measuring these responses independently from one another. Independent measurements allow researchers to study the antecedents and consequents of differing combinations of sympathetic and parasympathetic reactivity patterns. Though physiological reactivity indicators such as heart rate and blood pressure are easily measurable and have intuitive appeal given their widespread familiarity, both are caused by a combination of sympathetic and parasympathetic response. This makes it difficult to discriminate the independent or interactive contributions of these two autonomic systems to outcomes of interest (Berntson, Cacioppo, & Quigley, 1991). Thus, in the present study, sympathetic and parasympathetic influences on heart rate invoked by stress were

measured separately for all participants and then analyzed later in differing combinations indicative of less or greater overall autonomic reactivity.

The influence of parasympathetic withdrawal on heart rate is indexed by lower respiratory sinus arrhythmia (RSA), a measure of heart rate variability. The human heart rate varies considerably over the course of seconds and minutes, in part, as a function of breathing inspirations and expirations. During inspiration, the human heart rate typically accelerates, while during expiration, heart rate typically decelerates. RSA captures the magnitude of an individual's heart rate variability within the frequency of breathing. To demonstrate the specificity of the association between RSA and parasympathetic withdrawal, Berntson and colleagues conducted a series of pharmacological blockade studies in which they selectively disrupted sympathetic and parasympathetic activity. These studies confirmed that RSA specifically assesses the influence of parasympathetic withdrawal on the heart, independent from any sympathetic control (Berntson, Cacioppo, & Quigley, 1993; Cacioppo, Uchino, & Berntson, 1994).

Sympathetic influences on heart rate can be measured in several ways. One common method is to measure pre-ejection period (PEP), an index of the time interval from the start of the heart's ventricular depolarization to the opening of the aortic valve and the simultaneous onset of left ventricular ejection of blood, which is marked by the B-point in an impedance cardiograph waveform (Uchino, Cacioppo, Malarkey, & Glaser, 1995). Shorter PEP times indicate greater sympathetic nervous system influence on the heart's myocardial nerves, which results in stronger heart contractions during stress. Similar to the findings linking RSA specifically to parasympathetic influence on cardiac activity, pharmacological blockade studies have demonstrated that PEP, as an index of

myocardial contractility, is solely a reflection of sympathetic influence on the heart, independent of any parasympathetic effects (Berntson et al., 1993; Cacioppo et al., 1994). For methodological purposes in the current study, a highly correlated indicator of myocardial contractility, namely the RZ interval (RZ), was measured and analyzed instead of PEP.¹

Autonomic Stress Reactivity Patterns in Early Childhood

Within the pathway connecting early stress to heightened autonomic stress reactivity and mental illness, the present study focused specifically on how environmental differences in stress and support levels lead to higher or lower reactivity levels for young children. However, at the present time, there are no consensual definitions in the literature as to what constitutes “higher” versus “lower” autonomic reactivity. As such, it was crucial in the present study to operationalize these terms to allow for both a theoretical and empirical understanding of these pathogenic processes. For our purposes, reactivity magnitude was defined by the relative contributions of sympathetic activation and parasympathetic withdrawal to an individual’s overall autonomic stress response pattern. Varying combinations of these two stress response types represent four distinct autonomic response profiles in young children, namely coactivation, coinhibition, reciprocal parasympathetic activation, and reciprocal sympathetic activation (Berntson et al., 1991; Salomon, Matthews, & Allen, 2000). As described in the section below,

¹ The end of the PEP interval (i.e., the B-point) is often difficult to identify, particularly when data contain frequent movement artifacts, as is typically the case with child participants. A number of techniques have been proposed in the research literature to handle this logistical difficulty. One promising method used in the present study was to calculate the interval between the readily identifiable R-peak and dZ/dt_{\max} (i.e., Z) points. On a biological level, the same changes in myocardial contractility that lead to the opening of the aortic valve also impact peak aortic blood flow, which corresponds to the peak of the dZ/dt waveform. Thus, the RZ interval is a close linear function of PEP, accounting for nearly 95% of the variance in the B-point location across ages, genders, baseline measurements, and stressor measurements (Lozano et al., 2007). Due to its utility in this sample of young children, the RZ interval was used for all analyses as a measure of sympathetic activation.

categorization into these four response types has been found to predict individual differences in children's heart rate reactivity, experiences of internalizing versus externalizing psychopathology, and perceptions of family conflict. By viewing reactivity magnitude from this multi-system perspective, we hoped to improve upon the single-system view of reactivity presented in many past studies.

Reciprocal sympathetic activation refers to the combination of high sympathetic activation and high parasympathetic withdrawal in response to stress, and has been defined by many researchers as the most "highly reactive" autonomic stress response profile (e.g., Alkon et al., 2003). Individuals classified as having a reciprocal sympathetic activation profile typically have strong sympathetic responses during stress that engage them directly with the challenge at hand, with concurrent high levels of parasympathetic withdrawal. It should be noted that in response to the infrequent stressor, this highly reactive autonomic pattern can be quite adaptive. Porges and colleagues (1996) originally described parasympathetic withdrawal as an adaptive component of the overall stress response, in that it allows for the greatest increase in heart rate and metabolic output when the sympathetic fight or flight response is engaged during threats. However, repeated reciprocal sympathetic activation responses have significant mental health costs over time as they wear down an individual's ability to respond flexibly to environmental demands.

Over time, experiencing repeated parasympathetic withdrawal during strong sympathetic responses to stress increases risk for psychopathology. Children with a reciprocal sympathetic activation profile are more likely than children with other autonomic profiles to be diagnosed with internalizing disorders (Boyce et al., 2001).

Further, these children experience exaggerated heart rate reactivity throughout the day (Salomon et al., 2000). To use a metaphor, if stepping on a car's gas pedal represents sympathetic activation, and releasing the brake represents parasympathetic withdrawal, what combination of speed and braking would allow an individual to reach her intended destination as quickly and safely as possible? The quickest but most reckless combination would involve keeping one foot off the brake (i.e., high parasympathetic withdrawal) and the other foot on the gas pedal (i.e., high sympathetic activation). This pattern represents reciprocal sympathetic activation. On the other hand, the safest but slowest combination would involve keeping one foot on the brake (i.e., low parasympathetic withdrawal) and the other foot off the gas pedal (i.e., low sympathetic activation). This pattern represents *coinhibition*. Clearly neither of these combinations would successfully help an individual achieve her goal of both a quick *and* safe arrival. A balance of stepping on the gas pedal with regular braking would be most beneficial, ensuring the quickest and safest possible arrival. In physiological terms, this most "beneficial" combination of stepping on the gas (i.e., high sympathetic activation) with regular braking (i.e., low parasympathetic withdrawal) is called *coactivation* (Berntson et al., 1991). The coactivation profile was hypothesized in the present study to be the least reactive profile. Children with this coactivation profile experience significantly lower levels of self- and parent-reported family conflict than individuals with all other reactivity patterns (Salomon et al., 2000), suggesting more adaptive interactions with their home environments. Further, children with low levels of internalizing and externalizing symptoms were most likely to be classified as having coactivation profiles relative to their peers with high symptom levels (Boyce et al., 2001).

With reciprocal sympathetic activation defined as being at the high end of the reactivity continuum, and with coactivation at the low end, the two remaining profiles, coinhibition and reciprocal parasympathetic activation, fall in the middle. Both profiles reflect a lack of sympathetic activation during stress, thus children categorized as having either of these profiles show low levels of physiological engagement with environmental challenges. Children with externalizing disorders are more likely to be classified as having a coinhibition profile than any other autonomic profile (Boyce et al., 2001). Additionally, children with comorbid internalizing and externalizing disorders tend to experience low sympathetic activation regardless of parasympathetic level. Thus, the present study was agnostic as to the relative ordering of the two remaining profiles within the middle of the reactivity continuum, instead considering their common, defining feature to be low sympathetic activation.

Stability and Malleability of Children's Autonomic Stress Reactivity Patterns

By four to five years of age, children begin to develop moderately stable individual differences in their patterns of autonomic stress reactivity (Alkon et al., 2003; Bar-Haim, Marshall, & Fox, 2000; Calkins & Keane, 2004; Kagan, Reznick, Snidman, Gibbons, & Johnson, 1988). Autonomic reactivity patterns in this age range show moderate continuity with earlier (Calkins & Keane, 2004) and later stages in development (Marshall and Stevenson-Hinde, 1998). Consequently, many highly reactive pre-school age children continue to show heightened reactivity to stress in later childhood and adolescence. However, these estimates of *moderate* stability of autonomic reactivity also allow for some variability in children's autonomic response patterns over time and across situations. Contextual influences have been implicated as one source of this

variability. For example, assessments conducted at children's homes revealed lower reactivity levels than identical assessments in either laboratory or child care center settings (Alkon et al., 2003). For this sample, the familiarity of the home context may have reduced mean reactivity levels. The present study built on previous findings of between-group differences across contexts by investigating whether an experimental manipulation of the social environment significantly reduced autonomic reactivity among young children. Because differential responses to specific stressors may also contribute to this variability (e.g., Calkins & Keane, 2004), autonomic reactivity was measured across multiple types of stressors, as has been recommended by previous research (Boyce et al., 2001; Kamarck, Debski, & Manuck, 2000).

The Impact of Social Support on Autonomic Stress Reactivity

With both moderate stability and susceptibility to change, early childhood is an ideal time period to investigate the relative influences of dispositional and contextual variables on autonomic stress reactivity. One environmental influence found to reduce autonomic reactivity in adolescents and adults is the provision of social support during stressful experiences (Uchino, Cacioppo, & Kiecolt-Glaser, 1996). Social support has been conceptualized as “a form of social interaction or communication that fosters a feeling of well-being” (Burlinson, Albrecht, Goldsmith, & Sarason, 1994). Social support at the global, structural level (e.g., social network size) and at the situation-specific, functional level (e.g., emotional support during lab stressors) can buffer individuals from the negative effects of stress by reducing physiological arousal and improving physiological regulation (Uchino, Uno, & Holt-Lunstad, 1999). Several experimental studies have effectively manipulated social support levels by having a confederate behave

with high or low levels of social support (e.g., Hilmert, Kulik, & Christenfeld, 2002), resulting in reduced autonomic reactivity in the high support conditions. To date, no work has experimentally examined the effects of adult-provided social support on reactivity among young children. However, manipulations of interviewer demeanor have been found to affect preschool-age children's memory (Davis & Bottoms, 2002; Quas, Bauer, & Boyce, 2004), with children providing more accurate responses when interviewed in a supportive manner. This suggests that young children can recognize and utilize adult-provided social support during stressful situations. The present study directly assessed the physiological effects of adult-provided social support during stress for preschool-age children.

Biological Sensitivity to Context Theory

Conventional physiological reactivity (CPR) theory posits high-reactivity to be universally associated with negative health outcomes (e.g., Nesse & Young, 2000), citing evidence that stress response systems which once were advantageous in humans' evolutionary history have now become a health burden in the modern world. However, CPR theory does not examine the evolutionary development of *individual differences* in stress reactivity, which may have arisen to adapt particular individuals to environments with varying levels of stress and threat (Ellis & Boyce, 2008). As such, CPR theory deemphasizes the impact that variation in social context has on the association between reactivity and health outcomes. To address the importance of social context, Boyce and Ellis (2005) put forward the Biological Sensitivity to Context (BSC) theory, proposing that children's dispositional patterns of reactivity interact with the social context to influence the development of psychophysiological risk factors and psychopathology.

Drawing from evolutionary theory and evidence, the authors argued that exaggerated physiological reactivity reflects heightened sensitivity to cues in the social environment; therefore, highly reactive children are equally susceptible to social cues that afford protection (e.g., social support) as those that confer risk (e.g., family conflict). Highly reactive children are often more reflective and attentive to themselves and their social environments than less reactive children (e.g., Kagan, Snidman, Zentner, & Peterson, 1999). As such, high-reactivity can help children attend to and absorb the nurturance and available resources in more supportive environments. However, in more stressful, chaotic environments, as is often present among families living in poverty, high-reactivity can lead children to become unhealthily sensitive to perceived threats in ambiguous or novel situations (Ellis, Jackson, & Boyce, 2006).

Several recent studies have found preliminary support for the BSC theory. Quas et al. (2004) found that social support improved mean level memory performance; however this effect did not apply equally to highly reactive and less reactive children. In the social support condition, highly reactive children's memory performance was significantly *better* than that of less reactive children; while in the low-support condition, highly reactive children's memory performance was significantly worse than their less reactive peers. Similarly, Boyce et al. (1995) found that heightened autonomic reactivity was associated with increased frequency of respiratory illness among young children, but only when the child's family environment was characterized by high levels of stress. Highly reactive children in families with less stress and more nurturance actually showed a *lower* frequency of respiratory illness than their less reactive peers. The BSC theory holds promise, both for explicating previously inconsistent findings, and as it suggests that

contextual interventions should be effective in reducing reactivity among highly reactive children. According to BSC theory, more highly reactive children in the present study would be more receptive to the physiological benefits provided by the supportive experimenter. This prediction of BSC theory was tested against the prevailing CPR paradigm, which would predict that high-reactivity transmits uniformly greater risk to young children.

Study Overview and Hypotheses

The primary goals of the present study were to evaluate whether providing social support during mild stressors to preschool-age children living in poverty reduced autonomic stress reactivity relative to a control group without the intervention, and furthermore whether the social support intervention was differentially more effective for children with baseline patterns of high-reactivity. Patterns of autonomic responses to mild stressors were measured twice, allowing for the comparison of baseline autonomic reactivity patterns to reactivity patterns in subsequent supportive or neutral control conditions. BSC theory's prediction that more highly reactive children benefit proportionately more from social support was tested against CPR theory's prediction that more highly reactive children benefit proportionately less because high-reactivity confers uniformly greater risk. These aims were evaluated in a sample of children living in poverty, who are at greater risk for stress exposure, developing patterns of high-reactivity, and developing anxiety disorders than other children. However, the physiological processes under investigation are expected to operate similarly for children from families at all SES levels, suggesting indirect implications for highly stress reactive children not living in poverty.

METHOD

Participants

Seventy-three families with children between the ages of four and five participated in data collection sessions for the present study. Of these 73 families, seven did not finish the entire protocol due to either child refusal during data collection or unexpected equipment/software failure. Of the remaining 66 participants, one was removed from the analyses due to unusable physiological data caused by an inability to obtain adequate physiological signal strength during data collection. As a result, a total of eight participants were not included in analyses, leaving a sample size of 65 participants. The eight excluded participants did not differ significantly from the 65 included participants on any key demographic variables, including age (in months), sex, ethnicity (Latino or non-Latino), monthly income, marital status (married or not married), or educational status (completed high school or did not complete high school). The age difference between the two groups trended toward statistical significance ($t = -1.9, p < .10$), with non-included participants younger ($M = 51.4$ months, $SD = 4.8$) than included participants ($M = 55.8$ months, $SD = 6.2$). It should be noted, however, that 27% ($n = 17$) of the sample of included participants were 51 months or younger, suggesting acceptable generalizability of study results across the full age range of participants. Finally, six participants lacked usable physiological data for either parasympathetic or sympathetic responses at one or both of the phases, thus these participants were listwise deleted from analyses combining these two types of stress response.

Of the 65 participants in the final sample, 38 (58%) were girls and 27 (42%) were boys. Their ages ranged from 48 to 71 months ($M = 55.6$ months, $SD = 6.5$). Children came from diverse racial and ethnic backgrounds. Of the 62 children whose caregivers reported on their race/ethnicity, 61.3% were Latino/a, 17.7% were African-American, 14.5% were Multi-racial, 3.2% were Caucasian, 1.6% were American Indian, and 1.6% were Other. A full 49.2% of participants spoke Spanish as their primary language in the home. Due to their expressed preference, all of these families were administered Spanish-language protocols and questionnaires by bilingual experimenters. All verbal instructions, books, and videos used in the reactivity protocols were translated and back-translated by bilingual individuals prior to the beginning of the study. 54.0% of primary caregivers were currently married and 77.6% had completed high school/GED or higher. Of the 58 caregivers who reported on their highest level of educational attainment, 1.7% received a master's degree, 8.6% received a bachelor's degree, 1.7% received an associate's degree, 13.8% attended some college, 5.2% received a training certificate, 37.9% completed high school or GED equivalent, 22.4% did not complete high school, and 8.6% were currently attending school at any level.

Families were recruited through flyers and during information sessions at Denver metro-area Head Start centers. Interested parents were contacted by phone to schedule a visit to a university research laboratory for one session, lasting approximately 60 minutes. For their participation, families received transportation vouchers by mail prior to the session and monetary compensation at the completion of the session. The only eligibility criteria for participation in this study were that a family's income was at or below the federal poverty line and that the participating child was either four or five years of age at

the time of data collection. Since Denver-metro area Head Start centers only serve preschool-age children from families living in poverty, all interested families from our recruitment pool were eligible.

Procedure

Design Overview

Each 60-minute data collection session consisted of the following sequence of events: establishing informed consent, familiarizing the child with the physiological assessment equipment, applying electrodes/sensors, administering the Phase 1 (P1) baseline reactivity protocol, giving a 7-minute snack break, administering the Phase 2 (P2) experimental manipulation and reactivity protocol, conducting a manipulation check following P2 to assess the salience of the social support provision to participants, removing electrodes/sensors, and conducting a debriefing session with the family and giving the child time to play and relax. Prior to the session, children were randomly assigned to either the supportive or neutral control condition. All experimenters were blind to study hypotheses and trained to administer protocols for all conditions, including P1 and both P2 conditions. Based on evidence that social support has a stronger effect on reducing autonomic reactivity when provided by women (Glynn, Christenfeld, & Gerin, 1999), all experimenters were female. While children completed the physiological reactivity protocols, parents completed questionnaires in an adjacent room, where they were able to view their child on a video monitor. Parents were also given the option to stay in the room and sit behind their children if they or their children were uncomfortable with separating.

Following standardized reactivity protocol procedures (Alkon et al., 2003), the project leader placed disposable spot electrodes in the tetrapolar configuration on the child's neck and chest. The two outer (current) electrodes were placed on the back over the fourth cervical vertebra and the ninth thoracic vertebra, and the two inner (recording) electrodes were placed on the ventral thorax and suprasternal notch and xiphoid process. A respirometer belt was placed above the umbilicus and below the diaphragm to continuously monitor respiration rate throughout the protocol. Children were allowed to choose a toy after all of the electrodes/sensors were connected. All physiological data were filtered through the BioPac MP150 and MindWare Impedance Cardiograph machines. Physiological signals were monitored by the project leader on a PC-based computer using the BioPac AcqKnowledge software in an adjacent control room during the data collection session.

P1 – Baseline Reactivity Protocol

At P1, each child's pattern of sympathetic and parasympathetic physiological reactivity to a series of mildly challenging stressors was measured. For each child, a single composite score, called *baseline physiological reactivity*, was created taking into account both sympathetic and parasympathetic responses averaged across multiple stressors. The reactivity protocol began with the P1 experimenter reading a neutral, calming story to the child for two minutes, allowing the child to become familiar with the apparatus, experimenter, and setting. The experimenter then administered four sequential challenging tasks to assess the child's physiological reactivity. At the completion of these tasks, the experimenter read another neutral story to the child for two minutes. The child

was then given a seven minute break where he or she received another toy and a snack and played quiet games with the P1 experimenter.

P2 – Experimental or Control Condition Reactivity Protocol

Near the end of the break, the P1 experimenter casually informed the child that a new experimenter would take over. The P2 experimenter entered and behaved either neutrally or with social support for the next two minutes. The manipulation of social support involved differences in the demeanor and attitude of an unfamiliar experimenter during P2, so that in the neutral condition the new experimenter behaved in the same neutral manner as the first experimenter, while in the support condition, the experimenter provided structured social support. After the initial two minutes, the P2 reactivity protocol followed the same basic set of tasks as P1, beginning and ending with two minutes of neutral story reading. P2 stressors closely paralleled those in P1, reflecting the same overall types of stress (social, cognitive, physical, and emotional). The specific content of both the P1 and P2 reactivity protocols are described in more detail in the Measures section below.

Experimental Condition Manipulation (Neutral vs. Support)

Following strategies outlined by Davis and Bottoms (2002) and Quas et al. (2004), *social support* providing experimenters were trained to: (1) build rapport with the child by playing games and engaging in light conversation for two minutes upon entering the experiment room, (2) sit directly in front of the child and directly face him or her, (3) speak in a fluctuating, positive voice tone, (4) smile frequently, (5) maintain solid eye contact with the child as much as possible, and (6) provide verbal encouragement at prescribed times during the session.

In contrast, P1 experimenters and P2 control condition experimenters were trained to: (1) sit apart from the child for two minutes upon entering the experiment room, while appearing to do paperwork, (2) sit in front of but face slightly away from the child, (3) speak in a neutral, monotone voice, (4) not smile, (5) only maintain minimal eye contact with the child to ensure he or she was paying attention, and (6) never provide verbal encouragement during the session.

Manipulation Check and Debriefing

Immediately after reading the last story, the project leader entered the room and asked the P2 experimenter to leave for a minute. First, he presented the child with a piece of paper with five stick-figure faces showing expressions indicative of happy, slightly happy, neutral, slightly sad, and sad. The child was asked to “point to the face that shows how (name of P2 experimenter) feels.” Then, the project leader presented the child with a different set of five stick-figure faces showing expression indicative of calm, slightly calm, neutral, slightly angry, and angry, and repeated the same question. The left-to-right face order of faces was counterbalanced to account for potential ordering effects. This manipulation check was found to be effective in a previous, related study (Quas et al., 2004). The P2 experimenter, project leader, and the child’s parent then joined the child in the experiment room. The project leader removed the electrodes/sensors from the child in a friendly, playful manner. Children were permitted to assist in this process, if they desired. The child and parent were informed about the interviewers’ behaviors and the nature of the study, and the child was given the option to choose a game to play to ensure that he or she left the experiment feeling calm or happy. All children were praised for their efforts and chose a final toy for their participation in the study.

Measures

P1 Baseline Reactivity and P2 Experimental Condition Protocols

In consultation with its designers (A. Alkon, personal communication, November 2005), the present study included two slightly modified, standardized reactivity protocols (Alkon et al., 2003; Boyce et al., 2001) designed to be mildly stressful for four to five year-olds and to elicit individual differences in autonomic stress reactivity. The P1 baseline reactivity protocol begins with a baseline measure of autonomic activity, then presents the child with a series of four mildly challenging stressors found to elicit autonomic responses in four domains (social, cognitive, physical, and emotional), and ends with a measure of recovery from autonomic responses. Integrating data from responses to multiple stressors significantly improves the validity and reliability of assessing autonomic stress response patterns in young children (Boyce et al., 2001; Kamarck et al., 2000). The protocol consists of seven epochs presented in a fixed order to all participants, with physiological data collected continuously. The P2 protocol is nearly identical to the P1 protocol, with slightly modified tasks from identical stressor categories, administered in the same fixed order. All video clips and books were commercially available in English and Spanish and were shown or read in the language chosen by the primary caregiver at the beginning of the session.

For both protocols, Epoch 1 is a measure of baseline autonomic activity, consisting of a neutral story read aloud to the child. Epoch 2 is a social stressor, consisting of a structured social interview. Epoch 3 is a cognitive stressor, consisting of the child repeating increasingly difficult sets of digits at P1 and letters at P2. Epoch 4 is a physical stressor, involving the child identifying unknown liquids (lime juice at P1;

lemon juice at P2) placed on the tongue via pipette twice. Epoch 5 consists of a two-minute neutral video clip shown to the child and is followed immediately by Epoch 6 which consists of a two-minute emotionally stressful video clip shown to the child. Finally, Epoch 7 is a measure of autonomic recovery from the preceding emotional stressor and consists of a neutral story read aloud to the child (See Table 1 for more details and citations).

Physiological Reactivity Measures

Respiratory Sinus Arrhythmia (RSA) and RZ Interval (RZ) Data Scoring

RSA and RZ were scored separately using ANSLAB, a commercial physiological data scoring software package. To score RSA, data were screened and edited beat-by-beat for movement noise and other artifacts. RSA was calculated using the natural logarithm of the variance of high-frequency heart period within the frequency bandwidth associated with respiration for young children (0.15 – 0.80 Hz). To score RZ, ANSLAB first ensemble averaged the continuous physiological data for each minute. Minute-by-minute ensemble averaged data were then visually inspected for outliers, which were removed to create the best-fitting impedance waveforms. The cleaned minutes were then averaged together within each epoch to create one RSA and one RZ mean score per epoch. Finally, epoch mean scores were averaged across all four stressor tasks to create separate RSA and RZ overall task mean scores. Scores from the baseline epoch were subtracted from these overall task mean scores to create separate RSA and RZ raw difference scores. Greater parasympathetic withdrawal is reflected by lower RSA raw difference scores, while greater sympathetic activation is reflected by lower RZ raw difference scores. Children's raw difference scores were standardized into T-scores. RZ scores were

reverse-scored, such that greater sympathetic activation was reflected by higher RZ difference T-scores. Greater parasympathetic withdrawal was still reflected by lower RSA difference T-scores.

Summary Autonomic Reactivity Coding Systems and Analytic Strategies

Recent studies have emphasized integrating data from multiple measures of sympathetic and parasympathetic responses to create a more accurate and comprehensive picture of children's reactivity (Berntson, Norman, Hawkley, & Cacioppo, 2008; Quas et al., 2004). However, there is no single "gold standard" method of combining sympathetic and parasympathetic response data. Berntson and colleagues (2008) recently proposed a continuous autonomic index, Cardiac Autonomic Balance (CAB), which places high levels of reciprocal sympathetic activation and high levels of reciprocal parasympathetic activation on two ends of a reactivity continuum, described in more detail below. Though this strategy affords the benefit of a continuous dataset, the coactivation profile was operationalized in the present study as the least reactive profile, not reciprocal parasympathetic activation. Thus, use of this analytic strategy alone is insufficient for our purposes. As such, we explored an additional coding system, which we called the Reciprocal Sympathetic Activation / Coactivation Index (RCI). Using a categorical coding scheme, the RCI placed the coactivation and reciprocal sympathetic activation profiles on two ends of a continuum to directly examine children with the most reactive profile in relation to children with the least reactive profile, respectively. To best explore this newly created predictor variable, two separate analytic strategies using the RCI were implemented, one treating the predictor variable as categorical and the other treating it as continuous. In sum, three separate analytic strategies, namely CAB, RCI (continuous),

and RCI (categorical) used varying combinations of RSA and RZ difference T-scores to examine the present study's hypotheses. Each analytic strategy is described in more detail below.

Analytic strategy 1: Cardiac Autonomic Balance (CAB). Based on recent work by Berntson and colleagues (2008), RZ scores were subtracted from RSA scores to create an index of cardiac autonomic balance (CAB), where higher scores reflected greater reciprocal parasympathetic activation and lower scores reflected greater reciprocal sympathetic activation. For greater ease of interpretation, CAB scores were reversed in the present study so that higher scores reflected greater reciprocal sympathetic activation and lower scores reflected greater reciprocal parasympathetic activation. The outcome variable was created by subtracting P2 CAB scores from P1 CAB scores, reflecting the magnitude of reactivity reduction from P1 to P2 assessments. High baseline reactivity was defined as having high P1 CAB scores. Linear regression analyses were conducted with P1 CAB scores, condition assignment, and their interaction predicting CAB outcome scores. This new method of combining sympathetic and parasympathetic scores into one continuous index has been found in a previous study to be a significant predictor of adults' diabetic status, with the reciprocal sympathetic activation profile positively associated with concurrent diabetes. The present study was the first to use CAB scores to predict health-related outcomes in a sample of young children. According to BSC theory, social support provision should be related to greater CAB reduction for children with higher baseline reactivity. Conversely, according to CPR theory, social support provision should be related to less CAB reduction for children with higher baseline reactivity.

Analytic strategies 2 and 3: Reciprocal Sympathetic Activation / Coactivation Index (RCI), Continuous and Categorical Analyses. Although the CAB index provides a fully continuous integration of parasympathetic and sympathetic data, it does not allow for the direct comparison of the effect of social support on reactivity reduction for children with relatively high or low levels of reciprocal sympathetic activation and coactivation. Such a comparison would help directly assess the influence of social support on reactivity reduction as a function of baseline patterns of high versus low autonomic reactivity, as operationalized in the present study. To address this issue, we explored the use of an additional analytic strategy. First, we categorized participants as having a reciprocal sympathetic activation pattern, a coactivation pattern, or a pattern between these two ends of the reactivity continuum. To accomplish this, participants' RZ scores were first categorized as reflecting either high, middle or low sympathetic activation, with high scores defined as the top quartile of difference T-scores (coded a '3'), low scores defined as the bottom quartile of T-scores (coded a '1'), and middle scores defined as the middle fifty percent of T-scores (coded a '2'). Similarly, participants' RSA scores were categorized as reflecting high, middle or low parasympathetic withdrawal, with high scores defined as the top quartile of difference T-scores (coded a '3'), low scores as the bottom quartile of T-scores (coded a '1'), and middle scores defined as the middle fifty percent of T-scores (coded a '2').

Then, participants' recoded scores were combined to create a semi-continuous reactivity index (RCI), with higher RCI scores indicating more reciprocal sympathetic activation (i.e., high-reactivity) and lower scores indicating more coactivation (i.e., low-reactivity). To do this, participants with an RZ recoded score of '3' (high sympathetic

activation) and an RSA recoded score of '3' (high parasympathetic withdrawal) were coded as a '3' on the RCI (i.e., reciprocal sympathetic activation). Participants with an RZ recoded score of '3' (high sympathetic activation) and an RSA recoded score of '1' (low parasympathetic withdrawal) were coded as a '1' on the RCI (i.e., coactivation). All other combinations of RZ and RSA scores were coded as a '2' on the RCI, reflecting varying levels of sympathetic and parasympathetic influence on reactivity in between these two extremes. The outcome variable was created by subtracting P2 RCI scores from P1 RCI scores, reflecting the magnitude of reactivity reduction from P1 to P2 assessments. The impact of social support on reactivity reduction as a function of P1 RCI scores was analyzed both continuously using linear regression analyses and categorically using ANOVA and contrast analyses, with P1 RCI scores, condition assignment, and their interaction predicting RCI outcome scores. As mentioned earlier, because the RCI was a newly created predictor variable, we sought to explore its utility with multiple types of analyses. The outcome variable for both the continuous and categorical analyses was treated as continuous. According to BSC theory, social support provision should be related to greater RCI reduction for children with higher baseline reactivity. Conversely, according to CPR theory, social support provision should be related to less RCI reduction for children with higher baseline reactivity.

Demographics Questionnaire. Caregivers provided basic demographic and financial information about themselves and their child, including the child's age (in months), sex, and ethnicity (Latino/a or not Latino/a), as well as the caregivers' current income, reported completion or non-completion of high school/GED, and marital status

(married or not married). Caregivers also completed other questionnaires not examined in the present analyses.

RESULTS

Preliminary Analyses

Preliminary analyses were conducted to remove artifact contaminated data and to evaluate reactivity variables for independence and normality. Means and standard deviations of children's raw physiological reactivity scores at P1 and P2 are presented in Tables 2 and 3, respectively. To ensure the quality of each child's physiological data for each epoch, at least one minute per epoch had to be clean. A clean minute was defined as containing at least 30 seconds of usable data, based on current standards within psychophysiological research (D. Lozano, personal communication, November 8, 2005). A mean stressor score was calculated for a child as long as at least two epochs per phase contained sufficient usable data. The internal consistency of RSA and RZ composites consisting of the four stressor tasks within each phase was examined. Cronbach's alphas for all composites within each phase were greater than .90, confirming that averaging across tasks was appropriate for both RZ and RSA at both P1 and P2.

Bivariate correlations between the main reactivity variables and a number of demographic variables, including child's age, sex, and ethnicity, and caregiver's current income, reported completion or non-completion of high school, marital status, and language chosen for the study (English or Spanish) were examined. Demographic variables with significant associations were included as covariates for the relevant analysis. For the CAB analysis, caregivers' marital status was significantly correlated with the CAB outcome variable ($r = -.29, p < .05$), such that children with a married

caregiver showed greater reactivity reductions. For the RCI regression and ANOVA/contrast analyses, child's sex was significantly correlated with the P1 RCI score ($r = .28, p < .05$), such that boys were more likely than girls to show baseline patterns of high-reactivity. No other demographic and reactivity variables were significantly correlated.

To describe the characteristics of our sample further, we also calculated the percentage of participants in each of the four reactivity profile groups at P1. To be able to compare our percentages with those from a sample in an ongoing study of primarily Latino children living in poverty, we used procedures described by that study's Principal Investigator (A. Alkon, personal communication, September 15, 2008). P1 sympathetic and parasympathetic scores were first examined separately, with positive scores coded as 'activation' responses and negative scores coded as 'inhibition' responses for each response type. Then participants with activation on both scales were coded as coactivation, those with activation on the sympathetic scale and inhibition on the parasympathetic scale were coded as reciprocal sympathetic activation, those with activation on the parasympathetic scale and inhibition on the sympathetic scale were coded as reciprocal parasympathetic activation, and those with inhibition on both scales were coded as coinhibition. Percentages of participants in each category were as follows: 34% Reciprocal Sympathetic Activation, 31% Coinhibition, 25% Reciprocal Parasympathetic Activation, and 10% Coactivation. The order of these percentages from highest to lowest was similar to that of the comparison sample from another study, which contained the following percentages: 39% Reciprocal Sympathetic Activation, 25% Coinhibition, 20% Coactivation, and 16% Reciprocal Parasympathetic Activation. Our

sample contained relatively fewer children with the Coactivation response profile and relatively more children with the Reciprocal Parasympathetic Activation profile.

We also calculated the percentage of participants in each reactivity group by gender, though comparison data were not available for this breakdown. The percentages for Females/Males were as follows: 27/46% Reciprocal Sympathetic Activation, 38/21% Coinhibition, 27/21% Reciprocal Parasympathetic Activation, and 8/13% Coactivation. The most notable gender differences were in the Reciprocal Sympathetic Activation and Coinhibition groups, where females were more likely to be categorized as Coinhibition than Reciprocal Sympathetic Activation, while males showed the reverse pattern. Paralleling the results from the bivariate correlations described earlier, these findings suggest that males demonstrated higher reactivity than females in our sample.

Finally, we evaluated both the success of the random assignment procedure in eliminating pre-test group differences on key demographic variables as well as the salience of the social support manipulation to the child participants (i.e., the manipulation check, described earlier). Results from a series of t-tests provided no evidence for significant pre-test group differences between children in the control versus support conditions. Results from the manipulation check suggested that participants did not report differences in experimenter demeanor between the support and control conditions, averaging children's responses across the counterbalanced sad and angry face response choices ($t = -0.54, p = n.s.$).

Primary Analyses

Biological Sensitivity to Context (BSC) theory predicted that relative to the control condition, social support would reduce reactivity more for children with baseline

patterns of higher reactivity than for children with patterns of lower reactivity.

Conversely, conventional physiological reactivity (CPR) theory predicted that relative to the control condition, social support would reduce reactivity less for children with

baseline patterns of higher reactivity than for children with patterns of lower reactivity.

Primary analyses using the three analytic strategies discussed above tested the predictions of these two theories against one another. Results are presented separately below. All reported *B* coefficients are standardized.

Analytic Strategy 1: Cardiac Autonomic Balance (CAB)

A hierarchical linear regression analysis was conducted to examine the extent to which baseline CAB scores interacted with condition assignment to predict CAB score reductions from P1 to P2. Results are presented in Table 4. Continuous P1 CAB T-scores, condition assignment (0 = control, 1 = support), and the covariate of caregivers' marital status (0 = not married, 1 = married) were entered on the first step, and the interaction term created by multiplying P1 CAB T-scores by condition assignment was entered on the second step. The overall model was statistically significant, $F = 7.56(4)$, $p < .001$ and explained 37% of the variance ($R^2 = .37$). Baseline CAB scores, condition assignment, and marital status each significantly predicted CAB reduction scores (see Table 4 for *B* coefficients). The interaction between baseline CAB scores and condition assignment approached statistical significance, $B = .26$, $p < .10$. A plot of this interaction term revealed supporting evidence for the BSC theory's predictions (see Figure 1). In both the control and support conditions, having greater baseline levels of reciprocal sympathetic activation resulted in steeper reductions in reactivity from P1 to P2; however, this effect was significantly stronger in the support condition relative to the control condition. This

finding suggests that regression to the mean is not a likely explanation for the greater reactivity reduction among children with baseline patterns of high-reactivity, in that the change from P1 to P2 is significantly stronger in the support condition than in the control condition.

*Analytic Strategy 2: Reciprocal Sympathetic Activation / Coactivation Index (RCI),
Linear Regression Analysis*

A hierarchical linear regression analysis was conducted to examine the extent to which baseline RCI scores interacted with condition assignment to predict RCI score reductions from P1 to P2. Results are presented in Table 5. RCI reduction scores were regressed on continuous P1 RCI T-scores, condition assignment, the interaction term created by multiplying P1 RCI T-scores by condition assignment, and the covariate of child's sex (0 = female, 1 = male). The overall model was statistically significant, $F = 10.86(4)$, $p < .001$ and explained 45% of the variance ($R^2 = .45$). Baseline RCI scores significantly predicted RCI reduction scores, $B = .44$, $p < .01$. The interaction between baseline RCI scores and condition assignment approached statistical significance, $B = .85$, $p < .10$. A plot of this interaction term revealed supporting evidence for the BSC theory's predictions (see Figure 2). Relative to children in the control condition, children with reactivity profiles more reflective of reciprocal sympathetic activation in the support condition showed greater reactivity reductions than children with profiles more reflective of coactivation.

*Analytic Strategy 3: Reciprocal Sympathetic Activation / Coactivation Index (RCI),
ANOVA and Contrast Analyses*

ANOVA and contrast analyses were conducted to examine the extent to which baseline RCI profile interacted with condition assignment to predict RCI reduction scores from P1 to P2. Results are presented in Table 6. Six predictor groups were created with all possible combinations of P1 RCI profile membership (1 = coactivation, 2 = coinhibition or reciprocal parasympathetic activation, 3 = reciprocal sympathetic activation) and condition assignment (0 = control, 1 = support). These six groups and the covariate of child's sex were used to predict continuous RCI reduction scores. Results indicate that the grouping variable was a statistically significant predictor of RCI reduction score, $F = 7.95 (5), p < .001$, explaining 45% of the variance ($R^2 = .45$). Contrast analyses were then conducted to compare each group's mean RCI reduction score with the mean RCI reduction score of the rest of the sample. Despite low cell sizes, contrast analyses revealed that RCI reduction scores for children in the coactivation and reciprocal sympathetic activation groups in both the control and support conditions were significantly different from outcome scores for the remaining children with moderate reactivity in either the coinhibition or reciprocal parasympathetic activation profile groups (see Table 6 for full report of results). More specifically, children categorized as having a reciprocal sympathetic activation profile demonstrated greater reactivity reductions than all other children in the social support condition relative to the control condition. Further, children categorized as having a coactivation profile demonstrated less reactivity reduction than all other children in the social support condition relative to the control condition. Figure 3 depicts this interaction effect.

DISCUSSION

The overarching aim of the present study was to increase our understanding of the physiological and contextual causes and correlates of childhood anxiety. To accomplish this, we focused specifically on how the interaction between young children's patterns of autonomic reactivity and the availability of social support within their environments during stressful laboratory tasks predicted reductions in autonomic reactivity. Because heightened autonomic reactivity is a known predictor of childhood anxiety disorders, understanding factors that can reduce such reactivity has broader implications for preventing anxiety during children's development.

More specifically, we evaluated whether adding social support to preschool-age children's environments during stressful tasks would reduce the magnitude of their physiological responses. We also assessed whether children's typical patterns of reactivity influenced the effectiveness of this extra social support on reducing physiological reactivity, relative to reactivity reductions among children who did not receive additional support. The research literature does not provide a consensual hypothesis as to whether more highly reactive children, compared with less reactive children, would receive more physiological benefit from a socially supportive context. Conventional physiological reactivity (CPR) theory, which assumes uniformly high risk for stress reactive children, would predict that such additional contextual supports do not provide much protection for highly reactive children. In contrast, proponents of the Biological Sensitivity to Context (BSC) theory would predict that highly reactive

children actually are more attentive to, and absorbent of, supportive cues in their social environments than their less reactive peers; thus, these highly reactive children would experience relatively greater physiological benefits from more supportive relative to less supportive environments.

Results from the present study confirmed the BSC theory's predictions that exaggerated reactivity patterns would enhance the physiological benefits afforded by a socially supportive environment. Children with reactivity patterns more reflective of reciprocal sympathetic activation (i.e., high-reactivity) experienced greater reductions in reactivity than less reactive children, especially in the support condition. Although these findings only trended toward statistical significance, results from all three analytic strategies provided corroborating support for BSC theory predictions, thus lending more overall credibility to the findings. Though one might argue based on the simple effects depicted in Figures 1 and 2 that the baseline scores of highly reactive children simply regressed to the mean when assessed during the experimental or control condition, such an argument does not explain why highly reactive children's scores fell more drastically in the support condition than in the control condition. Further, results from the categorical RCI analysis depicted in Figure 3 reveal a dose-response relationship between social support and reactivity reduction. The low, moderate, and high reactive groups were essentially equivalent in reactivity reduction in the control condition, but showed increasingly stronger reductions in reactivity with each step up from low to high baseline reactivity in the support condition. As such, corroborating results from all three sets of analyses in the present study provided evidence specifically supporting BSC theory

predictions and refuting both CPR theory predictions and arguments for regression to the mean.

Building on a growing body of observational evidence that high-reactivity confers risk in more stress-laden environments while promoting resilience in more support-rich environments, the present study is among the first to provide experimental evidence for this claim. Such context-dependent findings regarding the impact of reactivity on health outcomes, until recently, have been framed as ‘unexpected’, as they were viewed within the prevailing CPR theory framework that high-reactivity uniformly confers risk. In line with BSC theory’s proponents (Boyce & Ellis, 1995), we do not view our context-dependent findings as scientific error, but instead as evidence that individual differences in children’s reactivity patterns interact with variations in the supportiveness of their social environments to produce differential outcomes.

We found support for the BSC theory within a sample of children living in poverty, who are at greater risk for developing patterns of high-reactivity than more affluent children. We chose to recruit such a sample for two purposes. First, given the increased prevalence of reactivity-mediated psychopathology among children from very low-income families, it is important to conduct prevention-oriented studies with samples from this population so that findings can be translated into direct implications for affected individuals. Second, the higher preponderance of exaggerated reactivity patterns among children living in poverty provides ample opportunity to investigate how dispositional and contextual factors influence changes in physiological responding. As such, research with this population also has broader implications for children from all segments of the population. Results from the present study highlight the importance of assessing patterns

of reactivity in addition to environmental support levels when identifying which young children are at greatest risk for developing psychopathology, particularly anxiety disorders.

However, current methods of “assessing” reactivity are highly varied, not only with regard to specific methodologies and technologies, but also in terms of theoretical perspectives on how to define reactivity. The present study contributed to the literature by integrating a number of emerging perspectives into its operational definitions of reactivity. First, rather than assessing only sympathetic or parasympathetic stress responses, we measured responses from each system independently and then combined them statistically to create indices of overall autonomic stress reactivity. From this multi-system perspective, the relative contributions of sympathetic activation and parasympathetic withdrawal to an individual’s overall pattern of autonomic stress response was the variable of interest instead of focusing unitarily on high or low levels from only one system or the other. Although the four autonomic response profiles derived from the four possible combinations of high or low reactivity levels from each system were proposed in the literature nearly two decades ago (Berntson et al., 1991), only a handful of studies have used this classification system since then to investigate physiological processes, particularly among young children.

Second, methods for analyzing combinations of sympathetic and parasympathetic responses as continuous data have only been proposed recently (e.g., Berntson et al., 2008). Though the present study utilized one such method, cardiac autonomic balance (CAB), this method alone did not allow us to examine reactivity on a continuum from reciprocal sympathetic activation to coactivation, the study’s operationalized definitions

of high and low reactivity, respectively. To address this gap, we explored an additional method which we termed the Reciprocal Sympathetic Activation / Coactivation Index (RCI), which placed these two profiles at two ends of the same continuum. Results using both analytic strategies provided corroborating evidence for BSC over CPR predictions, again highlighting the importance of assessing both reactivity patterns and social context in predicting the ability of social support to reduce reactivity. The utility of the RCI in predicting physiological and health-related outcomes will certainly require further investigation, as new methods of analyzing autonomic stress response data continue to emerge.

While these results provide promising support for the BSC theory, they also beg the question of *why* reactivity would function differently depending on social context. From an evolutionary perspective, a flexible autonomic stress response system makes sense, given the variability in stress and support levels present in human environments. Patterns of low to moderate reactivity would function well in normal environments with typical stress and support levels, as there would not be a need to regularly scan one's environment for potential threats. In contrast, patterns of heightened reactivity would function well in more threat-filled environments, where the individual's reactive tendencies could alert him or her to danger and provide more physiological resources for effective responding. In the modern world, however, children often experience more frequent stressors than in our evolutionary past, and many children do not have effective personal means or coping strategies by which to stave off danger, even with additional physiological resources provided by heightened autonomic reactivity. In evolutionary terms, patterns of heightened reactivity would also function well in particularly support-

rich, low-stress environments, where the individual's sensitivity to his or her environment would maximize the chances of attending to and absorbing all possible contextual resources. A recent study correlating the relative stress and support levels of children's early environments (both home and preschool) with patterns of autonomic and endocrine reactivity lend empirical support to these evolutionary claims (Ellis, Essex, & Boyce, 2005).

As was evident in our sample, not all children living in poverty demonstrate patterns of heightened stress reactivity. Some of this variability is likely due to individual differences in genetic predispositions (Matthews et al., 1988), which have been shown to influence the development of reactivity patterns during childhood. Further, poverty-related stress is not experienced identically by all individuals. In past work, we have shown that variations in poverty-related stress contribute to substantial differences in mental health outcomes as well as influence the relationship between self- and parent-reported involuntary stress responses on mental health (Wadsworth et al., 2008; Wolff et al., in press). Future longitudinal studies examining the development of stress reactivity patterns as a function of changing stress and support levels in children's prenatal and postnatal environments over time would shed much light on how these patterns form early in life.

Results from the present study demonstrate that high-reactivity does not necessarily predict poor outcomes, highlighting the importance of assessing features of the social environment. Future studies would benefit from measuring a range of other outcomes, particularly those related to physical and mental health. Given evidence that deficits in self-regulation skills underlie internalizing disorders (Evans, 2003), it will be

important to examine how the interaction between reactivity and context affects children's ability to learn and implement these skills. It may be that highly reactive children in stress-laden environments are at greatest risk for poor self-regulation. However, as this study demonstrates, reactivity does not confer uniform risk, and instead can become a protective factor when additional support is infused into children's environments during stressful situations. Continued confirmation of this finding has clear implications for preventing psychopathology among young children. First, this highlights the importance of systematically assessing both contextual and individual factors to optimize the impact of prevention programs. Identifying children at greatest physiological risk for psychopathology could help support-based prevention programs allocate limited resources and maximize their cost-effectiveness. Further, children with high-reactivity who currently reside in environments with high exposure to stress are in particular need of supportive interactions with adults, particularly when encountering specific stressful circumstances. While children with physiological precursors to anxiety may appear "fine" on the outside because their compliant behaviors draw little attention to themselves, adults could do much through supportive interactions to prevent the development of full-blown anxiety disorders.

The present study has many strengths and avenues for future research directions. As the first study to use an experimental design to assess both the stability and susceptibility to change of preschool-age children's autonomic responses to stress, results contribute vital knowledge about an important physiological risk factor for anxiety disorders. Further, we provided considerable experimental support for the Biological Sensitivity to Context theory, suggesting an important avenue for interventions aimed at

reducing high-reactivity for children in stress-laden environments, mitigating subsequent risk for anxiety disorders. An additional strength of the present study was its focus on understanding the effectiveness of social support to reduce stress in a sample of children living in poverty, many of whom are at increased risk for developing mental health problems as a result of exposure to poverty-related stress. Finally, the present study included a primarily Latino sample, including both Spanish- and English-speaking children, extending psychophysiological research to this important segment of children living in poverty. Further analyses from this rich dataset and future studies will investigate a number of related questions. First, it will be important to investigate in more detail the influences of age, sex, and ethnicity on the relationship between autonomic stress reactivity and social context. In our study, not only did nearly half of the sample indicate Spanish was the primary language spoken in their home, but there was also considerable ethnic diversity across the entire sample. Further analyses can examine the intersecting roles of language, ethnicity, and SES on the interactive effects of reactivity and social support. Additionally, future analyses can examine parent-reports of children's anxiety and video-recordings of children's anxious behavioral responses to stress. With these data, we will be able to investigate the more nuanced associations among observed, parent-reported, and physiologically assessed aspects of autonomic reactivity and anxiety. Finally, even though the physiological processes under investigation in the present study should be universal to children from all segments of the population, it would be beneficial to demonstrate this by transporting this experiment to samples of middle and upper income individuals. Such work could reveal more nuanced evidence of

commonalities as well as dissimilarities in physiological processes among children from multiple points along the SES gradient.

CONCLUSION

With nearly 1 in 10 children in this country diagnosed with an anxiety disorder before the age of six, it is incumbent upon researchers across multiple disciplines to investigate the underlying causes of anxiety, with the goal of infusing evidence-based, biopsychosocial principles into prevention and early intervention efforts. This research has particularly important implications for underserved children living in poverty, many of whom are exposed to undue amounts of stress and are at elevated risk for anxiety and heightened physiological reactivity. Fortunately, results from this study, along with a growing body of evidence supporting the Biological Sensitivity to Context theory, provide hope for easing the physiological pressures which contribute to the development of anxiety disorders. Systematic early assessments of children's physiological reactivity patterns, stress exposure levels, and the availability of social support in children's environments, not only would help identify individuals at greatest risk for sustained high-reactivity and the development of anxiety disorders, but would also provide valuable information about how to best alter children's environments to prevent such outcomes.

LITERATURE CITED

- Alkon, A., Goldstein, L. H., Smider, N., Essex, M. J., Kupfer, D. J., & Boyce, W. T. (2003). Developmental and contextual influences on autonomic reactivity in young children. *Developmental Psychobiology, 42*(1), 64-78.
- Angold, A., Egger, H. L., Erkanli, A., & Keeler, G. (in press). Prevalence and comorbidity of psychiatric disorders in preschoolers attending a large pediatric service. *Archives of General Psychiatry*.
- Attar, B. K., Guerra, N. G., & Tolan, P. H. (1994). Neighborhood disadvantage, stressful life events, and adjustment in urban elementary-school children. *Journal of Clinical Child Psychology, 23*, 391-400.
- Bar-Haim, Y., Marshall, P. J., & Fox, N. A. (2000). Developmental changes in heart period and high-frequency heart period variability from 4 months to 4 years of age. *Developmental Psychobiology, 37*(1), 44-56.
- Beidel, D. C. (1991). Determining the reliability of psycho-physiological assessment in childhood anxiety. *Journal of Anxiety Disorders, 5*, 139-150.
- Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review, 98*, 459-487.
- Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Respiratory sinus arrhythmia: Autonomic origins, physiological mechanisms, and psychophysiological implications. *Psychophysiology, 30*, 183-196.

- Berntson, G. G., Norman, G. J., Hawkley, L. C., & Cacioppo, J. T. (2008). Cardiac autonomic balance versus cardiac regulatory capacity. *Psychophysiology*, *45*, 643-652.
- Boyce, W. T., Chesney, M., Alkon, A., Tschann, J., Adams, S., Chesterman, B., et al. (1995). Psychobiologic reactivity to stress and childhood respiratory illnesses: Results of two prospective studies. *Psychosomatic Medicine*, *57*, 411-422.
- Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, *17*, 271-301.
- Boyce, W. T., Quas, J., Alkon, A., Smider, N. A., Essex, M. J., & Kupfer, D. J. (2001). Autonomic reactivity and psychopathology in middle childhood. *British Journal of Psychiatry*, *179*, 144-150.
- Briggs, R. (1982). *The Snowman*. London, UK: Snowman Enterprises.
- Brown, M. W. (1947). *Goodnight moon*. New York: HarperFestival.
- Burleson, B. R., Albrecht, T. L., Goldsmith, D. J., & Sarason, I. G. (1994). Introduction: The communication of social support. In B.R. Burleson, T.L. Albrecht, & I.G. Sarason (Eds.), *Communication of social support* (pp. xi-xxx). Thousand Oaks, CA: Sage.
- Cacioppo, J., Uchino, B., & Berntson, G. (1994). Individual differences in the autonomic origins of heart rate reactivity: The psychometrics of respiratory sinus arrhythmia and preejection period. *Psychophysiology*, *31*, 412-419.

- Calkins, S. D., & Keane, S. P. (2004). Cardiac vagal regulation across the preschool period: Stability, continuity, and implications for childhood adjustment. *Developmental Psychobiology, 45*(3), 101-112.
- Carlson, R. (1985). Gesell School Readiness Test. In D. Keyser & R. Sweetland (Eds.), *Test critiques*. Kansas City, KS: Test Corporation of America.
- Chen, E., Matthews, K. A., & Boyce, W. T. (2002). Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychological Bulletin, 128*(2), 295-329.
- Chorpita, B. F. (2002). The tripartite model and dimensions of anxiety and depression: An examination of structure in a large school sample. *Journal of Abnormal Child Psychology, 30*(2), 177-190.
- Costello, E. J., Egger, H. L., & Angold, A. (2005). 10-Year research update review: The epidemiology of child and adolescent psychiatric disorders: I. Methods and public health burden. *Journal of the American Academy Child and Adolescent Psychiatry, 44*, 972-986.
- Davis, S. L., & Bottoms, B. L. Effects of social support on children's eyewitness reports: A test of the underlying mechanisms. *Law and Human Behavior, 26*(2), 185-215.
- DuRant, R. H., Cadenhead, C., Pendergrast, A., Slavens, G., & Linder, C. W. (1994). Factors associated with the use of violence among urban Black adolescents. *American Journal of Public Health, 84*(4), 612-617.
- Egger, H. L., & Angold, A. Common emotional and behavioral disorders in preschool children: presentation, nosology, and epidemiology. *Journal of Child Psychology and Psychiatry, 47*(3/4), 313-337.

- Ellis, B. J., & Boyce, W. T. (2008). Biological sensitivity to context. *Current Directions in Psychological Science, 17*, 183-187.
- Ellis, B. J., Essex, M. J., & Boyce, W. T. (2005). Biological sensitivity to context: II. Empirical explorations of an evolutionary-developmental theory. *Development and Psychopathology, 17*, 303-328.
- Ellis, B. J., Jackson, J. J., & Boyce, W. T. (2006). The stress response systems: Universality and adaptive individual differences. *Developmental Review, 26*, 175-212.
- Evans, G. W., & English, K. (2002). The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development, 73(4)*, 1238-1248.
- Evans, B., Gideon, R., & Scheinman, A. (1986). *Stand By Me*. Universal City, CA: Sony Pictures.
- Evans, G. W., Gonnella, C., Marcynyszyn, L. A., Gentile, L., & Salpekar, N. (2005). The role of chaos in poverty and children's socioemotional adjustment. *Psychological Science, 16(7)*, 560-565.
- Glynn, L. M., Christenfeld, N., & Gerin, W. (1999). Gender, social support, and cardiovascular responses to stress. *Psychosomatic Medicine, 61*, 234-242.
- Heim, C., & Nemeroff, C. B. (2001). The role of childhood trauma in the neurobiology of mood & anxiety disorders: Preclinical & clinical studies. *Biological Psychiatry, 49*, 1023-1039.

- Hilmert, C. J., Kulik, J. A., & Christenfeld, N. (2002). The varied impact of social support on cardiovascular reactivity. *Basic and Applied Social Psychology, 24*(3), 229-240.
- Iaboni, F., Douglas, V. I., & Ditto, B. (1997). Psychophysiological response of ADHD children to reward and extinction. *Psychophysiology, 34*, 116-123.
- Johnston-Brooks, C. H., Lewis, M. A., Evans, G. W., & Whalen, C. K. (1998). Chronic stress and illness in children: The role of allostatic load. *Psychosomatic Medicine, 60*, 597-603.
- Kagan, J., Reznick, J. S., Snidman, N., Gibbons, J., & Johnson, M. O. (1988). Childhood derivatives of inhibition and lack of inhibition to unfamiliar. *Child Development, 59*, 1580-1589.
- Kagan, J., & Snidman, K. (1991). Temperamental factors in human development. *American Psychologist, 46*, 856-862.
- Kagan, J., Snidman, N., Zentner, M., & Peterson, E. (1999). Infant temperament and anxious symptoms in school age children. *Development and Psychopathology, 11*, 209-224.
- Kamarck, T. W., Debski, T. T., & Manuck, S. B. (2000). Enhancing the laboratory-to-life generalizability of cardiovascular reactivity using multiple occasions of measurement. *Psychophysiology, 37*, 533-542.
- Kaufman, A., & Kaufman, N. (1983). *Kaufman Assessment Battery for Children*. Circle Pines, MN: American Guidance Service.
- Kemeny, M. E. (2003). The psychobiology of stress. *Current Directions in Psychological Science, 12*(4), 124-129.

- Krauss, R. (1945). *The Carrot Seed*. New York: HarperFestival.
- Lamorisse, A. (1956). *The Red Balloon*. Chatsworth, CA: Homevision.
- Lozano, D. L., Norman, G., Knox, D., Wood, B. L., Miller, B. D., Emery, C. F., et al. (2007). Where to B in dZ/dt . *Psychophysiology*, *44*, 113-119.
- Marshall, P. J., & Stevenson-Hinde, J. (1998). Behavioral inhibition, heart period, and respiratory sinus arrhythmia in young children. *Developmental Psychobiology*, *33*(3), 283-292.
- Matthews, K. A., Manuck, S. B., Stoney, C. M., Rakaczky, C. J., McCann, B. S., Saab, P. et al. (1988). Familial aggregation of blood pressure and heart rate responses during behavioral stress. *Psychosomatic Medicine*, *50*, 341–352.
- Matthews, K. A., Salomon, K., Kenyon, K., Allen, M. T. (2002). Stability of children's and adolescents' hemodynamic responses to psychological challenge: A 3-year longitudinal study of a multiethnic cohort of boys & girls. *Psychophysiology*, *39*, 826-834.
- McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, *338*, 171-179.
- Nesse, R. M., & Young, E. A. ~2000!. Evolutionary origins and functions of the stress response. In G. Fink (Ed.), *Encyclopedia of stress* (Vol. 2, pp. 79–84). New York: Academic Press.
- Pfister, M. (1992). *Rainbow fish*. Zurich: Nord-Sud Verlag AG.
- Pfister, M. (1995). *Rainbow fish to the rescue*. Zurich: Nord-Sud Verlag AG.

- Pine, D. S., Cohen, P., Gurley, D., Brook, J., & Ma, Y. (1998). The risk for early-adulthood anxiety and depressive disorders in adolescents with anxiety and depressive disorders. *Archives of General Psychiatry*, *55*(1), 56-64.
- Porges, S. W. (1992). Autonomic regulation and attention. In B. Campbell, H. Hayne, & R. Richardson (Eds.), *Attention and information processing in infants and adults: Perspective from human and animal research* (pp. 201-223). Hillsdale, NJ: Erlbaum.
- Porges, S. W., Doussard-Roosevelt, J. A., Portales, A. L., & Greenspan, S. I. (1996). Infant regulations of the vagal “brake” predict child behavior problems: A psychobiological model of social behavior. *Developmental Psychobiology*, *29*(8), 697-712.
- Quas, J. A., Bauer, A., & Boyce, W. T. (2004). Physiological reactivity, social support, and memory in early childhood. *Child Development*, *75*(3), 797-814.
- Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, *128*(2), 330-366.
- Salomon, K., Matthews, K., & Allen, M. (2000). Patterns of sympathetic and parasympathetic reactivity in a sample of children and adolescents. *Psychophysiology*, *37*, 842-849.
- Sanchez, M. M., Ladd, C. O., & Plotsky, P. M. (2001). Early adverse experience as a developmental risk factor for later psychopathology: Evidence from rodent and primate models. *Development and Psychopathology*, *13*, 419-449.

- Scheeringa, M. S., Zeanah, C. H., Myers, L., & Putnam, F. (2004). Heart period and variability findings in preschool children with post traumatic stress symptoms. *Biological Psychiatry, 55*, 685-691.
- Spielberg, S. (1988). *The Land Before Time*. Universal City, CA: Amblin Entertainment.
- Uchino, B. N., Cacioppo, J. T., & Kiecolt-Glaser, J. K. (1996). The relationship between social support and physiological processes: A review with emphasis on underlying mechanisms and implications for health. *Psychological Bulletin, 119*(3), 488-531.
- Uchino, B., Cacioppo, J., Malarkey, W., & Glaser, R. (1995). Individual differences in cardiac sympathetic control predict endocrine and immune responses to acute psychological stress. *Journal of Personality and Social Psychology, 69*, 736-743.
- Uchino, B. N., Uno, D., & Holt-Lunstad, J. (1999). Social support, physiological processes, and Health. *Current Directions in Psychological Science, 8*(5), 145-148.
- U.S. Census Bureau. (2007, August). *Income, poverty, and health insurance coverage in the United States: 2006*. Retrieved June 14, 2008, from <http://www.census.gov/>.
- Wadsworth, M. E., Raviv, T., Reinhard, C., Wolff, B., Santiago, C. D., & Schachter, L. (2008). Indirect effects model of the association between poverty and child functioning: The role of children's poverty-related stress. *Journal of Loss and Trauma, 13* (2/3), 156-185.

Weems, C. F., Zakem, A. H., Costa, N. M., Cannon, M. F., & Watts, S. E. (2005).

Psychological response and childhood anxiety: Association with symptoms of anxiety disorders and cognitive bias. *Journal of Clinical Child and Adolescent Psychology, 34(4)*, 712-723.

Wolff, B., Santiago, C. D., & Wadsworth, M. E. (in press). Poverty and involuntary engagement stress responses: Examining the link to anxiety and aggression within low-income families. *Anxiety, Stress, & Coping*.

Table 1

P1 Baseline Reactivity and P2 Experimental Condition Physiological Data Collection Protocols

Epoch	Minutes	Task Type	P1 Task	P2 Task	Citation
1	1-2	Baseline Story	<i>Rainbow Fish</i>	<i>Goodnight Moon</i>	Brown, 1947; Pfister, 1992
2	3-4	Social Stressor	Interview	Interview	GSRST, Carlson, 1985
3	5-6	Cognitive Stressor	Digit recall	Letter recall	Kaufman & Kaufman, 1983
4	7	Physical Stressor	Lime juice	Lemon juice	Kagan & Snidman, 1991
5	8-9	Neutral video	<i>Red Balloon</i>	<i>The Snowman</i>	Lamorisse, 1956; Briggs, 1982
6	10-11	Emotional Stressor	<i>The Land Before Time</i>	<i>Stand By Me</i>	Evans, Gideon, & Scheinman, 1986; Spielberg, 1988
7	12-13	Recovery Story	<i>The Carrot Seed</i>	<i>Rainbow Fish and the Big Blue Whale</i>	Krauss, 1945; Pfister, 1995

Table 2
Means and Standard Deviations of Children's P1 Mean Raw Physiological Reactivity Scores and Mean Overall Difference Scores (P1 Task Mean – P1 Baseline) for the Total Sample

	<i>M</i>	<i>SD</i>
<i>P1 RSA</i>		
Baseline	7.60	1.23
Social Stressor	7.27	1.23
Cognitive Stressor	7.35	1.37
Physical Stressor	7.22	1.27
Neutral Video	7.53	1.18
Emotional Stressor	7.42	1.22
Recovery	7.64	1.50
RSA Difference	-0.27	0.74
<i>P1 RZ</i>		
Baseline	99.24	11.47
Social Stressor	100.40	11.33
Cognitive Stressor	100.11	11.82
Physical Stressor	101.62	11.60
Neutral Video	101.19	12.58
Emotional Stressor	99.53	12.13
Recovery	99.89	12.31
RZ Difference	1.05	3.33

Table 3

Means and Standard Deviations (in parentheses) of Children's P2 Mean Raw Physiological Reactivity Scores and Mean Overall Difference Scores (P2 Task Mean – P1 Baseline) for the Total Sample

	Overall Sample	Support Condition	Control Condition
<i>P2 RSA</i>			
Baseline	7.66 (1.51)	7.97 (1.47)	7.37 (1.52)
Social Stressor	7.50 (1.62)	7.81 (1.59)	7.21 (1.61)
Cognitive Stressor	7.69 (1.50)	7.97 (1.28)	7.42 (1.66)
Physical Stressor	7.60 (1.41)	7.97 (1.27)	7.24 (1.47)
Neutral Video	7.77 (1.35)	8.20 (1.02)	7.34 (1.51)
Emotional Stressor	7.87 (1.21)	8.18 (0.99)	7.56 (1.34)
Recovery	7.68 (1.36)	7.90 (1.02)	7.48 (1.62)
RSA Difference	0.04 (0.90)	0.13 (0.91)	-0.04 (0.91)
<i>P2 RZ</i>			
Baseline	98.12 (12.93)	96.58 (14.29)	99.56 (11.57)
Social Stressor	99.12 (12.30)	98.77 (13.81)	99.45 (10.87)
Cognitive Stressor	99.55 (12.61)	98.83 (15.21)	100.25 (9.67)
Physical Stressor	101.18 (12.10)	99.63 (15.02)	102.68 (8.41)
Neutral Video	100.88 (10.71)	99.14 (12.27)	102.81 (8.46)
Emotional Stressor	101.78 (10.98)	100.55 (12.47)	103.09 (9.19)
Recovery	99.44 (10.53)	98.78 (12.06)	100.17 (8.78)
RZ Difference	0.56 (5.70)	2.05 (5.76)	-0.89 (5.33)

Table 4

Analytic Strategy 1: Regression B Coefficients Predicting CAB Reduction Score from Children's P1 CAB T-scores, Condition, Caregivers' Marital Status, and P1 CAB T-Scores x Condition

	<i>B</i>
<i>Step 1</i>	
P1 CAB T-Scores	0.31*
Condition	0.24*
Caregiver Marital Status	0.27*
R^2	0.33
<i>Step 2</i>	
P1 CAB T-Scores x Condition	0.26 [^]
ΔR^2	0.04

Note. *B* = standardized betas.

* $p < .05$; [^] $p < .10$

Table 5

Analytic Strategy 2: Regression B Coefficients Predicting RCI Reduction Scores from Children's P1 RCI scores, Condition, Child's Sex, and P1 RCI Scores x Condition

	<i>B</i>
<i>Step 1</i>	
P1 RCI Scores	0.44**
Condition	-0.54
Child's Sex	0.01
R^2	0.41
<i>Step 2</i>	
P1 RCI Scores x Condition	0.85 [^]
ΔR^2	0.04

Note. *B* = standardized betas.

** $p < .01$; [^] $p < .10$

Table 6

Analytic Strategy 3: RCI Reduction Scores Means and Standard Deviations Listed by P1 RCI Scores x Condition Groups, and Results of Contrast Analyses (F) Between Each Group and All Other Participants, with Child's Sex as Covariate

	Reciprocal Sympathetic Activation						Coinhibition/Parasympathetic Activation						Coactivation					
	Control (n=6)			Support (n=3)			Control (n=21)			Support (n=24)			Control (n=2)			Support (n=3)		
	M	SD	F	M	SD	F	M	SD	F	M	SD	F	M	SD	F	M	SD	F
Mean RCI Reduction Score (P1 - P2)	0.33	0.52	4.23*	1.00	0.00	22.97***	-0.14	0.36	2.58	0.13	0.34	0.93	-0.50	0.71	4.99*	-0.67	0.58	12.23**

*** $p < .001$; ** $p < .01$; * $p < .05$

Figure 1: Analytic strategy 1 - Interaction of children's baseline CAB score and condition assignment predicts CAB reduction scores.

Figure 2: Analytic strategy 2 - Interaction of children's baseline RCI score and condition assignment predicts RCI reduction scores.

Figure 3: Analytic Strategy 3 – Interaction of children's baseline RCI profile and condition assignment predicts RCI reduction scores.

Figure 1.

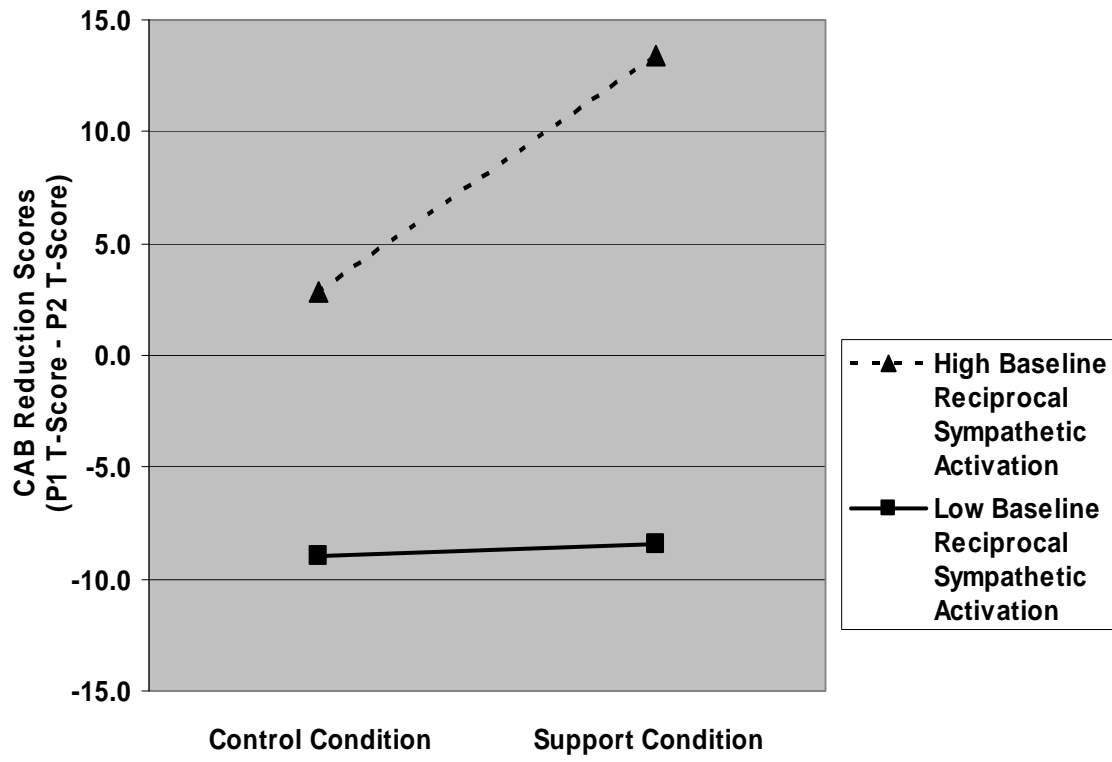


Figure 2.

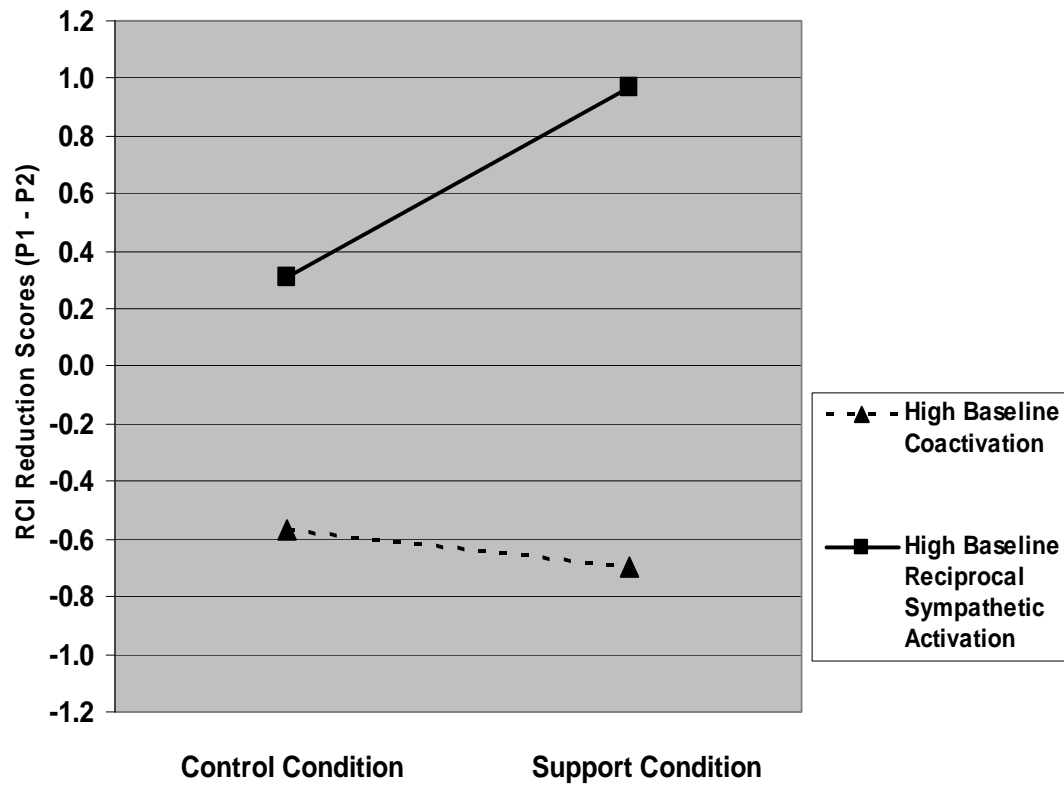


Figure 3.

