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UNIVERSITY OF CALIFORNIA

Los Angeles

Early Adversity, Social Functioning, Mood, and Physical Health:
Developmental and Daily Process Approaches

A dissertation submitted in partial satisfaction
of the requirements for the degree Doctor of Philosophy
in Psychology

by

Elizabeth Brehm Raposa

2015

ABSTRACT OF THE DISSERTATION

Early Adversity, Social Functioning, Mood, and Physical Health:
Developmental and Daily Process Approaches

by

Elizabeth Brehm Raposa

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2015

Professor Constance L. Hammen, Chair

Exposure to early stressful experiences has been associated with a variety of poor health outcomes in adolescence and adulthood (Felitti et al., 1998; Repetti, Taylor, & Seeman, 2002; Springer, Sheridan, Kuo, & Carnes, 2007). The current project was designed to explore specific psychosocial and biological mechanisms through which early adversity might prospectively shape physical health in adulthood. Study 1 used longitudinal data from a community sample to show that cumulative experiences of early adversity, as measured by contemporaneous maternal report, predicted poor self-reported and interviewer-rated physical health in young adults. Results suggested that early adverse experiences led to ongoing stress in social and nonsocial (e.g., academic) contexts, which in turn portended poor health. Elevated depressive symptoms accounted, in large part, for the effects of ongoing social stress on later risk for poor health. Study 2, using a subset of the Study 1 sample, showed that individuals who experienced early

adversity tended to have higher depressive symptoms partially as a result of close friendships with individuals with mental health problems. Study 3 examined the day-to-day dynamics of interpersonal and biological mechanisms of the effects of early adversity on physical health in a sample of college students. Results suggested that young adults who have experienced stressful family environments reported more instances of reassurance-seeking, aggression, and withdrawal on a day-to-day basis. In addition, early adversity predicted higher daily reports of negative affect. Together, these findings support the notion that early adversity can have a long-lasting impact on patterns of psychological, social, and biological functioning, and that early adversity and ongoing stress might contribute to poor physical health in part through their effects on social relationships and mood. Results have implications for the development of targeted interventions designed to prevent the long-term emotional and physical consequences of early life stress.

The dissertation of Elizabeth Brehm Raposa is approved.

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2015

DEDICATION

This dissertation is dedicated to my parents, Mary Ellen and Michael Raposa, my siblings Rosemary and Daniel Raposa, and my partner, Christopher Conway. Thank you for your unwavering love and support.

I also wish to thank my advisor, Dr. Constance Hammen, for her invaluable support and guidance over the past five years. I am extremely grateful to have had the opportunity to learn from you, and your approach to research and your commitment to training and service have served as an inspiration for me throughout my own career.

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ACKNOWLEDGEMENTS

This project was made possible through fellowship support from NIMH training grant MH15750 “Biobehavioral Issues in Mental and Physical Health,” a Psi Chi Graduate Research Grant, and a Dissertation Year Fellowship from the UCLA Graduate Division. Chapter 2 of the dissertation is a version of the published article, Raposa, E. B., Hammen, C. L., Brennan, P. A., O’Callaghan, F., & Najman, J. M. (2013). Early adversity and health outcomes in young adulthood: The role of ongoing stress. *Health Psychology, 33*(5), 410-18. Chapter 3 is a version of the published article, Raposa, E. B., Hammen, C., Brennan, P., & Najman, J. (2014). Characteristics of close friendships in young adulthood as a pathway from early adversity to depressive symptoms. *Journal of Clinical Child and Adolescent Psychology, 28*:1-9.

I wish to thank my doctoral committee chair, co-author, and advisor, Dr. Constance Hammen, for her guidance throughout the process of my dissertation. I would also like to thank my co-authors Dr. Frances O’Callaghan, Dr. Patricia Brennan, and Dr. Jake Najman for their helpful comments on drafts of published articles based on the dissertation, and my doctoral committee members Dr. Julie Bower, Dr. Rena Repetti, and Dr. Teresa Seeman for contributing their valuable time and resources to support me in the process of conducting this dissertation.

Many thanks to my research assistants, Rachel Butler, Joanna David, Leila Hariri, Elle Hennessee, Susette Moyers, and Lily Xuan Gong, for managing the logistics of collecting the data for the dissertation. This project would not have been possible without their help. Finally, I am grateful for the suggestions and encouragement of my labmates and classmates at UCLA.

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- Raposa, E. B., Hammen, C., Bower, J., Brennan, P., & Najman, J. (2014). A developmental pathway from early adversity to inflammation: The role of health behavior. *Psychological Science*, 25(6):1268-1274.
- Raposa, E. B., Hammen, C., Brennan, P., & Najman, J. (2014). Characteristics of close friendships in young adulthood as a pathway from early adversity to depressive symptoms. *Journal of Clinical Child and Adolescent Psychology*, 28:1-9.
- Raposa, E. B., Hammen, C., Brennan, P., & Najman, J. (2014). The long-term effects of maternal depression: Early childhood physical health as a pathway to offspring depression. *Journal of Adolescent Health*, 54, 88-93.
- Raposa, E. B., Hammen, C. L., Brennan, P. A., O'Callaghan, F., & Najman, J. M. (2013). Early adversity and health outcomes in young adulthood: The role of ongoing stress. *Health Psychology*, 33(5), 410-418.
- Raposa, E. B., Hammen, C., & Brennan, P. (2011). Effects of child psychopathology on maternal depression: The mediating role of child-related acute and chronic stressors. *Journal of Abnormal Child Psychology*, 39, 1177-1186.
- Raposa, E. B. & Hammen, C. (May 2015). The impact of early adversity on social experiences and mood: A daily processes approach. Symposium talk presented at the Association for Psychological Science Annual Convention, New York City, NY.
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- Raposa, E., Hammen, C., & Brennan, P. (November 2010). Child psychopathology and maternal depression: The mediating effects of acute stressors. Poster presented at Association for Behavioral and Cognitive Therapies Annual Convention, San Francisco, CA.
- Raposa, E., Holmes, M. K., Fournier, J. C., & DeRubeis, R. J. (November 2009). Therapy processes associated with poor outcomes in patients with comorbid personality disorder in cognitive therapy for depression. Poster presented at Association for Behavioral and Cognitive Therapies Annual Convention, New York, NY.
- Raposa, E., & Gentes, E. (November 2008). Intolerance of uncertainty, coping self efficacy, and the future events schemas of high perseverators. Poster. Proceedings of the Association for Behavioral and Cognitive Therapies Annual Convention, Orlando, FL.

Chapter 1: General Introduction

Early Adversity and Physical Health

Early adverse experiences in childhood, including chaotic or neglectful home environments and experiences of abuse, are widespread phenomena. Approximately one third of adults in community samples endorse experiencing some type of physical, emotional, or sexual abuse or neglect (Scher, Forde, McQuaid, & Stein, 2004). Importantly, individual early adversities often do not occur in isolation, with anywhere from 13% to 51% of adult respondents in large community samples endorsing experiences of more than one adversity, depending on the number of adversities surveyed (Kessler, Davis, & Kendler, 1997; Scher et al., 2004).

Exposure to these early stressful experiences has been associated with a variety of poor health outcomes in adolescence and adulthood (Felitti et al., 1998; Repetti, Taylor, & Seeman, 2002; Springer, Sheridan, Kuo, & Carnes, 2007). These types of negative early environments predict increased inflammation (Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Slopen, Koenen, & Kubzansky, 2012; Taylor, Lehman, Kiefe, & Seeman, 2006) and increased blood pressure (Luecken, 1998), two markers of risk for serious disease and death. Early adversities also predict the onset and course of a number of chronic illnesses marked by severe distress and disability (Davis, Luecken, & Zautra, 2005; Drossman et al., 1995), and early adversity has been linked to higher death rates from chronic disease (Power, Hypponen, & Smith, 2005).

Despite this striking evidence for the negative effects of early adversity on physical health, the specific psychosocial and biological mechanisms by which early adversity affects later health remain unclear. How do various early psychosocial experiences continue to shape children's emotions, thoughts, and behaviors? Moreover, how do these experiences and their consequences "get under the skin" to affect biological outcomes decades later? The goal of the

present set of studies is to explore several mechanisms by which early adversity might prospectively shape physical health in adulthood.

The current understanding of the effects of early adversity on later social relationships, mood, and physical health is reviewed below, and unresolved issues in these areas are discussed. Following this review is a detailed description of three studies designed to address specific questions about the impact of early adversity on health. In particular, the set of three studies will examine whether early adversity predicts continuing stress in adolescence and adulthood, particularly via its negative effects on an individual's ability to select into and promote warm and supportive relationships with peers. Studies will also explore whether individuals who have experienced early adversity evidence increased psychological distress and poorer physical health in response to social stress. Finally, one of the studies will explore whether individuals who have experienced early adversity evidence dysregulation in the hypothalamic-pituitary-adrenal (HPA) axis, a physiological system that has implications for later physical health.

Early Adversity and Susceptibility to Poor Health

Early adversity places children at risk for the development of physical health problems in a number of ways. Adverse early environments have been shown to cause information-processing biases in children, which have important implications for their mood and behavior. For example, early adversity has been linked to increased biases to perceive rejection (Feldman & Downey, 1994) and hostile intent (Dodge, Bates, & Pettit, 1990) in ambiguous social situations. These biases appear to be relatively pervasive, and are associated with maladaptive internal working models of interpersonal relationships in individuals who have experienced early adversity (Muller, Thornback, & Bedi, 2012; Stronach et al., 2011). In addition to having biased perceptions of social interactions, children who have experienced early life stress also show

deficits in their ability to understand, control, and appropriately express emotions, particularly in response to stressful situations (Repetti, Taylor, & Seeman, 2002). As a result, children who have experienced adversity are more likely to react with higher levels of negative emotion, such as hypervigilance and distress, in stressful situations (Davies & Cummings, 1998).

All of these observed deficits in children who have experienced adversity can contribute to behaviors that increase susceptibility to physical health problems. Individuals who have experienced adversity are more likely to use maladaptive coping strategies that involve tension reduction or escape (Johnson & Pandina, 1991; Valentiner, Holahan, & Moos, 1994) when exposed to stressful or conflictual interactions. They also exhibit increased rates of risky health behaviors, including drug and alcohol misuse (Kaufman et al., 2007; Mounts & Steinberg, 1995; Schilling, Aseltine, & Gore, 2007; Widom & White, 1997), cigarette smoking (Doherty & Allen, 1994), risky sexual behavior (Gilbert et al., 2008), and involvement with pregnancy (Scaramella, Conger, Simons, & Whitbeck, 1998). Maladaptive coping and increased rates of risky health behaviors are likely to have direct effects on physiological reactivity and risk for chronic health conditions, such as heart disease.

Aside from their direct effects on health behaviors and physiological functioning, information-processing biases and maladaptive coping behaviors are also likely to contribute to a cascade of ongoing difficulties that in turn increases risk for poor health. That is, exposure to early adverse experiences might make certain individuals prone to experience ongoing stress and mental health difficulties, and these difficulties might in turn compound these individuals' risk for health problems (Repetti et al., 2002). The next section details the literature on associations between early adversity and later experiences of stress, and discusses this pathway to poor physical health as one of the particular questions that will be the focus of the proposed research.

A later section (see *Role of Mood and Depression*) will discuss the ways in which mental health might also be involved in these cascading negative effects of early adversity.

Early Adversity and Ongoing Stress

One important mechanism by which early adversity might influence later physical health involves its contributions to continuing stress exposure. Children who are exposed to early adverse experiences might continue to be exposed to more stressful experiences, such as reduced support in social relationships or financial difficulties, throughout adolescence and young adulthood. This continuity of stress is hypothesized to occur for several reasons. First, experiences of early adversity tend to be rooted in relatively stable structural contexts, such as financial instability or family conflict, which increase the likelihood of later exposure to stress (Pearlin, 1989). In addition, early adversity has lasting effects on children's cognitive and interpersonal styles (Bifulco, Moran, Ball, & Lillie, 2002; Hankin, 2005), as well as on their acquisition of coping behaviors (Johnson & Pandina, 1991; Turner & Lloyd, 1995; Valentiner, Holahan, & Moos). These negative effects of early adversity are likely to influence the types of environments that children select into later in life, and thereby promote ongoing experiences of stress. Finally, the effects of early adversity on children's ability to process and regulate emotions are also likely to increase the probability that individuals will perceive stress and react in ways that escalate stressful circumstances, such as interpersonal conflict (Davies & Cummings, 1998).

In support of the hypothesis of "stress continuity," early adversity has been shown to create a vulnerability to increased chronic stress and episodic stressful events (Hammen, Hazel, Brennan, & Najman, 2012; Hankin, 2005; Hazel, Hammen, Brennan, & Najman, 2008), as well as more daily hassles (Uhrlass & Gibb, 2007), later in life. In addition, exposure to early life

stress has been linked to the occurrence of particular stressful experiences, including poor performance in school and failure to graduate, increased likelihood of arrest, increased likelihood of being fired, and teenage parenthood (Lansford et al., 2007; Pettit, Bates, & Dodge, 1997).

These ongoing stressful experiences have been shown to partially explain the effects of childhood adversity on later mental health, particularly depression, in several recent studies (Hazel et al., 2008; Kessler & Magee, 1994; Turner & Butler, 2003; Uhrlass & Gibb, 2007). For example, in one study of young adults, increases in experiences of daily hassles mediated the relationship between exposure to emotional maltreatment during childhood and later depressive symptoms (Uhrlass & Gibb, 2007). Another study found that elevated levels of chronic stress in relationships with children and spouses in adulthood mediated the relationship between exposure to family violence in childhood and reports of recurrent depression in adulthood (Kessler & Magee, 1994). Two other studies have examined indices of multiple early adversities and have shown that ongoing chronic stress also serves as a mediator of the relationship between cumulative early stress exposure and the onset of depression later in life (Hazel et al., 2008; Turner & Butler, 2003).

Despite recent findings highlighting the impact of stress continuity on later depression, it is unclear whether ongoing experiences of stress might serve as a mechanism of the relationship between early adversity and later physical health outcomes. There is substantial evidence that both social (Cohen et al., 1997; Troxel, Matthews, Gallo, & Kuller, 2005) and non-social stress (Deinzer, Kleineidam, Stiller-Winkler, Idel, & Bachg, 2000; Paik, Toh, Lee, Kim, & Lee, 2000) are associated with indicators of poorer physical health. Both types of stress are thought to impact physical health by triggering biological processes, such as inflammation, associated with negative health outcomes (Avitsur, Powell, Padgett, & Sheridan, 2009; Miller, Rohleder, & Cole,

2009). In addition, stress negatively influences individuals' health behavior, including smoking, drinking, sexual behavior, and exercise and eating patterns (Conway, Vickers, Ward, & Rahe, 1981; Steptoe et al., 1996). Despite these multiple pathways from ongoing stress to poor physical health, few studies have undertaken to examine the role of continuing stress in the relationship between early adversity and later physical health.

Two studies have shown that a particular type of ongoing stress, increased difficulties in social relationships, might play a role in the cascading effects of early stress environments on physical health in adulthood. Taylor and colleagues (2004) reported on a model of the long-term effects of early adversity, which showed that harsh early environments led to increased anxiety, which in turn was indirectly related to poorer physical health through its negative effects on social relations. Similarly, Lehman and colleagues (2005) showed that harsh early family environments led to poorer psychosocial functioning, indicated in part by more negative social interactions and fewer positive social interactions, and that poor psychosocial functioning in turn predicted worse metabolic functioning. These studies indicate that ongoing stress in social relationships might be one pathway by which early stressful experiences lead to poorer physical health. However, models including both ongoing social stress and non-social stress will be needed to determine whether stressful social experiences play a unique role in this link between early adversity and later physical health. In addition, exploration of social stress across a variety of relationships (e.g., friends, family, romantic partners) is needed to better understand what types of stressful social contacts might be most important in explaining the link between early adversity and later physical health.

Early Adversity and Social Behavior

One way in which early adverse environments might shape individuals' relationships and lead to elevated social stress involves the effects of early experiences on social behavior. That is, individuals who have been exposed to early adversity might be more likely to exhibit maladaptive social behaviors, thereby increasing their risk of experiencing social conflict or isolation. There is substantial evidence that harsh early environments, particularly those marked by maltreatment and family conflict, are associated with social impairment in young children. Children who have been exposed to these adverse environments have less interpersonal skill and self-control in interactions with peers (Crockenberg & Lourie, 1996; Fantuzzo et al., 1998; Howes & Espinosa, 1985) and tend to show decreased pro-social behavior in interactions with others (Conaway & Hansen, 1989; Howes & Espinosa, 1985). In examinations of specific social behaviors, children exposed to early adversity tend to show elevated levels of disruptive behavior and aggression (Bolger & Patterson, 2001; Conaway & Hansen, 1989; Hart et al., 1998; Kaufman & Cicchetti, 1989; Kim & Cicchetti, 2010; Teisl & Cicchetti, 2007), as well as increased withdrawal (Kaufman & Cicchetti, 1989), in social interactions.

In studies of aggressive behavior, early stressful contexts are associated with elevated rates of physical, verbal, and relational aggression in children (Bolger & Patterson, 2001; Conaway & Hansen, 1989; Hart et al., 1998). In addition, children who have been maltreated are more likely to be nominated as someone who starts fights by their peers (Kaufman & Cicchetti, 1989; Teisl & Cicchetti, 2007). Aggressive behavior in children who have experienced adversity is hypothesized to occur because chaotic or violent early environments foster the development of cognitive biases that cause individuals to over-estimate the hostile intent of others, and to more frequently access aggressive behavioral responses to others' behavior (Dodge, Bates, & Pettit,

1990). In addition, early stressful home environments and experiences of maltreatment have been associated with deficits in the ability to understand and regulate emotion, which is thought to contribute to elevated rates of disruptive and aggressive behavior in children who have experienced early life stress (Repetti et al., 2002). In a recent study designed to test these hypotheses, children who had experienced physical abuse showed an increased likelihood to misperceive hostility from others, as well as to evidence poorer emotion regulation. Both the bias toward perceiving hostility and poor emotion regulation were shown to partially mediate the relationship between experiences of physical abuse and aggressive behavior in childhood (Teisl & Cicchetti, 2007).

Studies also show a link between exposure to early life adversity and later withdrawal behavior in children. For example, one study found that children who had been maltreated were rated as more likely to isolate themselves or avoid contact with others by camp counselors (Kaufman & Cicchetti, 1989). Withdrawal behavior has been hypothesized to serve the function of reducing exposure to conflict. That is, withdrawal might allow an individual to distance oneself from danger, while also signaling to the attacker that the individual does not want to escalate conflict (Gilbert, 2000; MacLean, 1990). Thus, withdrawal might indicate a child's learned response for coping with harsh or chaotic early environments.

An important question is whether the effects of early adverse experiences on social behavior persist into adulthood. Although most existing studies focus on outcomes in childhood and adolescence, there is some evidence that there are far-reaching effects of early adversity on social behavior, lasting throughout adulthood. Experiences of childhood maltreatment are associated with increased reports of aggressive behavior in adulthood, such as insulting and hitting during conflict resolution with romantic partners (Styron & Janoff-Bulman, 1997),

increased submissive behavior (Celik & Odaci, 2012), and increased likelihood of using disengagement as a strategy for coping with stressful tasks (Luecken, Rodriguez, & Appelhans, 2005). Experimental manipulations of social situations also show maladaptive behavior in young adults who have experienced early life stress. In two such studies, Larkin and colleagues looked at young adults' positive and negative behaviors in response to interpersonal role-plays that involved conflict. During these conflict tasks, young adults who reported experiencing negative family environments were rated as showing more negative verbal (e.g., disagreeing, complaining) and negative non-verbal (e.g., not tracking or turning away) behaviors (Larkin, Frazer, & Semenchuk, 1996; Larkin, Frazer, & Wheat, 2010). Nevertheless, further research is needed to determine whether maladaptive social behaviors, such as aggressive behavior and withdrawal, persist throughout adulthood for individuals who have experienced early adversity.

Early Adversity and Quality of Close Relationships

Given the links between early adversity and certain maladaptive social behaviors, such as aggression and withdrawal, an important question is whether early adverse experiences might portend negative consequences for an individual's ability to develop supportive social relationships. It is possible that elevated levels of maladaptive social behavior might cause children to be rejected by most peers, and to select into relationships with peers who also exhibit maladaptive behaviors. Social impairment, marked by characteristics such as poor social skills, aggression, withdrawal, or excessive reassurance-seeking, has long been associated with rejection by normally functioning peers (Black & Hazen, 1990; Gottman, Gonso, & Rasmussen, 1975; Prinstein, Cheah, Borelli, Simon, & Aikins, 2005). Thus, individuals exhibiting social impairment are likely to have fewer opportunities to associate with socially skillful peers. Moreover, there is also evidence to support the idea that socially impaired individuals might

select into relationships with other individuals who evidence maladaptive social behavior. For example, individuals who exhibit elevated levels of aggression and internalizing symptoms, and endorse more loneliness, are more likely to be friends with peers with similar traits (Ellis & Zabatany, 2007; Mercer & Derosier, 2010; Van Zalk et al., 2010).

It is therefore important to explore whether these processes are also evident in the social development of individuals who have been exposed to early adversity. This is particularly important given some research showing that acceptance by one's peers might counterbalance the negative effects of at least some forms of early adversity (Criss, Pettit, Bates, Dodge, & Lapp, 2002). Evidence suggests that children with a history of maltreatment are less popular with their peers and are more likely to be rejected by others (Bolger & Patterson, 2001; Fantuzzo et al., 1998; Kim & Cicchetti, 2010), at least partially because of their increased aggressive and disruptive behaviors (Bolger & Patterson, 2001). Exposure to early life stress also predicts increased ratings of loneliness in children (Howe & Parke, 2001; Kerns et al., 1996). Less is known about the characteristics of the friends of children who have experienced early adversity. However, children who have experienced a wide variety of early adversities report that the friendships they do have tend to be higher in conflict and betrayal, more critical, and less positive and supportive than other children's friendships (Howe & Parke, 2001; Kerns et al., 1996; McCloskey & Stuewig, 2001).

Research on the negative impact of early adversity on supportive relationships in adulthood has mostly focused on the effects of childhood maltreatment on adult romantic relationships. Individuals who have experienced adversities such as abuse, unpredictable parental involvement, and neglect show worse overall functioning in adult romantic relationships (Hill, Young, & Nord, 1994; McCarthy & Taylor, 1999). Colman and Widom (2004) interviewed over

one thousand adults (average age = 28.7 years), about half of whom had experienced physical abuse, sexual abuse, and/or neglect in childhood according to court and official records.

Childhood abuse and neglect were linked to increased likelihood of adult romantic relationship difficulties, including separation and divorce. In addition, women who had experienced abuse or neglect were less likely to rate their partners as supportive, caring, and open to communication than other women. Another large study ($n = 497$), which examined outcomes for adult women, found that childhood experiences of physical, emotional, and sexual abuse were associated with increased rates of sexual problems in adult romantic relationships. In addition, women who had experienced emotional abuse were more likely to report that their romantic partners were uncaring and over-intrusive (Mullen et al., 1996). Finally, adult women who have experienced parental maltreatment in childhood also endorse having romantic partners who are more violent and aggressive (Ornduff, Kelsey, & O'Leary, 2001) and report having romantic relationships marked by higher levels of quarreling and violence (Brown, Craig, Harris, & Handley, 2008) than other women.

Thus, several large studies indicate that, at least for women, there are long-lasting effects of early adverse experiences on quality and outcome of relationships in adulthood. However, an interesting, but unresolved question is how these poor-quality relationships develop for individuals who have experienced early life stress. That is, studies have not specifically examined whether individuals who have experienced adversity might be rejected by normally functioning peers and select into relationships with peers who exhibit social deficits. Moreover, very little research has explored whether early adversity has an impact on close relationships other than romantic relationships in adulthood.

Predictors of friendship quality might be particularly important to examine during the transition to adulthood. Friends become particularly salient aspects of one's social network throughout adolescence and young adulthood (Parker, Rubin, Erath, Wojslawowicz, & Buskirk, 2006; van Lieshout, Cillessen, & Haselager, 1999), and friendship quality is an important predictor of psychological adjustment during the transitions that mark young adulthood, particularly the transition to college (Buote et al., 2007; Swenson, Nordstrom, & Hiester, 2008). Despite this evidence for the link between quality of friendships and later adjustment, few studies have examined the effects of early adversity on non-romantic, close relationships in adulthood. One study found that adults who report more cumulative early adversity (e.g., parental divorce, parental absence, abuse, neglect) also endorsed more negative aspects of close relationships in mid-adulthood (Ford, Clark, & Stansfeld, 2011), and that parental divorce in particular was associated with having a smaller social network. However, more research is needed to clarify the associations between early adversity and friendship quality, especially during young adulthood.

Social Functioning and Health

Functioning in social relationships in turn has important implications for later physical health. Loneliness and social isolation have been associated with significantly elevated risk for mortality (Berkman & Syme, 1979; House, Landis, & Umberson, 1988), and chronic interpersonal stress has also been associated with numerous indicators of poor physical health, such as inflammation, metabolic syndrome, and increased risk of upper respiratory infection (Cohen et al., 1997; Miller, Rohleder, & Cole, 2009; Troxel, Matthews, Gallo, & Kuller, 2005). Importantly, recent evidence indicates that social stressors might evoke different physiological and behavioral coping responses from those evoked by other types of stress (Dickerson,

Gruenewald, & Kemeny, 2004; Weiner, 1992). That is, although many stressors are likely to exert effects on the body, different stressors are hypothesized to elicit different biological adaptations, depending on the goals or resources that are threatened by the specific type of stressor (Weiner, 1992; Dickerson, Gruenewald, & Kemeny, 2004). Several studies designed to test this hypothesis have suggested that social stress might have a unique impact on physiological systems important for physical health, such as the immune system and the HPA axis.

Dickerson and colleagues (2004) measured cortisol patterns in participants who were asked to perform a stressful speech task and math task either in the presence or absence of an evaluative audience. They found that laboratory stressors that involve social-evaluative threat elicited a stronger cortisol response than laboratory stressors that took place in the absence of an evaluative audience. In a similar study, Dickerson and colleagues (2009) showed that participants who performed these tasks in front of an evaluative audience also had increased inflammatory activity in response to the stressors, as well as impaired regulation of the inflammatory response by the HPA axis, relative to participants in the non-evaluative condition (Dickerson et al., 2009). Consistent with these findings, a meta-analysis of cortisol responses to laboratory stressors found that tasks that contained social-evaluative threat (such as giving a speech in front of an evaluative audience) elicited larger cortisol responses than tasks that did not involve a social-evaluative component (Dickerson & Kemeny, 2004).

Social stress might also have a particularly potent effect on later physical health via its unique relationship with mental health. Stressful life events that are marked by interpersonal content have been found to be especially predictive of later depression relative to other types of stressors (Hammen, 2005; Slavich, O'Donovan, Epel, & Kemeny, 2010). This is important given that depression has been associated with markers of physical health, such as inflammation, as

well as several chronic diseases marked by increased inflammation (Kiecolt-Glaser & Glaser, 2002). The unique effects of social stress on depressed mood therefore could have implications for the effects of social and non-social stress on physical health (see *Role of Mood and Depression* section below for further discussion of this topic). Despite this evidence for the unique effects of social stress on later physical health, few studies have examined social and non-social stress simultaneously but separately. More research is therefore needed on the specific roles of social and non-social domains in the effects of stress on physiological functioning and physical health.

Another limitation in the current literature on the effects of social stress on physical health involves the lack of naturalistic studies examining this question. Research in this area thus far is largely limited to laboratory situations, typically involving a specific type of social evaluative threat that could be highly context-specific. An important question is whether these relationships between social stress and markers of physical functioning are also evident in studies that use a more naturalistic design to examine the impact of reports of stress in daily social interactions on health. There is some evidence that daily stressors in general are linked to daily symptoms of physical distress. For example, in studies of the effects of overall daily stress on physical symptoms, individuals report more symptoms of upper-respiratory infections on days when they endorsed more daily hassles (Baker, 2006), and endorse higher numbers of daily hassles directly preceding a period of increased levels of somatic symptoms (such as headaches and stomachaches; DeLongis, Folkman, & Lazarus, 1988). In addition, a number of studies in clinical populations, such as patients with irritable bowel syndrome (IBS; Levy, Cain, Jarrett, & Heitkemper, 1997), recurrent abdominal pain (RAP; Walker, Garber, Smith, Van Slyke, & Claar,

2001), and sickle-cell disease (Gil et al., 2004), show that symptoms of disease fluctuate with daily stress levels.

However, the question of whether social stress, in particular, might be driving fluctuations in daily physical symptoms remains unanswered, since daily stress studies have infrequently distinguished between social and non-social stressors. In one study that looked only at daily interpersonal stressors, physical symptoms (e.g., dizziness, backaches) were more prevalent on days with increased interpersonal stress (Robbins et al., 1974). This specific link between daily interpersonal stress and physical symptoms has also been found in patients with heart failure (Carels et al., 2004). In addition, there is some evidence that different types of daily social interactions might be associated with fluctuations in ambulatory blood pressure. One study examined adults' ambulatory blood pressure during daily social interactions over the course of three days, and found that blood pressure differed according to the type and quality of social interaction (Holt-Lunstad, Uchino, Smith, Olson-Cerny, & Nealey-Moore, 2003). Interactions with family members were associated with lower blood pressure relative to interactions with non-family members. In addition, interactions with more ambivalent social network members, defined as people toward whom participants felt both high levels of positivity and high levels of negativity, were associated with increased systolic blood pressure. These findings indicate that certain types of daily social interactions might predict fluctuations in physical functioning in healthy adults. Nevertheless, more research is needed to explore the relationships between naturalistic experiences of social stress and physical symptoms. Moreover, it is necessary to explore the physiological mechanisms by which social stressors affect physical functioning, in order to better understand the pathways by which naturalistic stressors impact physical health.

Early Adversity and the HPA Axis

One important pathway by which early adversity and ongoing social stress might compromise later physical health is via chronic activation of the HPA axis (Repetti, Robles, & Reynolds, 2011). The HPA axis is a biological system primarily responsible for the physiological stress response, which prepares the body to respond to threat (Heim & Nemeroff, 2001). When a stressor activates the HPA axis, corticotrophin-releasing factor (CRF) is released from the paraventricular nucleus (PVN) and travels to the anterior pituitary, where it stimulates the production and release of adrenocorticotropin (ACTH). ACTH in turn stimulates the production and secretion of glucocorticoids, in particular cortisol, from the adrenal glands. In addition to being released as an acute response to stressors, basal cortisol levels also follow a regular daily pattern of fluctuation. In healthy individuals, cortisol levels rise sharply immediately before awakening, and then decrease throughout the course of the day (Posener, Schildkraut, Samson, & Schatzberg, 1996).

Glucocorticoid receptors can be found on many different types of cells throughout the body, allowing cortisol to have wide-ranging downstream effects on other bodily systems. Cortisol therefore mediates a number of biological processes, including the trafficking of immune cells and energy metabolism (Raison & Miller, 2003). In addition, cortisol can easily cross the blood-brain barrier and is known to affect multiple brain regions, including the hippocampus, which plays an important role in negative feedback on the HPA axis (Heim & Nemeroff, 2001). As a result of these diverse effects, persistent alterations in the functioning of the HPA axis can have important and widespread implications for later health. Levels of circulating cortisol have been associated with immunosuppressive effects that can lead to poor health (Reiche, Nunes, & Morimoto, 2004). Moreover, disruptions in HPA axis functioning have

been linked to increased coronary calcification (Matthews, Schwartz, Cohen, & Seeman, 2006) and earlier mortality from breast cancer (Abercrombie et al., 2003; Sephton, Sapolsky, Kraemer, & Spiegel, 2000).

Importantly, HPA axis functioning, both in terms of cortisol reactivity to stress and diurnal cortisol rhythm, appears to be sensitive to adverse social experiences early in life. Early studies indicated that prolonged early life stress might lead to heightened levels of cortisol in children, consistent with early theories of the physiological effects of stress (Selye, 1956). Early life stressors such as parental loss, family conflict, harsh discipline, and maltreatment are linked to both increased cortisol reactivity to an acute stressor (Bugental, Martorell, & Barraza, 2002; Luecken & Appelhans, 2006; Wismer Fries, Shirtcliff, & Pollak, 2008) and elevated basal levels of cortisol (Gunnar, Morrison, Chisholm, & Schuder, 2001) in children.

As a result of these findings, one early hypothesis concerning the effects of early life stress on HPA axis functioning posited that early stress causes excessive cortisol release. Moreover, this dysregulation is thought to be self-perpetuating, in that elevated levels of cortisol can damage negative feedback mechanisms in this system, thereby leading to uniform increases in basal cortisol and cortisol reactivity to stress. The theory of allostatic load (McEwen, 1998) suggests that biological systems, such as the HPA axis, immune system, and cardiovascular system, constantly adapt in response to internal and external stressors in order to maintain stability and protect the body in the short-term. However, frequent or prolonged stress can lead to allostatic load, or excessive wear and tear on the body's regulatory systems, due to chronic overactivity or underactivity of these biological systems. Thus, a failure to shut off the stress response in the HPA axis and show a typical decrease in cortisol across the day could lead to damage in systems acted on by cortisol.

In contrast to findings of increased basal cortisol and cortisol reactivity, a compelling body of more recent research has emerged indicating that prolonged exposure to stress can also lead to a pattern of down-regulation in HPA axis functioning, or hypocortisolism (Gunnar & Vasquez, 2001; Heim, Ehlert, & Hellhammer, 2000). Hypocortisolism is characterized by a blunted response to stressful events and a flattened diurnal rhythm of cortisol, with relatively low levels early in the morning that remain low throughout the day. This pattern of hypocortisolism has been found in populations of workers experiencing elevated levels of stress and burnout (Caplan, Cobb, & French, 1979; Pruessner, Hellhammer, & Kirschbaum, 1999), as well as patients diagnosed with stress-related physical disorders, such as fibromyalgia, chronic fatigue, and chronic pain (Heim et al., 2000).

Hypocortisolism has also been found in investigations of the effects of early life stress on the HPA axis. Animal studies indicate that disruptions in typical care by the mother result in abnormalities such as a flatter diurnal cortisol rhythm (Sanchez et al., 2005). Studies of children exposed to early stress show similar disturbances in HPA axis functioning. Children living in orphanages evidence a flattened diurnal cortisol rhythm, with lower early morning cortisol levels that remain constant over the course of the day (Carlson & Earls, 1997; Gunnar, 2000). There is also some evidence of a blunted response to acute stressors in children who have experienced adversities such as neglect or physical abuse (Carpenter et al., 2007; Carpenter, Shattuck, Tyrka, Geraciotti, & Price, 2011).

It is possible that these flat and low diurnal cortisol rhythms are another consequence of allostatic load in the face of frequent or prolonged stress (McEwen, 1998). That is, hypocortisolism in children who have experienced early adversity might represent a shift in the functioning of the HPA axis meant to accommodate chronic activation of this system.

Importantly, a down-regulation in HPA axis functioning in response to stress is expected to be accompanied by a compensatory up-regulation in other allostatic systems, particularly the immune system (McEwen, 1998). Thus, if cortisol secretion is decreased as a result of early adversity, an enhanced immune response, marked by elevated levels of inflammatory cytokines, could be seen in response to stress. This pattern could have significant implications for the physical health of individuals showing hypocortisolism in response to early life stress, given the negative effects of elevated inflammation on health (Yeh & Willerson, 2003).

Despite evidence for both hypercortisolism and hypocortisolism in response to early adversity in children, little research has examined whether the effects of early life adversity on patterns of HPA activity persist into adulthood. A few studies have shown elevated basal levels of cortisol in adults who have experienced childhood adversities such as parental loss and maltreatment (Gonzalez, Jenkins, Steiner, & Fleming, 2009; Nicolson, 2004; Nicolson, Davis, Kruszewski, & Zautra, 2010). Several other studies have shown evidence of hypocortisolism or flatter diurnal cortisol slope in adults who have experienced childhood neglect, physical abuse, and unaffectionate parental relations (Taylor, Karlamangla, Friedman, & Seeman, 2011; van der veegt, van der Ende, Kirschbaum, Verhulst, & Tiemeier, 2009; Weissbecker, Floyd, Dedert, Slamon, & Sephton, 2005). Thus, there is evidence that early adversity can have long-lasting damaging effects on physiological functioning. However, much of the current research on the effects of early adversity on HPA axis functioning in adults has been done in clinical populations, such as women with chronic pain (Nicolson et al., 2010) or women with fibromyalgia (Weissbecker et al., 2005). More research in healthy adult populations is therefore needed to tease apart the long-lasting effects of early adversity versus the effects of mental or physical health problems. Moreover, further investigation of the effects of early adversity on

HPA axis dysfunction in adulthood is needed to determine the specific type of dysfunction and its implications for health.

The Role of Mood and Depression

It is crucial to acknowledge the role of mood, particularly negative affect and depressed mood, in the effects of early adversity and ongoing stress on physical health. Various forms of early adversity have been linked to increased rates of depression (Batten, Aslan, Maciejewski, & Mazure, 2004; Bernet & Stein, 1999; Hazel et al., 2008), and there is a robust relationship between stress and later depression (Hammen, 2005). In addition, experiences of early adversity have been shown to lead to more reports of negative mood on a daily basis, as well as greater fluctuations in daily mood (Wonderlich et al., 2007).

Increased negative affect on a daily basis is thought to be at least partially due to increased mood reactivity to daily stress in individuals who have experienced maltreatment. That is, individuals who have experienced early life adversity might be less able to regulate negative affect in the face of daily hassles or acute stressors. In support of this theory, some research has shown that children who have experienced adverse early environments endorse more negative emotions, including distress, anger, and fear, in response to acute stressors such as parental conflict (Ballard et al., 1993; Davies & Cummings, 1998). In addition, daily interpersonal hassles have been found to be perceived as more stressful and to elicit greater increases in negative affect for adolescents who have experienced maltreatment in a romantic relationship than for adolescents who have not experienced maltreatment (Gallaty & Zimmer-Gembeck, 2008). However, further research is needed to determine whether early adverse environments in childhood predispose individuals to experience greater negative affect in response to stressful experiences on a daily basis, and whether this increased mood reactivity persists into adulthood.

Research has also linked depressed mood with indicators of poor health, such as inflammation and increased morbidity from physical health conditions (Kiecolt-Glaser & Glaser, 2002). Depression likely affects physical health through mechanisms similar to stress. For example, depression is strongly linked to HPA axis dysregulation (Gold et al., 1988; Pariante & Lightman, 2008) and to elevated inflammatory markers (Maes, 2008), both of which have been associated with the physiological response to stress. In a model examining the reciprocal influences of physical and mental health, Wickrama and colleagues (2005) found that early life adversity contributed to the development of physical disorders and internalizing disorders, such as depression, throughout adolescence and young adulthood. However, levels of physical and mental health disorders in adolescence remained correlated even when taking into account the role of family adversity. The authors suggested that these findings indicate that early family adversity contributes to an increased risk for the development of both mental and physical health disorders, but mental and physical health continue to have reciprocal influences on one another that are not due solely to a common association with early adversity.

Thus, increased mood reactivity, and particularly depressed mood, might be important mechanisms in the relationships between early adversity, later stress, and physical health. Nevertheless, research often fails to take into account the impacts of early adversity and ongoing stress on depressed mood when examining markers of health. Further examination of the interrelationships among these variables is therefore needed to better understand the indirect effects of early adversity and stress on physical health, via increases in negative mood.

Methodological Issues in Studies of Early Adversity, Stress, and Health

Measurement of Early Adversity. Previous research has used widely varying approaches to the measurement of early adversity. Many studies have examined the effects of specific types

of abuse, such as physical abuse or sexual abuse (e.g., Teisl & Cicchetti, 2007; Weissbecker et al., 2005). Other studies have focused on outcomes in populations that have experienced severe neglect, such as children who have lived in orphanages (e.g., Gunnar et al., 2001). However, research indicates that most adversities do not occur in isolation, and instead, adversities tend to be highly clustered (Dong et al., 2004; Finkelhor, Ormrod, & Turner, 2007; Green et al., 2010; Scher et al., 2004). For example, a specific adversity, such as physical abuse, is likely to be accompanied by chaotic or disrupted family environments characterized by marital conflict, poor parental mental health, and/or low levels of warmth among family members (Downey & Coyne, 1990; Finkelhor & Baron, 1986; Mullen et al., 1996). As a result, studies that focus on the outcomes associated with a single adversity, such as physical abuse or marital violence, are likely to be over-attributing outcomes to that individual adversity, rather than taking into account a cluster of correlated adversities (Kessler et al., 1997).

Measures that account for the overlap of adversities are therefore likely to be more accurate representations of stressful early life contexts. For example, several studies have used composites of adversity that encompass a wide variety of early experiences, including parental loss or absence, parental mental illness, emotional, physical, and sexual maltreatment, and parental rejection (e.g., Brown et al., 2008; Ford et al., 2011). Other studies have attempted to measure the underlying construct of negative family relationships, rather than specific adversities associated with this construct (Repetti et al., 2002; Taylor et al., 2004). The current project will utilize two measures of adversity that aim to encompass a number of aspects of chaotic home environments. Two studies (Study 1 and Study 2) will use a composite of early adverse experiences consisting of several individual adversities associated with maladaptive family functioning, such as maternal mental illness, parental separation, and financial hardship. This

early adversity composite will be used in accordance with a literature indicating that increases in cumulative early adversity, as measured by composites of a number of adverse events, have implications for physical health (e.g., Dube et al., 2009; Felitti et al., 1998). The third study will utilize a measure of negative family relationships, in accordance with a literature indicating that this construct is associated with a number of early adversities and has negative implications for health (e.g., Repetti et al., 2002; Taylor et al., 2004). In particular, we will examine home environments in which children are exposed to low parental warmth, high conflict, and unpredictable daily experiences.

Measurement of stress. In studies of stress and its effects of physical health, the conceptualization and measurement of stress have been highly diverse, ranging from the psychological construct of perceived stress (e.g., Troxel et al., 2005), to animal models of social conflict (Avitsur et al., 2009), to laboratory tasks of social evaluation (Dickerson et al., 2004). However, few studies have examined the physiological effects of stressors that naturally occur on a regular basis in individuals' daily lives. An important question therefore remains about whether findings concerning the effects of social and non-social stressors on physiological responses in the laboratory might extend to more naturalistic settings. Research in this area is necessary for expanding our understanding of the health implications of daily experiences of stress, and has the potential to provide clinically relevant information. Thus, the current project utilizes more contextualized and naturalistic measures of stress in order to explore the generalizability of laboratory findings. Studies 1 utilizes an assessment of both chronic and acute stress across a number of life domains, taking into account the context in which the stress occurs to create an objective rating of stress. Study 3 involves assessment of naturalistic daily stressors and their effects on daily outcomes. In each of these studies, social stress and non-social stress

are examined separately in order to determine whether they have differential effects on outcomes.

Project Overview

In view of the far-reaching consequences of early adverse experiences on individuals' physical health, more research is needed on the multiple mechanisms by which early life stress continues to shape physical functioning in early adulthood. Much existing research has focused on examining the impact of early life stress on physiological reactivity and social functioning in children. However, key questions remain about whether maladaptive physiological and social functioning exist beyond childhood, and whether they account for the long-term effects of early adversity on later physical health. Moreover, previous studies have relied heavily on retrospective reports of early adversity, and often focus on specific aspects of adversity, such as physical abuse or sexual abuse.

The current dissertation is designed to address these gaps in the literature, using a series of three studies. The first two studies involve secondary analysis of previously collected prospective, longitudinal data uniquely able to address some of these unresolved issues. The third study involves original data collection developed for the dissertation. The three studies examine five primary questions, as detailed below, about the impact of early adverse environments on social functioning, physiological reactivity, and physical health in young adulthood.

1. Does early adversity influence physical health in young adulthood?

Study 1 examines whether cumulative experiences of early adversity, as measured by contemporaneous maternal report, predicts poorer physical health in young adults, using a longitudinal dataset. Study 3 examines whether early adversity, as measured by respondent

retrospective report, predicts increased daily physical symptoms in response to daily stressors in healthy young adults.

2. Does the negative impact of childhood adversity on social functioning extend into adolescence and young adulthood? If so, what types of social dysfunction are present?

Study 1 examines whether a cumulative measure of early adversity predicts increased chronic and acute social stress across family and peer relationships in adolescence. Study 2 investigates whether early adversity might cause individuals to have close friends who show elevated levels of psychopathology in young adulthood. Study 3 will examine whether individuals who have experienced early adversity exhibit certain maladaptive social behaviors (aggression, withdrawal, and reassurance-seeking), as well as fewer positive social behaviors, on a daily basis.

3. How does problematic social functioning during adolescence and young adulthood play a role in the relationship between early adversity and physical health problems?

Study 1 compares social stress and non-social stress as mechanisms of this relationship in a prospective, longitudinal study, using a contextual measure of objective chronic and episodic stress across a number of social and non-social domains. Study 3 examines whether early adversity strengthens the association between daily social stressors and daily physical symptoms, which could lead to chronic physical complaints.

4. How does early adversity affect mood, and what role does negative mood play in the effects of early life stress and social difficulties on later physical health?

Study 1 examines the effects of early adversity on later depression, and tests depression as a mechanism of the effects of early life and ongoing stress on physical health in young adulthood. Study 2 investigates psychopathology in close friends as a specific social pathway by

which early adversity might predict increased rates of depressive symptoms. Finally, Study 3 explores whether early adversity strengthens the association between daily experiences of stress and negative mood. Study 3 also tests whether negative mood accounts for the relationship between daily stress and daily physical symptoms.

5. Does early adversity lead to abnormal diurnal cortisol rhythm in young adulthood?

Study 3 examines the effects of early adversity on diurnal cortisol rhythm in a sample of healthy young adults.

Chapter 2: Early adversity and health outcomes in young adulthood: The role of ongoing stress

As described above, there is striking evidence for the long-term health consequences of exposure to stressful conditions in childhood, but the specific mechanisms of this relationship remain largely unknown. Early adversity appears to create a vulnerability to continuing chronic stress and episodic stressful events later in life (Hammen et al., 2012; Uhrlaas & Gibb, 2007). However, no study to-date has examined social and non-social stress as specific mechanisms of the relationship between early adversity and poor physical health while taking into account the role of depression, which has robust relationships with both stress and health. In addition, previous studies have relied almost exclusively upon retrospective reports of early adversities, and the conceptualization and measurement of stress have been highly diverse.

The current project seeks to address these gaps in the existing literature by examining the effects of early adversity on physical health, and testing ongoing social stress, non-social stress, and youth depression as specific mechanisms of this relationship. Social and non-social stressors have both been shown to have detrimental effects on health (Cohen et al., 1997; Deinzer et al., 2000; Paik et al., 2000; Troxel et al., 2005). These two types of stress are examined simultaneously but separately in the current model given evidence that social stress might have stronger effects on depression (Hammen, 2005) and indicators of poor physical health (e.g., Dickerson et al., 2004). Analyses utilize a longitudinal dataset with contemporaneous assessment of early childhood adversity at multiple timepoints during the first 5 years of the child's life, contextual assessment of levels of social and non-social stress during adolescence, and several measures of physical health in young adulthood, in order to more precisely determine the temporal relationships among variables of interest. In addition, a composite of early adversities

was used in order to better capture the interrelatedness of early childhood adversities, and their cumulative impact on future health.

Hypotheses

1. Consistent with previous findings showing the negative impact of early life stress on physical health (e.g., Felitti et al., 1998; Repetti et al., 2002), children's exposure to adversity by age 5 will predict poorer physical health outcomes (self-rated health, interviewer-rated health, and presence of chronic disease) in young adulthood.
2. Consistent with evidence supporting models of continuity of stress (Hazel et al., 2008; Urhlass & Gibb, 2007), cumulative early adversity by age 5 will predict higher levels of chronic and acute stress across social domains and non-social domains during adolescence. Due to a lack of evidence about specificity in stress continuity, no a priori hypotheses are made about the relative strength of the relationships between early adversity and later stress in social versus non-social domains.
3. Levels of social and non-social stress reported by adolescents will in turn have direct effects on physical health, due to evidence that both types of stress can negatively impact health (Deinzer et al., 2000; Troxel et al., 2005). Both the non-social and social stress composites are also hypothesized to have indirect effects on physical health via depression, consistent with evidence that depression is correlated with physical illness (Frerichs, Aneshensel, Yokopenic, & Clark, 1982; Maes, 2008). However, this path is expected to be stronger for social stressors, given previous research showing that social stressors are particularly potent predictors of later depression (Hammen, 2005; Slavich et al., 2010).

Method

Participants

From a birth cohort study of children's development through age 5 including more than 7,000 children (the Mater-University of Queensland Study of Pregnancy (MUSP); Keeping et al., 1989), 815 mother-child pairs were selected for a follow-up study of children at risk for depressive and other disorders at youth age 15, based on mothers' reports of depressive symptoms on the Delusions-Symptoms States Inventory (DSSI; Bedford & Foulds, 1978) during pregnancy and periodically until child age 5. These families were selected to represent a range of symptom presence, chronicity, and severity of maternal depression, later verified by diagnostic interviews (see Hammen & Brennan, 2001 for further details). From the original sample, 991 families were targeted for inclusion in the follow-up, and 815 consented and were included. The adolescent sample at age 15 was 50.6% male and 49.4% female. The families were largely lower and lower-middle income and predominantly Caucasian (91.4%; 3.6% Asian; 5% other or not reported).

At youth age 20, the mother-child pairs that participated at age 15 were again contacted for follow-up, and 705 (363 females) completed age 20 procedures. Mother-child pairs that participated in the age 20 follow-up did not differ from those who did not participate in terms of youth history of depression by age 15 ($\chi^2(1, n = 705) = 1.33, p = .25$) or mothers' marital status at youth age 15 ($\chi^2(2, n = 791) = .79, p = .48$). Youth who did not participate in age 20 procedures had lower family income at age 15 ($t(782) = 2.39, p < .05$) and were more likely to be male ($\chi^2(1, n = 815) = 8.71, p < .005$).

Procedure

Mothers completed measures at 4 time points during the child's early life (during pregnancy, 3-4 days after the child's birth, 6 months after birth, age 5), and at youth ages 15 and 20. Children completed measures at ages 15 and 20. At time points prior to and including age 5, questionnaires administered to mothers asked about maternal symptomatology, mothers' stress and social experiences, children's health and behaviors, and family demographic information. Interviews and questionnaires administered to mothers and youth at youth ages 15 and 20 asked about mother and child psychopathology, youth chronic and acute stress exposure, and youth physical health. Postgraduate students were trained to appropriately conduct and reliably score interviews for the assessments at youth ages 15 and 20. Participants all gave informed consent (assent) and the institutional review/ethics panels of the University of Queensland, Emory University, and the University of California, Los Angeles approved the research protocol for the ages 15 and 20 follow-ups.

Measures

Early adversity. Five indicators were used to index early life exposure to adversity during the first five years of the child's life, based on information provided in the mother questionnaires at the 4 time points in early childhood (except for maternal psychopathology). These included two binary variables, partner separation or partner change, and maternal Axis I diagnosis in the first five years (scored present/absent). Mothers' history of any Axis I diagnosis (excluding specific phobia) between the child's birth and child age 5 was measured using the Structured Clinical Interview for DSM-IV for lifetime disorders (SCID; First, Spitzer, Gibbon, & Williams, 1995) administered to mothers at youth age 15. The most common diagnoses were major depressive disorder (n = 78), dysthymic disorder (n = 68), and social phobia (n = 34). There were three continuous measures of adversity through age 5: family income based on an average of

maternal ratings of income at early childhood assessments (on a 7-point scale), relationship discord (mean of mothers' reports of relationship satisfaction on the 8-item satisfaction scale of the Dyadic Adjustment Scale (DAS; Spanier, 1976); alphas, .85 to .97), and mothers' stressful life events based on a checklist of 9 interpersonal, health, or occupational problems that occurred over the past 6 months at prenatal and post-natal assessments.

Pearson and point-biserial correlations among individual early adversities and health outcomes are presented in Table 1. A count of the number of adversities for each child was used as a cumulative measure of early childhood adversity, due to the fact that adversities have been found to cluster, and cumulative risk might be an important predictor of physical health. In order to create this composite, each of the continuous measures of early adversity was coded as either present or absent, using the 33rd percentile as the cut-off point. This cut-off point has been used in previous studies and was chosen to balance the need for a sufficient sample size for meaningful analyses with the selection of a reasonably adverse level of severity for each variable (Hazel et al., 2008). Due to the fact that few participants had all 5 adversities, participants with 4 or more adversities were combined to create a composite with a more normal distribution (range 0 to 4).

Stress. Measures of youth stress were derived from semi-structured interviews with adolescents at youth age 15, using the UCLA Life Stress Interview (Hammen, 1991). The life stress interview is a face-to-face semi-structured interview that uses standard questions to probe adolescents' experiences with chronic and acute stress. Chronic stress assessment involved probing each of several domains of functioning, and the interviewer assigned an objective severity rating for the level of chronic stress in each domain. Each domain was rated on a 5-point scale, using behaviorally specific anchor points (with 1 indicating exceptionally good conditions

and 5 indicating extreme adversity). The four domains with social content (romantic relationships, relationship with a best friend, family relationships, and social life) were summed. Similarly, the two domains with non-social content (academic performance and school behavior) were summed. Intraclass correlations for domains in the current sample were social life, .63; close friendship, .76; relationship with family members, .84; romantic relationship, .55; academic performance, .94; school behavior, .88.

Acute stress (life events) was assessed with a contextual approach, with the interviewer eliciting specific information about the nature and circumstances of each acute social or non-social stressor reported by adolescents as occurring in the past 12 months. Interviewers wrote narratives of each event that were presented to a team of raters who were blind to the adolescent's depression status and subjective reactions to the event. Individual acute stressors were judged by the team as having primarily social or non-social content, and were rated by the team for severity, taking into account the context in which the stressor occurred in order to judge the objective level of stress that the event would cause to the average individual. Examples of social stressors included a fight with a best friend or a break-up with a significant other. Examples of non-social stressors included failing an important exam or having one's parents called in for a meeting with the principal. Stress severity was rated on a 5 point scale, with 1 indicating no stress and 5 indicating extremely severe stress. Interrater reliabilities based on independent ratings for 89 cases of both social and non-social stress yielded intraclass correlations of .92 for severity ratings. Severity levels were summed across all events with social content and across all events with non-social content.

Chronic and acute stress totals were combined to create a composite of social stress and a composite of non-social stress in adolescence. Composite measures of acute and chronic stress

have been hypothesized to be more accurate indices of overall stress burden, since acute stressors can often arise out of chronically stressful contexts (Pearlin, 1989). In accordance with previous literature, the chronic and acute social stress variables were standardized and summed to create a measure of total social stress burden (Hazel et al., 2008; Turner, Wheaton, & Lloyd, 1995).

Depression. The presence or absence of youth depression between ages 15 and 20 was assessed at age 20 using the SCID, which covered the past five years since the age 15 assessment. Independent judges' ratings of taped interviews yielded a significant Kappa for depression over the past 5 years (0.89). For the current project, onset of youth depression was coded as present between ages 15 and 20 if youth met criteria for major depression, dysthymia, or depression not otherwise specified at any point during the five-year period.

Physical health. Physical health in young adulthood was measured in three ways. First, the Physical Functioning subscale of the SF-36 Health Survey (Ware, Snow, & Kosinski, 2000), a well-validated self-report measure of health-related quality of life, was administered at the youth age 20 follow-up. The Physical Functioning subscale contains 10 items that evaluate the extent to which a person is limited in the performance of physical activities by their health (Ware, Jr. & Sherbourne, 1992). This subscale has been shown to be one of the best measures of pure physical health out of the SF-36 subscales and is able to predict severity of chronic medical conditions (McHorney, Ware, & Raczek, 1993). The alpha coefficient for these 10 items in the current sample was .92, indicating high internal consistency.

Second, general health functioning over the past 6 months was assessed at age 20 using the Health of Self chronic stress domain of UCLA Life Stress Interview. Using both general questions and specific probes, interviewers determined each youth's general functioning in the health domain, using behaviorally specific anchor points. Behavioral anchors for these ratings

included information relevant to markers of actual disease (e.g., if the participant was overweight, ratings of health were automatically rated .5 points worse). The quality of each youth's health was rated using a 5-point scale, with 1 indicating exceptionally good health and 5 indicating a severe, life-threatening health problem (interrater reliability: .77).

Finally, at age 20 youth completed a checklist to report whether they had one or more of 16 chronic diseases, and chronic illness was scored as present/absent. The most commonly endorsed chronic illnesses were asthma ($n = 73$), migraines ($n = 41$), and eczema ($n = 22$).

Physical health outcomes were validated against other variables in the dataset to attempt to ensure their relevance for actual disease outcomes. Both self-reported physical functioning on the SF-36 and interviewer-ratings of physical health were significantly correlated with age 20 youth reports of somatic symptoms including twitching, feeling dizzy, feeling overtired, aches and pains, headaches, nausea, eye problems, rashes, stomachaches, heart pounding or racing, and numbness or tingling, on the Young Adult Self Report of Child Behavior Check List (all p 's < .05). In addition, both physical health outcomes were found to predict actual healthcare utilization at age 20 (both p 's < .001).

Data Analytic Procedures

In order to evaluate the effects of early adversity on later physical health in the current sample, linear regression analyses were used to examine the prospective effects of early adversity (up to age 5) on Physical Functioning scores and interviewer-rated health at age 20. Logistic regression analysis was used to examine the prospective effect of early adversity on the presence or absence of chronic disease at age 20. The presence/absence of youth childhood chronic illness and youth gender were controlled for in these analyses.

Using a structural equation modeling (SEM) framework, this study then tested the indirect effect of early adversity on physical health via social stress, non-social stress, and depression (see Figure 1). SEM permits the simultaneous examination of multiple indirect pathways from early adversity to young adult health outcomes. Due to univariate and multivariate non-normality of the data, robust maximum likelihood procedures were used to estimate standard errors. Overall model fit was evaluated using several standard fit indices, including the likelihood ratio chi-square test, the comparative fit index (CFI; Bentler, 1990), the root-mean-square error of approximation (RMSEA; Browne & Cudeck, 1993), and the weighted root mean-square residual (WRMR; Muthén & Muthén, 1998-2007). All analyses were carried out in Mplus v5 using the WSLMV estimator to accommodate categorical endogenous variables (Muthén & Muthén, 1998-2007).

Results

Descriptive statistics for all main study variables, as well as Pearson and point-biserial correlations among these variables, are presented in Table 2.

First, we tested the effect of early adversity on physical health in young adulthood, using each of the three health outcome variables. Results of the corresponding regression analyses are presented in Table 3. These analyses revealed that the experience of a greater number of adversities by age 5 predicted significantly worse scores on the Physical Functioning subscale of the SF-36 at age 20, covarying for youth gender and childhood chronic illness. In addition, the number of early adversities by age 5 was marginally significant in predicting worse interviewer-rated health over the past 6 months at age 20, covarying for youth gender and childhood chronic illness. However, the logistic regression analysis revealed that early adversity was not a significant predictor of the presence of chronic disease at age 20.

Second, we evaluated a mediational model that examined the effects of the number of early adversities on social, non-social stress, and the presence of depression in adolescence, as well as the effects of both types of stress on depression and physical health in young adulthood (Figure 1). Social and non-social stress, as well as the two physical health outcomes, were allowed to correlate. Chronic disease was not included as an outcome, given that earlier analyses indicated that the direct association between early adversity and chronic disease was not statistically significant. Gender was covaried for in all stages of the analysis where it was found to be a significant predictor. Standardized beta values are shown in Figure 1.

Fit indices indicated that the model tested provided a good overall fit to the data χ^2 ($df = 4, N = 815$) = 10.42, $p = .03$; CFI = .98; RMSEA = .04 (90% CI .01, .08); WRMR = .56. As hypothesized, early adversity had a significant indirect effect on both self-reported physical functioning ($\beta = .05, p \leq .001$) and interviewer-rated health ($\beta = .09, p < .001$). In addition, most hypothesized direct paths within the model were significant. Early adversity significantly predicted both the social and non-social stress composites, as well as the presence of depression between ages 15 and 20. Social stress in adolescence in turn also predicted the presence of youth depression, but non-social stress did not predict depression. Social stress in adolescence, non-social stress in adolescence, and the presence of youth depression between ages 15 and 20 all had significant direct effects on interviewer-rated health at age 20. In addition, non-social stress in adolescence and youth depression between ages 15 and 20 had significant direct effects on self-reported physical functioning at age 20, although social stress in adolescence did not reach significance as a predictor of self-reported physical functioning ($p = .11$).

Discussion

The present study explored the effects of early childhood adversity on physical health in young adulthood, and examined several pathways involved in these effects: ongoing social stress, non-social stress, and depression in adolescence. Results suggested that early adverse experiences significantly compromise physical health, as measured by the physical functioning subscale of the SF-36 and interviewer ratings of physical health, in young adulthood, a developmental period typically associated with robust health and prior to the development of most chronic diseases. In addition, findings showed that continuity of both social and non-social stress, as well as depression, are important mechanisms of this relationship. Higher levels of social and non-social stress in adolescence, as well as the presence of clinically significant depression between ages 15 and 20, had effects on physical health, and early adversity had a significant indirect effect on both physical health outcomes via these mechanisms.

Results suggesting that early adversity predicts poorer physical functioning and interviewer-rated health in young adulthood are consistent with previous research on the effects of early adversity on later physical health (e.g., Felitti et al., 1998). However, previous studies have relied almost exclusively on retrospective reports of childhood adversity, which tend to have high rates of false negatives and significant measurement error (Hardt & Rutter, 2004). The use of retrospective reporting for early adversity is likely to be especially problematic in studies of adults, given that they must report on experiences from many years earlier. The present project improved upon these designs by using a longitudinal dataset to show that early adversities measured by contemporaneous maternal report during the first five years of the child's life, were predictive of multiple measures of poor physical health in young adulthood.

Past studies of early adversity have also tended to focus on the negative effects of particular adversities, such as physical abuse (e.g., Shaw & Krause, 2002) or socioeconomic status (e.g., Power, Hypponen, & Smith, 2005). However, existing evidence suggests that early adverse experiences often occur in clusters rather than in isolation, and a composite of adversity might therefore more accurately represent the cumulative effects of multiple early adverse contexts (e.g., Evans & Kim, 2007; Green et al., 2010). As a result, the present study built upon past findings by using a measure of cumulative adversity that encompasses a number of different adversities to which a child may be exposed, consistent with previous research on the effects of early adversity on adult mental health (Hazel et al., 2008; Green et al., 2010).

Despite the robust effects of early adversity on self-report and interview measures of physical health, early adversity did not predict the presence of chronic disease by age 20. The fact that physical health was examined in young adulthood likely contributed to this finding, given that most chronic diseases do not develop by age 20. Markers of risk for future disease, such as measures of general health and indicators of inflammation, might therefore be better measures of physical health in young adulthood. In addition, it is possible that covarying for childhood history of chronic illness also played a role in this finding, since the few chronic illnesses that are present in young adulthood might already have developed by age 5 (e.g., asthma). More sensitive measures of fluctuations in the symptom severity of chronic illnesses might therefore be necessary to explore whether early adversity and social stress affect the course and eventual outcomes of childhood chronic illnesses in young adulthood.

Results also provide support for the hypothesis that early adversity predisposes children to experiences of ongoing stress in adolescence, both in social and in non-social domains. The magnitude of the effects of early adversity on social and non-social were essentially the same.

These findings are consistent with theories of stress that emphasize that stressful experiences do not usually occur in isolation. Instead, early adversity often predicts later experiences of stressful life events, resulting for some individuals in continued exposure to stress that can compound the deleterious effects of early adversity (e.g., Hazel et al., 2008). This continuity in stress exposure is thought to occur for several different reasons. First, early adversity and stressful life events are often rooted in stable contexts, such as socioeconomic disadvantage or family dysfunction, that predispose individuals to experience greater stress at other time points as well (Pearlin, 1989). In addition, adversities experienced in early childhood likely interfere with the acquisition of social skills and cognitive schemas, which can in turn affect individuals' selection into certain social and academic environments (Bifulco et al., 2002; Turner & Lloyd, 1995).

Previous work has provided some evidence in support of these theories, showing that early adversity might cause higher levels of overall stress at later time points (e.g., Hazel et al., 2008), and interfere specifically with later social relationships (Malinosky-Rummell & Hansen, 1993; Shaw & Krause, 2002). However, these studies have relied largely upon retrospective self-reports of childhood abuse, as well as self-report measures of stressful events later in life. Moreover, none of these studies has examined the specific effects of early adversity on social and non-social stress simultaneously. The present study therefore expanded upon these previous findings by examining in a single model, contextual, interviewer-rated measures of chronic and acute stress across multiple domains of social and non-social functioning.

Finally, results also showed that there was a significant indirect effect of early adversity on both self-reported physical functioning and interviewer-rated health through social stress, non-social stress, and depression in adolescence. Both stress domains and depression were significantly predicted by early adversity, and each of these variables also had negative effects

on physical health. However, social stress appeared to be having negative physical health effects at least partially through depression, while non-social stress did not predict later depression and instead only had direct effects on physical health. Overall, these path model findings suggest that both social stress and non-social stress in adolescence are important mechanisms by which early adversity has lasting effects on physical health in young adulthood. This is consistent with an existing body of literature showing that stress has important implications for later physical health, and can lead to increased risk for serious illness and death (e.g., Cohen et al., 1997; Troxel et al., 2005). In addition, results suggest that depression also plays an important role in the effects of early adversity and social stress on health. The lack of a significant effect of non-social stress on depression is consistent with a body of evidence suggesting that social stress might be a more potent predictor of later depression than non-social stress (e.g., Hammen, 2005). Non-social stress might therefore have effects on physical health through pathways other than depression.

Limitations and Future Directions

Despite the conceptual and methodological advantages of the current study, several limitations must be acknowledged. First, our measures did not include several common adversity variables, such as neglect, and analyses focused on cumulative risk, rather than examining the relative impacts of specific adversities or adversity clusters. Future studies would benefit from prospective studies of the effects of specific types of adversity using more precise methods, to supplement our focus on cumulative risk. In addition, neither of the two physical health outcomes used in path model analyses is a directly observed physiological marker of disease risk. The physical functioning subscale of the SF-36 has been validated using clinically accepted markers of serious health conditions (Ware et al., 2000), and interviewers used behavioral

anchors related to disease markers, such as weight, in order to make health ratings. Nevertheless, future work will need to examine similar models using biological markers of disease to more precisely determine the effects of early adversity and later stress on physical health conditions.

Present analyses were conducted with a community sample over-selected for the presence of maternal depression (and hence, youth depression). This method had the advantage of emphasizing the effects of stress and depression, but the disadvantage of lack of generalizability to normal populations. That is, maternal depression can have a number of genetic and behavioral influences on offspring that might contribute to their social and academic functioning, as well as physical and mental health. To address the depression issue in the current analyses, early maternal depression was included as a part of the early adversity composite, and youth depression was included in the hypothesized path model. As a result, direct paths from social and non-social stress to physical health represent the unique effects of each stress domain on health, co-varying for the effects of youth depression. Nevertheless, findings still are likely not indicative of what would be found in a truly random community sample. Finally, the current sample was limited in terms of socioeconomic diversity due to the population served by the Mater Hospital, and ethnic diversity due to the Australian general population of that era. As a result, findings should be replicated in more ethnically diverse populations, and in random community samples that are more representative of the general population in terms of socioeconomic status.

Future studies in this area might benefit from comparing different ways in which specific clusters of adversities affect physical health. There are likely differential effects on health for different combinations of contextual stressors and adversities (e.g., Green et al., 2010). In addition, it is possible that acute and chronic stress play different roles in the relationship

between early adversity and physical health, and future studies might investigate more complex models that take this into account. Future research should also examine the biological pathways involved in the effects of early adversity on social functioning and physical health. In particular, markers of inflammatory activity have been studied in relation to both early adversity (Danese et al., 2007; Slopen et al., 2012) and social stress and behavior (Eisenberger, Inagaki, Mashal, & Irwin, 2010; Miller et al., 2009), and inflammation is associated with the development of various serious illnesses.

Finally, small effect sizes in the current findings indicate that the amount of variance accounted for by the hypothesized mechanisms is relatively small, and additional variables not included here are therefore contributing to the outcomes. Future studies should consider models that include potential biological mechanisms, as well as additional psychosocial and environmental factors, such as ongoing financial instability, poor access to healthcare, and risky health behaviors. As noted above, other refined measures of early adversity exposure and objective health outcomes might also yield stronger patterns of relationships. Studies that address these increasingly complex models and identify robust predictors will be needed to translate long-term longitudinal models into clinically relevant targets. Moreover, although the present study represents an advance in our theoretical understanding of the long-term effects of early adversity, it is necessary to further explore earlier indicators of these negative pathways, such as cognitive biases or behavioral tendencies that might lead to stressful social interactions in childhood and early adolescence. Such developmentally informed analyses might help to refine the questions of what interventions at what time points could reduce risk for poor social and physical functioning in adolescence and young adulthood.

Chapter 3: Close friends' psychopathology as a pathway from early adversity to young adulthood depressive symptoms

As noted above, there is a large body of evidence indicating that children who have experienced emotionally and behaviorally disruptive early stress exhibit significant difficulties in interpersonal relationships. These children have been shown to engage in increased rates of problematic social behaviors, such as aggression and withdrawal (Bolger & Patterson, 2001; Kim & Cicchetti, 2010; Teisl & Cicchetti, 2007), and these social skills deficits in turn have been shown to lead to higher rates of peer rejection (Bolger & Patterson, 2001; Fantuzzo et al., 1998). However, it remains relatively unknown what types of friendships individuals who have experienced early life stress do tend to have, and what impact these friendships have on individuals' own emotional and behavioral functioning. Peer rejection might lead to fewer opportunities to form relationships with socially competent peers, making individuals who have experienced adversity more likely to seek out relationships with peers that exhibit similar social deficits, including various internalizing and externalizing symptoms. Little research has examined such friend selection processes for individuals who have experienced early adversity. Children who have experienced early adversity report that they perceive the friendships they do have to be marked by more negative qualities, such as conflict, betrayal, and criticism, and fewer positive qualities, such as supportiveness (Howe & Parke, 2001; Kerns et al., 1996; McCloskey & Stuewig, 2001). However, more research is needed to determine the characteristics of friends selected by individuals who have experienced early adversity.

In addition, it is important to examine whether difficulties in close relationships persist beyond childhood into adolescence and young adulthood. There have been a few studies of adult romantic relationships among those abused as children. In these studies, individuals who were

maltreated in childhood describe their romantic partners as more violent and aggressive (Ornduff et al, 2001), as well as more uncaring and over-intrusive (Mullen et al., 1996). However, further research is needed to determine whether early adversity has a long-term impact on selection into other close relationships beyond childhood. Moreover, current research has largely focused on maltreatment (e.g., physical abuse, sexual abuse) in examining the effects of early adversity on later social functioning. Few studies have examined the long-term effects of a wider range of adversities on characteristics of interpersonal relationships, despite evidence that some adversities, such as maltreatment and parental mental illness, tend to occur in clusters, and cumulative risk might be an important predictor of mental and physical health (Dong et al., 2004; Finkelhor, Ormrod, & Turner, 2007; Green et al., 2010).

Finally, it is important to examine whether the impact of early adversity on friendships might have implications for depression later in life. Stressful interpersonal relationships in general have been identified as a potent risk factor for later depression (Hammen, 2005; Rudolph et al., 2000). Hammen's (1991) model of stress generation suggests that individuals vulnerable to depression tend to contribute to the occurrence of stressors, such that stressful interpersonal contexts and depressive symptoms might have a bi-directional relationship over time. Consistent with models of stress generation, youth at-risk for depression have been found not only contribute to stressful life events, but also to create environments that are dysfunctional and persistently stressful, thus provoking continuing risk for depressive experiences (Hammen, Brennan, & Le Brocque, 2011; Katz, Hammen, & Brennan, 2013; Keenan-Miller, Hammen, & Brennan, 2007). Thus, individuals who have experienced early life stress might be more likely to select close friends who are themselves less capable of providing support and engaging in adequate conflict resolution, thereby contributing to increased risk for depression for the target

individual. This may be particularly true of close friendships during adolescence and young adulthood, when friends become a primary source of advice and support (van Lieshout et al., 1999).

Friends who exhibit problem behaviors, such as externalizing or externalizing symptoms, might also influence youth depressive symptoms through a sort of “contagion effect.” In past research, adolescent friends have been shown to influence each other’s levels of internalizing and externalizing symptoms, both in best friend dyads and larger peer groups (Howes, Hokanson, & Loewenstein, 1985; Prinstein, Meade, & Cohen, 2003; Rosenquist, Fowler, & Christakis, 2010; Stevens & Prinstein, 2005; Van Zalk et al., 2010). Thus, forming problematic friendships with peers who show elevated rates of psychopathology could in turn lead to increased distress for the target individual as a result of the transmission of maladaptive thoughts or behaviors.

The current study therefore sought to examine whether early adversity is associated with increased rates of psychopathology in close friends in young adulthood, and whether these problematic friendships in turn create risk for depression in the target individual. In addition, analyses tested the indirect effects of early adversity on later depressive symptoms, via best friend psychopathology, to determine whether selection into problematic best friendships might serve as a mediator of this relationship, consistent with theories of stress generation. Pathways were examined using a prospective, longitudinal dataset and a large community sample uniquely suited to addressing questions about social functioning and depressive symptoms across development.

Hypotheses

1. Experiences of early life stress (up to age 5) will be associated with higher levels of psychopathology (externalizing symptoms, internalizing symptoms, and personality

pathology) in target youths' best friends in young adulthood. This is consistent with evidence that children who have experienced early adversity are more likely to be rejected by normally functioning peers (Bolger & Patterson, 2001; Fantuzzo et al., 1998; Kim & Cicchetti, 2010). This hypothesis is also consistent with evidence that adults who have experienced early life stress tend to have romantic partners with increased levels of psychopathology, particularly externalizing symptoms (e.g., Ornduff et al., 2001).

2. Greater levels of psychopathology in peers will be associated with increases in depressive symptoms in target youth (controlling for target youths' past depressive symptoms), consistent with evidence that distress within friendships, as well as exposure to elevated levels of psychopathology in peers, are linked to increased rates of depressive symptoms (Hogue & Steinberg, 1995; Van Zalk et al., 2010). We will also explore whether this relationship remains over and above the effects of youth chronic stress on depressive symptoms.
3. Gender was explored as a potential moderator of each of these paths. Previous research has not specifically examined the role of gender in these associations. However, we tentatively hypothesized that females would show stronger associations between best friend psychopathology and depressive symptoms, given that depression becomes more common for females than males in adolescence (Hankin et al., 1998), and female best friends tend to engage in excessive disclosure and co-rumination, which can promote increased contagion of internalizing symptoms (Rose, 2002; Stevens & Prinstein, 2005).

Method

Participants and Procedure

The sample of 815 mother-child pairs followed from birth to youth age 15 described above were used for the current analyses. Of the 815 adolescents that participated at age 15, 527 adolescents (65%) nominated a peer to fill out questionnaires at the age 20 assessment. Of these 527 adolescents who nominated a peer to fill out questionnaires at the age 20 assessment, 252 (47.8%) nominated a romantic partner and 265 (50.3%) nominated a best friend. The remaining 10 participants nominated a sibling. Only characteristics of best friends were examined for the current study. Adolescents who nominated a best friend instead of a romantic partner were significantly less likely to be in a romantic relationship ($\chi^2(1, 517) = 256.98, p < .001$) and were more likely to be male ($\chi^2(1, 517) = 5.98, p < .05$). However, there were no significant differences between adolescents who nominated a best friend and adolescents who nominated a romantic partner in terms of maternal depression history ($\chi^2(1, 517) = 3.03, p = .08$), severity of peers' internalizing symptoms ($t(515) = .87, p = .38$), severity of peers' externalizing symptoms ($t(515) = .16, p = .87$), or severity of peers' personality pathology symptoms ($t(514) = .79, p = .43$).

When youth were ages 22-25, participants were contacted for a final follow-up to complete additional questionnaires. Of the 265 adolescents with friend information at the age 20 assessment, 175 participated in the age 22 assessment. The 175 adolescents with friend data included in the age 22 sample did not differ from the original sample (of 815 adolescents at age 15) in terms of maternal depression history ($\chi^2(1, 815) = 2.70, p = .10$), gender ($\chi^2(1, 815) = 1.67, p = .20$), youth experiences of early adversity by age 5 ($t(814) = 1.05, p = .30$), youth

depressive symptoms at age 15 ($t(803) = .50, p = .62$), youth externalizing symptoms at age 15 ($t(792) = -1.55, p = .12$), or youth total chronic stress at age 20 ($t(703) = .76, p = .45$).

Measures

Early adversity. A latent factor of early adversity was created using the five indicators of stressful environments obtained by maternal report during the first five years of the child's life described above: parental separation or maternal partner change, maternal Axis I diagnosis in the first 5 years, financial hardship, parental discord, and mothers' stressful life events. All models were also run with an alternative composite variable of early adversity, which was created by standardizing continuous early adversities and summing across adversities. Models using this early adversity composite variable showed identical fit and patterns of significance to models using the latent variable of early adversity.

Friend psychopathology. Psychopathology in the best friends of target youth at age 20 was measured using a latent variable indicated by three measures: internalizing symptoms, externalizing symptoms, and personality pathology. Friends' internalizing and externalizing symptoms were assessed using the Young Adult Self-Report (YASR; Achenbach, 1997), a well-validated self-report questionnaire designed to measure emotional and behavioral problems in young adults. Respondents rate how true various symptom descriptors are of themselves, ranging from 0 (not at all true) to 2 (very true or often true). The current project utilized the sum of the Anxious/Depressed and Withdrawn subscales as a measure of friends' internalizing symptoms, and a sum of the Intrusive, Aggressive, and Delinquent subscales as a measure of externalizing symptoms.

Friends' personality pathology was measured using the Personality Diagnostic Questionnaire (PDQ; Hyler et al., 1988), a self-report questionnaire derived from the personality

disorders section of the DSM-III. Each item on the PDQ is derived from the DSM-III diagnosis for a particular personality disorder and has been subjected to content analysis to ensure its face validity and consistency with DSM criteria. The PDQ has been shown to be a good indicator of overall personality disturbance, and distinguishes between individuals with high and low likelihood of personality disturbance, based on comparisons with clinician assessments of patients with and without personality disorder (Hyler et al., 1988). In the current project, the total index score of overall personality pathology was used.

Youth depressive symptoms. Target youth depressive symptoms at ages 20 and 22-25 were assessed using the Beck Depression Inventory—II (BDI-II; Beck, Steer, & Brown, 1996), a well-validated, self-report questionnaire. Coefficient alphas in the current sample were .93 for age 20 and .94 for ages 22-25.

Youth chronic stress. Target youth experiences with chronic stress across a number of domains were measured at age 20 using the UCLA Life Stress Interview, a semi-structured interview described above (Hammen & Brennan, 2001). The age 20 version of the interview probes several developmentally appropriate domains: social life, close friendship, romantic relationships, family relationships, financial difficulties, work problems, academic problems, health of self, and health of close family. For each domain, trained advanced graduate student interviewers used standard probes and semi-structured follow-up queries to make an objective rating of chronic stress on a 5-point scale (from “1” superior/exceptional functioning to “5” severe difficulties, using behaviorally anchored descriptors). Total chronic stress levels were computed by summing across all domains. Evidence of the convergent and predictive validity of the UCLA Life Stress Interview in the current sample is reported in Hammen, Brennan, &

Keenan-Miller (2008). In the current sample, the mean intraclass correlation across all domains at age 20 was $r = .81$.

Data Analytic Procedures

A structural equation modeling (SEM) framework was used to test the effects of early adversity on best friend psychopathology, as well as the effects of best friend psychopathology on youth depressive symptoms (see Figure 2). Follow-up analyses explored the unique role of each component of best friend psychopathology (internalizing symptoms, externalizing symptoms, and personality pathology) in the model. A second model, identical to the first, but also including youth chronic stress at age 20 as a covariate in predicting target youth depressive symptoms, was then tested. In all models, youth depressive symptoms at age 20 were included as a second pathway from early adversity to youth depressive symptoms at ages 22 to 25, in order to control for the effects of continuity of depression in target youth. Gender was controlled for in all paths of these models, due to increased rates of depression in females across adolescence and adulthood (Hankin et al., 1998). Finally, the potential role of gender as a moderator of the first model was explored using multiple group analyses.

All analyses were carried out in Mplus v5 (Muthén & Muthén, 1998-2007), using full information maximum likelihood methods to accommodate missing data. Due to univariate and multivariate non-normality of the data, robust maximum likelihood procedures were used to estimate standard errors. Overall model fit was evaluated using several standard fit indices, including the likelihood ratio chi-square test, the comparative fit index (CFI; Bentler, 1990), the root-mean-square error of approximation (RMSEA; Browne & Cudeck, 1993), and the standardized root mean-square residual (SRMR; Hu & Bentler, 1998).

Results

Descriptive statistics for all main study variables, as well as Pearson correlations among these variables, are presented in Table 4. Both covariates, youth depressive symptoms at age 20 ($r = .55, p < .001$) and youth chronic stress at age 20 ($r = .32, p < .001$), were significantly correlated with youth depressive symptoms at ages 22-25.

Results from the SEM analyses, including standardized beta values, are presented in Figure 2. Fit indices indicated that overall the hypothesized model provided a good fit to the data χ^2 ($df = 40, N = 816$) = 70.41, $p < .01$; CFI = .97; RMSEA = .03 (90% CI .02, .04); SRMR = .04. Factor loadings for parental separation ($\beta = .75, p < .001$), maternal psychopathology ($\beta = .41, p < .001$), parental discord ($\beta = .59, p < .001$), maternal stress ($\beta = .56, p < .001$), and financial hardship ($\beta = .60, p < .001$) provided evidence that these variables were indicators of a single latent factor of early adversity. Factor loadings for peer internalizing symptoms ($\beta = .76, p < .001$), externalizing symptoms ($\beta = .66, p < .001$), and personality pathology ($\beta = .94, p < .001$) provided evidence that these variables were indicators of a single latent factor of peer psychopathology. Early adverse experiences by age 5 had a significant indirect effect on depressive symptoms at ages 22 to 25, via depressive symptoms at age 20 ($\beta = .12, p < .001$), and the indirect path from early adverse experiences by age 5 to depressive symptoms at ages 22 to 25 via best friend psychopathology was marginally significant ($\beta = .04, p = .06$).

In addition, all hypothesized direct paths in the model were found to be significant. Experiences of early adversity by age 5 predicted having a best friend with higher levels of psychopathology at age 20, over and above the effects of gender ($\beta = .26, p \leq .001$). Having a best friend with elevated rates of psychopathology at age 20 in turn predicted higher depressive

symptoms for the target individual over the next two to five years, controlling for gender and target depressive symptoms at age 20 ($\beta = .17, p < .01$).

Additional exploratory analyses examined the roles of the individual components of best friend psychopathology in the model. Best friend internalizing symptoms (χ^2 (df = 24, $N = 816$) = 46.39, $p < .01$; CFI = .96; RMSEA = .03 (90% CI .02, .05); SRMR = .04; indirect path: $\beta = .04, p = .08$) and personality pathology (χ^2 (df = 24, $N = 816$) = 49.92, $p < .01$; CFI = .96; RMSEA = .04 (90% CI .02, .05); SRMR = .04; indirect path: $\beta = .04, p = .06$) served as marginally significant pathways from early adverse experiences to later youth depressive symptoms, with these models showing similar fit and patterns of significance as the latent factor of best friend psychopathology. In contrast, best friend externalizing symptoms did not have a unique direct effect on youth depressive symptoms at ages 22 to 25 ($\beta = .03, p = .67$).

A second model was then run controlling for youth chronic stress at age 20 in predicting youth depressive symptoms at ages 22-25 from best friend psychopathology. Importantly, results showed that higher rates of psychopathology in best friends continued to significantly predict target youth depressive symptoms over the next two to five years ($\beta = .16, p < .05$), even though youth chronic stress was also a significant predictor of later depressive symptoms ($\beta = .11, p < .05$).

Finally, the potential role of gender as a moderator of the effects of early adversity on friend psychopathology, as well as the effects of friend psychopathology on increases in depressive symptoms, was examined. Multiple group analyses suggested that neither the effects of early adversity on friend psychopathology (Wald χ^2 (1) = .86, $p = .35$) nor the effects of best friend psychopathology on later youth depressive symptoms (Wald χ^2 (1) = .06, $p = .81$) differed significantly by gender.

Discussion

The current study used a prospective, longitudinal dataset in order to explore the long-term effects of early adversity on characteristics of individuals' best friends in young adulthood, and the implications of these best friendships for young adults' own depressive symptoms. Findings indicate that individuals who have been exposed to a cluster of adverse early life experiences, including maternal psychopathology and a number of associated stressful conditions, have best friends with elevated levels of psychopathology in young adulthood. Having best friends with elevated rates of psychopathology, particularly internalizing and personality pathology, in turn predicts higher depressive symptoms for target individuals over the next two to five years.

These results highlight that individuals who have experienced early adversity, especially this cluster of early stressors associated with maternal psychopathology, might be more likely to select into best friendships with more psychologically distressed others in young adulthood. A large body of evidence suggests that individuals who have experienced early adversity show increased social difficulties in interactions with peers, particularly in childhood. Children who have experienced early life stress are more likely to exhibit problematic behaviors in social interactions with peers (e.g., Bolger & Patterson, 2001; Teisl & Cicchetti, 2007) and to rate their friendships as higher in negative qualities and lower in positive qualities (e.g., McCloskey & Stuewig, 2001). However, little research has examined whether these social difficulties also exist in adulthood. The current findings therefore address current gaps in the literature by showing that individuals who have experienced early adversity have involvement with peers suffering from psychological distress throughout adolescence and young adulthood.

The specific mechanisms that accounted for individuals' selection into friendships marked by higher psychopathology in the current study were not able to be tested. However, it might be speculated that individuals exposed to early adversity continue to show interpersonal dysfunction in close relationships for several reasons. First, these individuals are more likely to exhibit problematic social behaviors that lead to rejection by normally functioning peers (Bolger & Patterson, 2001; Fantuzzo et al., 1998). Thus, it is possible that individuals who have experienced adversity and have subsequent social difficulties tend to be de-selected by normally functioning peers and then select into friendships with problematic peers. In addition, similarity-attraction theory suggests that individuals tend to select friends who have similar attitudes and behaviors, because this facilitates communication and understanding (Berger & Calabrese, 1975; Byrne & Nelson, 1965). As a result, individuals who have experienced adversity might be more likely to struggle with problematic social behaviors, such as aggression or withdrawal, and in turn select friends who exhibit similar behaviors. Finally, individuals who have experienced early adversity might be more likely to suffer from higher levels of psychopathology themselves, and friends' rates of internalizing and externalizing symptoms might become more similar over time as a result of influence processes (Howes et al., 1985; Rosenquist et al., 2010; Stevens & Prinstein, 2005). That is, individuals who have been exposed to early adversity might exhibit more internalizing and externalizing symptoms that over time increase their friends' levels of psychopathology. Further research is needed to explore these specific mechanisms by which experiences of early adversity might lead individuals to become involved in problematic friendships.

The current results also showed that close friends' elevated rates of psychopathology in young adulthood were associated with higher levels of depressive symptoms in target individuals

over the next five years, controlling for past youth depressive symptoms and youth chronic stress. In addition, the indirect effect of early adversity on later youth depressive symptoms was marginally significant, providing tentative support for best friend psychopathology as one pathway for the effects of early adversity on later depression. This finding is consistent with the fact that friendships with peers become increasingly salient during adolescence and young adulthood (Brown, 1990; Parker et al., 2006). Because close friends become a primary source of social and emotional support in young adulthood, psychopathology within friendships can have a significant impact on mental health. Results are also consistent with theories of stress generation, which posits that individuals at-risk for depression, or other psychopathology, as a result of early adverse conditions, might create or select themselves into potentially problematic circumstances that provoke future depression (Hammen, Brennan, & LeBrocque, 2011; Katz, Hammen, & Brennan, 2013).

However, it is unclear from the present findings exactly why friends' psychopathology might be related to depressive symptomatology. Interestingly, youth chronic stress across a number of domains (including social ones) did not fully account for the relationship between friend psychopathology and depressive symptoms. Thus, although friends' psychopathology might lead to increased conflict or decreased support in close friendships, it appears that other factors are also playing a role in predicting depression in the target individual. It is possible that processes within close friendships, such as co-rumination, might lead internalizing symptoms to be "contagious." This contagion hypothesis is consistent with the fact that exploratory analyses showed peer internalizing symptoms were predictive of target youth depressive symptoms, while peer externalizing symptoms were not. It is less clear why peer personality pathology was also uniquely predictive of target depressive symptoms, but it is possible that depression-like

personality symptoms, such as social isolation or suicidality, could account for this finding. Third variables that affect both friends, such as a stressful school or neighborhood context, might also contribute to psychopathology in both the target individual and his/her friend. Finally, it is possible that early adverse experiences lead to affect dysregulation and behavioral problems in the target individual that contribute both to selecting friends with psychopathology and increasing depression over time.

Gender moderation analyses indicated that neither path in the model was significantly different for males versus females. These results are somewhat surprising, given that reciprocity in close relationships tends to be more valued for adolescent girls than boys (Parker & Asher, 1993), and female best friends are more likely to engage in excessive disclosure and co-rumination, which can promote increased contagion of internalizing symptoms (Rose, 2002; Stevens & Prinstein, 2005). Moreover, depressive symptoms tend to increase more dramatically for females than for males throughout adolescence (Nolen-Hoeksema & Girgus, 1994). Thus, future research should further examine the specific mechanisms by which gender might moderate peer selection and/or socialization, as well as the impact of qualities of best friendships on individuals' emotional functioning.

Limitations and Implications

Several limitations of the current study should be noted. First, the current dataset did not allow us to explore the trajectory of psychopathology in both target individuals and their best friends over time. In addition, the current sample suffered from a relatively high rate of attrition, given the longitudinal nature of the study. Although completers and dropouts did not differ on major variables of relevance, it is possible that variables not measured in the current study might have been correlated with attrition. For example, individuals without close peer relationships

might have been more likely to drop out before the age 20 assessment involving peer questionnaires. The present study also utilized a community sample that was over-selected for maternal depression. This selection process provided some unique advantages for our analyses. First, we were better able to examine the presence of maternal psychopathology as an early adverse environment. Second, this sample gave us insight into predictors of elevated depressive symptoms in offspring, a common problem in offspring of depressed mothers. Nevertheless, future research should examine the effects of early adversities on young adults' friendships and depressive symptoms in other samples that are more representative of the general population. Finally, the current sample was also limited in terms of both ethnic and socioeconomic diversity due to the population served by the Mater Hospital, as well as the Australian general population of that era (born in 1980's). As a result, findings should be interpreted in light of these limitations, and should be replicated in more ethnically and socioeconomically diverse populations.

Despite these limitations, the present study highlights selection into close friendships with youth who have elevated levels of psychopathology as one pathway by which exposure to early stressful conditions might have long-term effects on youth mental health. Findings indicate that for individuals who have experienced maternal psychopathology and other related early adversities, social difficulties can persist far beyond childhood and influence the course of later depressive symptoms. These results have important implications for clinical interventions designed to prevent the development of depressive symptoms. For example, psychosocial interventions that target functioning in close friendships during the transition to adulthood might be particularly important for individuals at risk for depressive symptoms due to experiences of early adversity.

Future research might benefit from exploring other aspects of friendships, such as closeness, conflict, and communication styles, as potential mechanisms of the effects of early adversity on risk for depression. In addition, it will be important for future work to consider potential moderators of the effects of early adversity on functioning in close friendships. For example, a close relationship with a parent or other family member early in life might buffer the negative effects of early adversity on later social functioning. Identifying these mechanisms and moderators of the effects of early adversity on later relationships with peers could in turn aid in the development of more targeted interventions to prevent the development of depression in at-risk youth.

Chapter 4: Implications of risky early family environments for social experiences and health:

A daily processes approach

As discussed above, early adversity is associated with maladaptive social behaviors in children, particularly aggression (e.g., Teisl & Cicchetti, 2007) and withdrawal (e.g., Kaufman & Cicchetti, 1989). Moreover, there is some evidence that the negative effects of early adverse experiences on social behavior might persist into adulthood, with adults who have experienced early adversity reporting elevated rates of aggressive behavior, such as insulting and hitting (Styron & Janoff-Bulman, 1997), as well as increased withdrawal behavior, such as disengagement and avoidance in response to conflict (Luecken et al., 2005; Valentiner et al., 1994). However, most of the existing studies of social functioning in adults who have experienced early life stress have focused specifically on the effects of sexual abuse on behavior in romantic relationships. No study to-date has examined the effects of a broader range of early adversities on levels of aggressive and withdrawal behavior across multiple relationships in adulthood, nor has research examined daily patterns of interactions. Thus, the first objective in the current study was to capture day-to-day variability in social behaviors in healthy young adults as a function of experiences of early adversity.

A second objective of the current study was to examine daily affective and physical reactivity to social stress, as well as the effects of early adversity on these two types of stress reactivity. There is evidence that daily stress leads to increased daily physical complaints (Carels et al., 2004; Holt-Lunstad et al., 2003; Robbins et al., 1974) and negative affect (Bolger, DeLongis, Kessler, & Schilling, 1989; Mroczek & Alemda, 2004). However, most studies have examined the effects of overall daily stress on physical and emotional outcomes, rather than examining the specific effects of social stressors (versus non-social stressors). Moreover, early

life stress has not yet been examined as a specific moderator of the associations between daily stress and these outcomes. Experiences of early life stress have been associated with impaired emotion regulation, as well as increased negative affect in response to stressors (Davies & Cummings, 1998; Repetti et al., 2002). Thus, an important remaining question is whether individuals who have experienced early adversity might have greater negative affect and physical complaints in response to social stressors on a daily basis. Importantly, since daily mood and daily physical symptoms will both be assessed in the current project, analyses will be able to explore whether daily associations between social stress and physical symptoms are present even when negative affect is covaried for. Previous studies examining stress and daily physical symptoms have typically measured subjective reports of physical health without measuring negative affect, which also tends to increase in response to stress and could account for elevated rates of somatic symptoms.

A final objective of the current study was to examine the effects of early adversity on cortisol diurnal rhythm. Early adversity has been shown to result in disruption of the diurnal rhythm of cortisol during childhood (Dozier et al., 2006; Gunnar & Vasquez, 2001), but it is unclear whether this physiological dysregulation persists beyond childhood and adolescence, and whether it exists in otherwise healthy individuals. In accordance with studies showing hypocortisolism in children who have experienced early adversity (Carlson & Earls, 1997; Gunnar, 2000), the current project therefore examined whether early adversity leads to a flattened diurnal rhythm of cortisol in a population of healthy young adults.

A daily diary format was used, given that it is uniquely suited to answering the above questions. This method allowed for assessment of young adults' naturally occurring social behavior, in contrast to questionnaires, which require participants to make generalizations about

their social behaviors over a longer retrospective period and variable social contexts. Moreover, the daily diary method is an idiographic approach that allows one to examine whether individual traits, such as experiences of early adversity, influence the relationship between two within-subjects variables, such as daily stress and daily negative mood.

Hypotheses

1. First, early adversity will be associated with higher daily withdrawal behaviors, consistent with previous findings that children and young adults who have experienced early adversity receive higher withdrawn behavior ratings (Kaufman & Cicchetti, 1989) and report using more disengagement and avoidance to cope with conflict (Luecken et al., 2005; Valentiner et al., 1994). Second, early adversity will be related to higher daily aggressive behaviors, consistent with evidence that early adversity is linked to increased aggression in children and adults (Conaway & Hansen, 1989; Kim & Cicchetti, 2010; Styron & Janoff-Bulman, 1997; Trickett & McBride-Chang, 1995). Third, early adversity will predict lower rates of positive social behavior, consistent with findings that children who have experienced early life stress show decreased pro-social behavior in interactions with peers (Conaway & Hansen, 1989; Howes & Espinosa, 1985). Finally, early adversity will predict increased reassurance-seeking behavior. This is a tentative hypothesis based on findings that multiple types of early adverse experiences lead to insecure attachment (McCarthy & Taylor, 1999; Styron & Janoff-Bulman, 1997), a construct that has been associated with excessive reassurance-seeking behavior (Shaver, Schachner, & Mikulincer, 2005).
2. Levels of daily social stress will predict increased same-day levels of physical symptoms, consistent with prior work on daily associations between stress and physical health (e.g.,

Baker, 2006; Holt-Lunstad et al., 2003). It is unclear whether experiences of early adversity might moderate these associations, although some work indicates that negative affect and self-esteem, two psychological constructs associated with past adversity (Egeland & Sroufe, 2006; Kaufman & Cicchetti, 1989; Mullen et al., 1996), can moderate the relationship between daily stress and physical symptoms (DeLongis, Folkman, & Lazarus, 1988; Walker, Garber, Smith, Van Slyke, & Claar, 2001). Finally, it is expected that the relationship between daily stress and physical symptoms will be attenuated when accounting for daily negative affect. This is consistent with a body of literature showing a strong link between overall mood and physical health, and some evidence indicating that daily negative affect might be associated with risk for minor illnesses, such as colds (Evans & Edgerton, 2011).

3. Levels of daily social stress will predict increased same-day levels of negative affect, consistent with prior work on daily associations between stress and mood (e.g., Bolger et al., 1989; DeLongis, Folkman, & Lazarus, 1988). In addition, experiences of early adversity are hypothesized to moderate these associations, given evidence that early adversity leads to greater fluctuations in daily mood (Wonderlich et al., 2007) as well as more negative emotion in response to stress (e.g., Ballard et al., 1993; Davies & Cummings, 1998).
4. Experiences of early adversity will be associated with abnormal cortisol diurnal rhythm. Although research on the effects of early adversity on diurnal cortisol levels in healthy adults is relatively scarce, and findings in children are inconsistent, early adversity is hypothesized to be associated with a flattened diurnal cortisol rhythm, marked by a lower cortisol awakening response, and a less steep decline in cortisol levels over the course of

the day. This is consistent with previous evidence indicating that repeated early life stress might lead to hypocortisolism in children and adolescents (Gunnar & Vasquez, 2001; Taylor et al., 2011).

Method

Participants

Participants were 131 UCLA undergraduates (65% female) enrolled in psychology courses and offered course credit for completion of the study. This sample size has been shown in previous studies to be sufficient to detect daily relationships among stress, mood, and health (e.g., Baker, 2006; Holt-Lunstad et al., 2003). In addition, this sample size was chosen to balance statistical power with logistical considerations, such as funding for cortisol assays and the anticipated number of undergraduate students meeting the pre-screening criteria for the study. The sample was ethnically diverse, (44% Asian, 29% Caucasian, 15% Latino, 5% African American, 7% Multiracial), as well as socioeconomically diverse (average income was "\$50,000 to \$99,999;" see Table 5 for a full summary of demographic variables).

Prior to enrollment in the study, potential participants attended a pre-screening session, during which they completed the Risky Families Questionnaire (RFQ), a well-validated questionnaire designed to assess perceived levels of conflict and parental warmth in family environments (Taylor et al., 2004), and they also answered additional questions about their physical and mental health (see Appendix A for pre-screening items). Participants who scored higher than 2.5 out of 5 on the RFQ were preferentially recruited in order to obtain a sample that represented a range of early adversity scores. Participants who endorsed experiences of sexual abuse, past or present diagnosis of Posttraumatic Stress Disorder (PTSD), a major medical or health problem (e.g., asthma, diabetes), steroid medication use, or BMI \geq 30 were excluded from

the study. The average score on the RFQ for the sample was 2.11 out of 5 (SD = .69), which is a level comparable to that of other studies that have used college student samples (Edge, Ramel, Drabant, & Kuo, 2009; Winer, 2013).

Two participants did not complete daily diary surveys. Seventeen participants were excluded from analyses of diurnal cortisol: two were excluded due to situations that might influence diurnal cortisol rhythms (i.e., pregnancy, working the night shift); one was excluded because of extreme cortisol values, possibly due to the flu; four never returned saliva samples; and eleven participants were added to the daily diary sample to increase the variability in early adversity scores after cortisol collection was complete, and therefore were included in analyses of daily diary measures but not cortisol. Participants who were not included in analyses of diurnal cortisol did not differ from the rest of the sample in terms of early adversity scores, $t(129) = .99, p = .33$, age, $t(128) = .45, p = .65$, BMI, $t(128) = .57, p = .57$, gender, $\chi^2(1, n = 131) = .28, p = .60$.

Procedure

Students who met criteria for the study based on the pre-screening questionnaire attended a baseline visit, during which they were asked to provide informed consent and completed questionnaires about early adversity, social functioning, and physical and mental health. They were then instructed on how to complete daily diary assessments. Participants completed the first daily diary assessment on the night of the baseline visit, and the remaining daily diary assessments on the following 13 days. Each evening an automated email message reminded participants to complete daily diaries at bedtime (between 8 PM and 3 AM), and provided a link to the daily diary website. To encourage compliance, participants who completed all daily diary surveys on time were entered into a drawing for gift certificates.

Finally, participants were provided with Salivettes and instructed about how to collect saliva samples four times daily for three consecutive weekdays, beginning as close to the baseline assessment as possible. In order to encourage compliance, participants were asked to send a text message with a picture of the completed sample to the experimenter when they had collected each cortisol sample, and they also recorded the time each sample was taken on an accompanying form. At the baseline assessment, research assistants encouraged participants to take any missed cortisol samples as soon as they remembered, and to note down the time of the actual sample, rather than the time it was supposed to be taken. Throughout cortisol sampling, research assistants were available to discuss any problems or obstacles that participants encountered during sample collection. In addition, participants who completed all cortisol assessments on time were entered into a second drawing for gift certificates as further incentive to complete samples on time.

Measures

Baseline Assessment

The main measure of early adversity was the RFQ, which was administered at pre-screening as described above. On the RFQ, participants report the extent to which they have been exposed to a variety of stressful family environments (e.g., verbal abuse, family substance abuse) between the ages of 5 and 15 on a scale from 1 “Not at all” to 5 “Very often” (see Appendix A for a full list of items). The RFQ has shown high agreement with clinical interviews designed to assess early life stress, and scores on the RFQ have been reliably linked to adverse mental and physical health outcomes (Lehman et al., 2005; Taylor et al., 2004). Cronbach’s alpha for the RFQ in the current sample was .87.

Several supplemental questionnaires were administered at baseline in order to provide construct and convergent validity for the RFQ as a measure of early adversity, and to describe the general psychological and physical health of the sample. These supplemental questionnaires are listed below.

Early adversity. Additional details of participants' experiences of specific types of abuse and neglect in childhood were assessed using the Childhood Trauma Questionnaire (CTQ; Bernstein & Fink, 1998). The CTQ is a 28-item self-report questionnaire that assesses individuals' experiences with physical abuse, sexual abuse, emotional abuse, physical neglect, and emotional neglect during childhood. Items assessing sexual abuse were not administered, given that experiences of sexual abuse were an exclusion criterion for the study, as noted above. The CTQ defines the remaining types of adversity in the following way: *physical abuse* includes bodily assaults on the child by an older person that pose a risk of, or result in, injury; *emotional abuse* involves verbal assaults on the child's sense of worth or well-being, as well as any humiliating, demeaning, or threatening behavior directed towards the child by an older person; *physical neglect* involves the failure of caregivers to provide a child's basic physical needs, such as food, shelter, safety, supervision, or health; and *emotional neglect* involves the failure of caregivers to provide a child's basic psychological and emotional needs, such as love, encouragement, belonging, and support (Kong & Bernstein, 2009). The CTQ shows good convergent validity with trauma histories from other measures (Bernstein et al., 1994), and has been shown to be reliable and appropriate for use in community samples (Scher et al., 2001). Cronbach's alpha for the CTQ items used in the current sample was .84.

Finally, participants were administered a checklist of additional adversities at baseline. Adversities on the checklist included parental divorce/separation, death of a parent or sibling,

witnessing a violent crime, exposure to war or an armed conflict, and family qualifying for or receiving welfare benefits. Participants were asked to endorse any of the adversities that they have experienced and indicate the age at which they were experienced.

Physical health. Overall physical health at baseline was assessed using the SF-36 Health Survey (Ware, Snow, & Kosinski, 2000), a standardized measure that includes items pertaining to physical functioning, vitality, bodily pain, and general perceptions of health. The summary and subscale scores of the SF-36 have been shown to have good reliability, and to be valid indicators of current health status in community samples (Ware & Gandek, 1998). Cronbach's alpha for the current sample was .88.

Depression. Current depressive symptoms at baseline were measured using the Beck Depression Inventory – 2nd Edition (BDI-II). The BDI-II consists of 21 items rated on a 4-point scale from 0 to 3. The BDI-II was written to reflect current diagnostic criteria for a major depressive episode, and shows a high internal consistency in college student samples (.93; Beck, Steer, & Brown, 1996). Cronbach's alpha was .90 in the current sample.

Anxiety. Current anxiety symptoms at baseline were measured using the Penn State Worry Questionnaire (PSWQ), a well-validated self-report questionnaire (Meyer, Miller, Metzger, & Borkovec, 1990). Cronbach's alpha was .95 in the current sample.

Perceived loneliness. Participants' current loneliness was measured using the UCLA Loneliness Scale—Revised. The UCLA Loneliness Scale—Revised is a well-validated and reliable self-report measure that assesses respondents' satisfaction with current social relationships, as well as their feelings of loneliness (Russell, Peplau, & Cutrona, 1980). Cronbach's alpha was .83 in the current sample.

Perceived stress. Overall levels of perceived stress in the past month were measured using the Perceived Stress Scale (PSS). The PSS is a commonly used and well-validated measure of individuals' perceptions of how uncontrollable, unpredictable, and overloaded with stress their lives are (Cohen & Williamson, 1988). Cronbach's alpha was .88 in the current sample.

Rejection sensitivity. Rejection sensitivity at baseline was measured using the Rejection Sensitivity Questionnaire (RSQ; Downey & Feldman, 1996). The RSQ is a self-report questionnaire that was developed to assess individuals' feelings of anxiety and expectations about interpersonal situations that contain the possibility of rejection. The RSQ is well-validated and has been shown to diverge from related constructs. It has also been shown to have good test-retest reliability (.83 over a 2-3-week period and .78 over a 4-month period; Downey & Feldman, 1996). Cronbach's alpha was .79 in the current sample.

Current family environment. Participants' current family relationships were assessed at baseline using Parental Bonding Inventory (PBI; Parker, Tupling, & Brown, 1979), a well-validated self-report measure. Questions were re-written to ask about current relationships (rather than childhood relationships) with the participant's mother and father separately. The PBI has shown good reliability and validity (Parker et al., 1979), and has been shown to assess family relationships independently of mood effects (Parker, 1983). The PBI has two subscales: a care subscale that assesses the extent to which the parent is perceived as warm and affectionate, and an overprotection subscale that assesses the extent to which the parent is perceived as controlling and constraining. The average Cronbach alpha for the mother and father care subscales in the current sample was .83 (.82 for mothers and .84 for fathers), and the average Cronbach alpha for the overprotection subscales was .91 (.90 for mothers and .92 for fathers).

Daily Diary Assessment

Daily social behavior. Social behavior was assessed using 21 questions about daily reassurance-seeking (5 items; e.g., “I found myself asking the people I feel close to how they *truly* feel about me.”), withdrawal (5 items; e.g., “I avoided others”), aggressive behavior (7 items; e.g., “I got so mad I yelled at or insulted someone”), and positive social behavior (4 items; e.g., “I showed affection toward someone else”). Questions about social behaviors were drawn from a variety of measures meant to capture aspects of social behavior, including the Depressive Interpersonal Relationships Inventory (DIRI; Joiner, 1994), the Inventory of Interpersonal Problems—48 item version (IIP; Gude, Moum, Kaldestad, & Friis, 2000), the Aggression Questionnaire (Buss & Warren, 2000), and the relational aggression subscales of the Revised Self Report of Aggression and Social Behavior (Morales & Crick, 1999). These items were chosen because they have been shown to be representative of overall reassurance-seeking, withdrawal, aggression, and positive social behavior, but are also likely to occur on a daily basis. In addition, a variety of aggressive behavior items were selected to be able to measure different types of aggressive behavior (i.e., relational aggression and physical aggression). Each social behavior was endorsed as either present or absent each day, and if an item was endorsed as present, participants were asked to report how many times it occurred that day. Counts of reassurance-seeking, withdrawal, aggression, and positive social behavior were used as four social behavior outcomes. Daily social behavior items are presented in Appendix B, grouped by type of social behavior.

Daily stress. A checklist of 14 social stressors was created using items drawn from instruments designed to elicit self-reports of recent social stress, including the social conflict subscale of the Diary of Ambulatory Behavioral States (DABS; Kamarck et al., 1998), the Inventory of Small Life Events (Zautra, Guarnaccia, & Dohrenwend, 1986), the Objective and

Subjective Event Checklist (Seidlitz & Diener, 1993), and the Brief Adolescent Life Event Scale (Shahar, Henrich, Reiner, & Little, 2003). Items were chosen to represent a range of negative social experiences that might occur on a daily basis, such as rejection, conflict, and criticism. Example items include “had an argument/problem with significant other” and “was rejected or excluded from a group event (party, group project, etc.)” Participants endorsed each item as either present or absent over the past day, and if an item was marked as present, indicated the number of times that the event occurred throughout the day. A count of all events endorsed for a given day was then used as a measure of daily social stress.

Daily non-social stressors were also assessed in order to evaluate whether effects on daily physical symptoms and mood were unique to social stressors. A checklist of 8 non-social stress items was drawn from similar measures to the social stress items, including the Inventory of Small Life Events (Zautra, Guarnaccia, & Dohrenwend, 1986), the Objective and Subjective Event Checklist (Seidlitz & Diener, 1993), and the Brief Adolescent Life Event Scale (Shahar, Henrich, Reiner, & Little, 2003). Non-social items were chosen to represent a wide range of stressful experiences across academic, work, and financial domains. Non-social stress items were also marked as present or absent, and a count of all events endorsed for a given day was used a measure of daily non-social stress. A full list of daily stress items is included in Appendix C.

Daily health. Participants rated how much they have been bothered by a number of commonly reported symptoms or discomforts adapted from the Pennebaker Inventory of Limbic Languidness (PILL; Pennebaker, 1982), from 0 (not at all) to 4 (extremely). Symptoms on this checklist include bodily pains (e.g., headaches), symptoms of upper respiratory infections (e.g., nasal congestion, coughing), digestive system problems (e.g., diarrhea), and several other symptoms, such as skin rashes and dizziness. Because previous factor analyses using the PILL

symptoms have consistently shown one general factor (Pennbaker, 1982), all items were summed into an overall physical complaint score for the day. A list of daily physical health items can be found in Appendix D.

Daily mood. Daily mood was assessed using 31 items from the Positive and Negative Affect Scales-Expanded Form (PANAS-X; Watson & Clark, 1994), a validated measure of positive and negative affect. Items were drawn from scales shown to measure overall positive and negative affect, as well as specific types of negative affect, including fear, hostility, and sadness. Participants rated the extent to which they have felt each of the items over the past day from 1 (very slightly or not at all) to 5 (extremely). A list of daily mood items can be found in Appendix E.

Cortisol. Participants collected saliva samples at home four times per day for three days using cotton swabs, or “Salivettes” (Sarstedt, Inc.). Samples were collected immediately upon awakening (before getting out of bed), 30 minutes after awakening, 8 hours after awakening, and at bedtime. Participants were instructed not to brush their teeth, eat, drink, or smoke in the 30 minutes before each sample is collected. Research assistants discussed each participant’s typical daily schedule with him or her at the baseline assessment, and helped the participant plan the samples based on the participant’s projected waking time and bedtime. Participants stored saliva samples in a refrigerator until the end of all three collection days, and then returned the samples to the laboratory. If a participant did not have access to a refrigerator, he or she returned the samples to the research assistant at the end of each day to be stored. All samples were then frozen locally at -20°C. Samples were shipped in three batches to the laboratory of Dr. Clemens Kirschbaum in Hamburg, Germany. After thawing, Salivettes were centrifuged at 3,000 rpm for 5 min, which resulted in a clear supernatant of low viscosity. Salivary cortisol concentrations

were measured with a commercially available chemiluminescence immunoassay with high sensitivity (IBL, Hamburg, Germany). The intra and interassay coefficients for cortisol were below 8%. All cortisol concentrations are presented in nmol/l.

Data Analysis

Hypotheses were examined using hierarchical linear modeling (HLM). HLM is appropriate for research designs in which individuals are observed at multiple time points, as in daily diary studies. In these situations, the nesting of time points within individuals creates dependencies among observations within subjects, violating assumptions of the independence of errors needed in the OLS regression framework, and leading to inflated rates of Type I error (Snijders & Bosker, 1999). HLM accounts for the nesting of time points within individual by estimating both within-person (Level 1) and between-person (Level 2) error variances.

Hypothesis 1 was tested using four separate HLM functions, each predicting a different type of daily social behavior from early adversity score, controlling for gender. The following is an example of the HLM functions tested.

$$\text{Level 1: } AGG_{ti} = \beta_{0i} + r_{ti}$$

$$\text{Level 2: } \beta_{0i} = \gamma_{00} + \gamma_{01}(\text{GENDER}_i) + \gamma_{02}(\text{RFAVG}_i) + u_{0i}$$

AGG_{ij} represents the number of aggressive behaviors reported on day t . Scores on the RFQ were entered grand-mean centered into the Level 2 equation, and gender was entered as an uncentered covariate on Level 2.

Hypothesis 2 was examined using HLM functions such as the following,

$$\text{Level 1: } PHY_{ti} = \beta_{0i} + \beta_{1i}(\text{SOCIALST}_i) + r_{ti}$$

$$\text{Level 2: } \beta_{0i} = \gamma_{00} + \gamma_{01}(\text{GENDER}_i) + \gamma_{02}(\text{SOCIALAVG}_i) + u_{0i}$$

$$\beta_{1i} = \gamma_{10} + u_{1i}$$

where PHY_{ti} represents the total severity of physical symptoms on day t for individual i and $SOCIALST_i$ represents the count of social stressors on day t for individual i . The effect of gender on physical symptoms is controlled for by including it as a between-subjects predictor of the intercept on Level 2. Level 1 predictors, such as $SOCIALST_i$ were person-mean centered, such that $SOCIALST_i$ represents the difference between the number of social stressors occurring on day t for individual i and that individual's average level of daily social stressors (Mroczek & Almeida, 2004; Sholz, Kliegel, Luszczynska, & Knoll, 2012). The individual's overall average for daily social stress was included as a Level 2 predictor of the intercept, $SOCIALAVG$. This method allows for the disaggregation of the within-person (β_{1i}) and between-person (γ_{11}) effects of daily social stress on physical symptoms (Raudenbush & Bryk, 2002). The potential influence of early adversity on the relationship between social stress and physical symptoms was assessed using the same functions as specified above, except that early adversity score was added as a Level 2 predictor of both the Level 1 intercept (β_{0i}) and slope (β_{1i}). The cross-level interaction between early adversity and $SOCIALST_i$ was then examined in order to explore the moderating role of early adversity in the association between social stress and daily physical symptoms.

Hypothesis 3 was tested using HLM functions such as the following,

$$\text{Level 1: } NEG_{ti} = \beta_{0i} + \beta_{1i} (SOCIALST_i) + r_{ti}$$

$$\text{Level 2: } \beta_{0i} = \gamma_{00} + \gamma_{01}(GENDER_i) + \gamma_{02}(SOCIALAVG_i) + u_{0i}$$

$$\beta_{1i} = \gamma_{10} + u_{1i}$$

where NEG_{ti} represents the severity of overall negative affect on day t for individual i and $SOCIALST_i$ represents the count of social stressors on the same day. Again, daily stress predictors, such as $SOCIALST_i$, were person-mean centered, and between-person effects of social stress and gender were accounted for by entering these variables as predictors of the

intercept on Level 2. The influence of early adversity on the relationship between social stress and negative affect was assessed using the same functions, except that early adversity was added as a Level 2 predictor. The cross-level interaction between early adversity and $SOCIALST_i$ was then examined in order to explore the stress-sensitizing effects of early adversity.

Hypothesis 4 was examined by using a growth curve modeling framework to test the effects of early adversity on diurnal cortisol slope across the day. Consistent with past research (e.g., Adam, 2006; Cohen et al., 2006), cortisol levels at the second time point (30 minutes after waking) were not included in analyses of diurnal cortisol slope. This method allows for the measurement of the diurnal slope independent of the cortisol awakening response, which has been shown to be influenced by different neurobiological processes than the rest of the curve (Adam & Kumari, 2009). The equations below reflect a three level structure in which the remaining three measurement occasions are nested within days, which are in turn nested within individuals.

$$\text{Level 1: } CORT_{jti} = \beta_{0ti} + \beta_{1ti}(TIME_{ti}) + \beta_{2ti}(TIME_{ti})^2 + r_{jti}$$

$$\text{Level 2: } \beta_{0ti} = \gamma_{00i} + u_{0ti}$$

$$\beta_{1ti} = \gamma_{10i} + u_{1ti}$$

$$\beta_{2ti} = \gamma_{20i} + u_{2ti}$$

$$\text{Level 3: } \gamma_{00i} = \pi_{000} + \pi_{001}(EA) + \pi_{002}(GENDER) + \pi_{003}(AGE) + e_{00i}$$

$$\gamma_{10i} = \pi_{100} + \pi_{101}(EA) + e_{10i}$$

$$\gamma_{20i} = \pi_{200} + \pi_{201}(EA) + e_{20i}$$

where $CORT_{jti}$ represents amount of cortisol at measurement occasion j on day t for individual i and $TIME_{ti}$ represents time since awakening on day t for individual i . The effects of gender and

age were controlled for at Level 3 in the intercept equation. Cortisol values were positively skewed, and therefore were log transformed for these analyses.

Models were first run without the inclusion of early adversity group on Level 3 in order to determine the appropriate function for modeling cortisol output (i.e., linear versus quadratic trend). Then, early adversity was included as a between-subjects predictor, and the cross-level interactions between early adversity and the relevant polynomial terms were examined in order to test whether early adversity influences the diurnal cortisol slope.

Results

Descriptive Statistics

Descriptive statistics for the supplemental baseline measures, as well as their correlations with RFQ average scores, are presented in Table 6. Early adversity scores on the RFQ showed a significant, positive relationship with scores on the CTQ, as well as the Early Childhood Adversity Checklist. Higher early adversity scores on the RFQ were also associated with higher levels of depressive symptoms and worry, poorer health, greater loneliness, higher rejection sensitivity, higher current perceived stress, lower levels of perceived maternal and paternal care, and higher levels of perceived paternal overprotectiveness.

Descriptive statistics and example items for daily diary questionnaires are presented in Table 7. On average, participants completed 12.48 (SD = 2.22) out of 14 daily diaries and completed 92% (SD = 13%) of submitted surveys on time (between 8PM and 3AM), a rate comparable to or better than that of other daily diary studies conducted in college student samples (e.g., Covault et al., 2007; Sahl, Cohen, & Dasch, 2009). The pattern and significance of results were identical when analyses controlled for whether daily diaries were completed on time. Analyses were also run without participants who responded to less than 50% of all daily

diaries ($n = 6$), and the pattern and significance of results remained the same, except where noted.

Average cortisol levels (nmol/l) at waking ($M = 20.97$, $SD = 11.15$), 8 hours after waking ($M = 9.77$, $SD = 5.54$), and bedtime ($M = 4.33$, $SD = 4.31$) were similar to averages at these time points reported for other adult samples (e.g., Cohen et al., 2006). In accordance with standard practices in the literature, cortisol values more than three standard deviations above the mean for a given time point were excluded from analyses (Adam & Kumari, 2009). In addition, measurements were excluded for days with extreme waking day lengths (greater than 20 hours; see Karlamangla et al., 2013). Waking cortisol values were excluded if the sample was taken more than 10 minutes after wake-up, to ensure a valid waking cortisol level when calculating the diurnal slope (Adam & Kumari, 2009; Kunz-Ebrecht et al., 2004).

Early Adversity and Daily Social Behavior

Results for analyses examining the effects of early adversity on social behavior are presented in Table 8. Early adversity predicted more instances of daily reassurance seeking, controlling for the effects of gender. In addition, early adversity was marginally statistically significant in predicting more instances of daily withdrawal and aggression. Early adversity was not a statistically significant predictor of the number of daily positive behaviors. When low responders (completed less than 50% of surveys) were eliminated from analyses, early adversity became a significant predictor of aggressive behaviors ($b = .56$, $SE = .25$, $p < .05$) and withdrawal behaviors ($b = .89$, $SE = .39$, $p < .05$).

Daily Stress and Physical Symptoms

Daily social stress and daily non-social stress predicted higher same-day physical symptoms (see Table 9). When social and non-social stress were examined simultaneously as

Level 1 predictors in the same model, both social stress ($b = .17, SE = .05, p < .001$) and non-social stress ($b = .17, SE = .05, p < .001$) remained significant predictors of higher same-day physical symptoms. Moreover, both daily social stress ($b = .08, SE = .04, p = .054$) and non-social stress ($b = .06, SE = .02, p < .05$) predicted same-day physical symptoms even when controlling for the prior day's physical symptoms. However, the effect of daily social stress on same-day physical symptoms, controlling for continuity in physical symptoms, became marginally significant when low responders were eliminated from the analyses ($b = .08, SE = .04, p = .06$).

When the potential role of negative affect in these findings was examined, both daily social stress and daily non-social stress continued to have a unique effect on same-day physical symptoms, over and above the significant effects of negative affect on physical symptoms (see Table 10).

Early adversity did not have a main effect on daily physical symptoms, and did not moderate the effects of daily social stress or non-social stress on same-day physical symptoms (see Table 11).

Daily Stress and Negative Affect

Daily social stress and daily non-social stress both predicted higher same-day negative affect, controlling for the effects of gender (see Table 9). When social and non-social stress were examined simultaneously as Level 1 predictors in the same model, both types of stress had unique effects on higher same-day negative affect (social: $b = .44, SE = .08, p < .001$; non-social: $b = .46, SE = .10, p < .001$). In addition, daily social stress ($b = .43, SE = .09, p < .001$) and non-social stress ($b = .34, SE = .08, p < .001$) continued to predict same-day negative affect even when controlling for the prior day's negative affect.

Early adversity had a significant main effect on daily negative affect, such that greater early adversity led to higher reports of daily negative affect, over and above the effects of gender (see Table 11). However, early adversity did not moderate the effects of daily social stress or non-social stress on same-day negative affect (see Table 11).

Early Adversity and Diurnal Cortisol

Initial analyses showed that the quadratic term, which modeled a quadratic relationship between time and cortisol levels across the day, was not significant ($b = .01$, $SE = .01$, $p = .25$). In addition, initial analyses suggested little to no variability of diurnal cortisol slope across days (Level 2). As a result, a three-level model examined the effects of early adversity on the linear diurnal slope of cortisol, and the random coefficient for the linear slope at Level 2 was omitted (see Table 12 for final model results). Results suggested that there was a significant negative linear effect of time on cortisol, such that cortisol levels were high upon awakening and decreased across the course of the day. When early adversity was included on Level 3, it did not significantly predict diurnal cortisol slope.

Discussion

The present study used a daily diary methodology to investigate the impact of early life stress on day-to-day social functioning, stress reactivity, and diurnal cortisol slope in healthy young adults. Consistent with our hypotheses, early adversity predicted greater reassurance-seeking on a daily basis, and early adversity was marginally significant in predicting elevated rates of daily aggression and withdrawal. In addition, both daily social stressors and non-social stressors were associated with increased same-day physical symptoms and negative affect. However, in contrast to our hypotheses, the effects of daily stress on physical symptoms remained even after accounting for negative affect. Early adversity had main effects on daily

negative affect, but not daily physical symptoms, and did not moderate the effects of social and non-social stress on physical symptoms or negative affect. Finally, results showed that early adversity did not influence diurnal cortisol slope.

Previous studies on the effects of early adversity on social behaviors, such as aggression or withdrawal, have tended to examine these relationships in abused or neglected children. The current results extend this literature by suggesting that a broader measure of risky early family environments predicts elevated reassurance-seeking, and possibly also aggression and withdrawal, in healthy young adults. Moreover, the use of a daily diary format was able to capture the occurrence of these behaviors within a naturalistic setting, across a number of relationship contexts. Results suggesting that early adversity might lead to elevated rates of daily aggression and withdrawal within relationships in young adulthood is consistent with and extends a large body of evidence showing that abuse or neglect is linked to aggression and withdrawal in children (Kaufman & Cicchetti, 1989; Teisl & Cicchetti, 2007). Although previous literature has not directly examined the effects of early adversity on daily reassurance-seeking, this finding is consistent with evidence that early adverse experiences lead to insecure attachment styles (Styron & Janoff-Bulman, 1997), which have in turn been linked to excessive reassurance-seeking (Shaver, Schachner, & Mikulincer, 2005).

The current study also examined young adults' experiences of physical symptoms and negative affect in response to stress. Much of the previous work on stress and physical and mental health has failed to examine the roles of social and non-social stressors separately, but simultaneously. This is an important line of inquiry given theories suggesting that different types of stressors might evoke different behavioral or physiological coping responses, depending on the goals or resources that are being threatened (Weiner, 1992; Dickerson, Gruenewald, &

Kemeny, 2004). In particular, several studies have shown that social stressors have an especially potent impact on physiological response to a laboratory stressor (Dickerson et al., 2009; Dickerson & Kemeny, 2004) and negative affect (Hammen, 2005). However, few studies have examined the effects of different types of stressors in a naturalistic setting. In contrast to theories of stress specificity, current findings suggest that both social and non-social stressors uniquely predict increases in daily physical symptoms and negative affect. It is possible that the use of a college student sample could have contributed to these findings, given that academic non-social stressors are particularly salient for this population. Analyses were also able to examine whether the effects of daily stress on physical health were accounted for by negative mood, given the close links between markers of poor physiological functioning and depression (e.g., Kiecolt-Glaser & Glaser, 2002). Results showed that both social and non-social stressors predicted daily physical symptoms even after controlling for daily negative affect.

In addition to examining the separate effects of daily social and non-social stress on physical and emotional functioning, the current study examined the role of early adversity in reactivity to these stressors. Early adverse experiences predicted increased negative affect on a daily basis, consistent with between-subject findings that individuals who have experienced early adversity have higher rates of emotional difficulties, such as depression and anxiety (Kessler & Magee, 1993; Phillips et al., 2005). However, early adversity did not have a main effect on daily physical complaints, and did not amplify the effects of daily stress on physical symptoms and negative affect. Previous research has not specifically examined the moderating role of early adversity in the effects of stress on daily physical and emotional complaints. However, there is evidence that early adversity can lead to higher negative affect in response to stressors in children (Ballard et al., 1993; Davies & Cummings, 1998), as well as a stronger relationship

between stressful life events and depression in adulthood (Kendler, Kuhn, & Prescott, 2004; Starr, Hammen, Conway, Raposa, & Brenna, in press). Moreover, increased neurobiological sensitivity to stress has been found to underlie the association between early life stress and later depression and anxiety (Heim & Nemeroff, 2001), and these physiological alterations could also have implications for physical health.

It is possible that current findings are not consistent with these theories of early adversity and increased stress sensitivity because of the college student sample used. Individuals who have experienced early life adversity, but have been able to function adequately in the social and academic contexts of college, might have learned to successfully cope in the face of stress despite heightened vulnerability. Thus, these individuals might be able to adapt to stressors without experiencing elevated physical or emotional symptoms as a function of their early family experiences. In addition, the daily diary design of our study might not have captured stressors severe enough to persistently alter physical and psychological responses to stress. Previous between-subjects studies on early adversity and stress sensitivity have tended to use measures of major life stressors (e.g., sexual abuse, death of a parent). In contrast, the time course of the present study was relatively short (two weeks), and most stressors captured were likely minor, daily hassles (e.g., a fight with a roommate). Thus, it is possible that the effects of early adversity on stress reactivity only become evident in the presence of more severe stressors.

Finally, the present study did not find effects of early life stress on diurnal cortisol slope. This finding is in contrast to a substantial body of literature that has linked early adversities, such as abuse and neglect, to abnormal cortisol rhythm in children (Carlson & Earls, 1997; Gunnar, 2000; Gunnar et al., 2001). However, very few studies have examined whether the negative impact of early adversity on HPA axis functioning persists into adulthood (for exceptions, see

Taylor et al., 2011; van der Vegt et al., 2009), and the few existing studies have tended to examine this question in samples of adults suffering from chronic physical or mental illness. It is possible that there are important psychosocial factors, such as depression, that influence whether early adversity leads to lasting alterations in HPA axis functioning after childhood. In addition, as noted above, it is possible that our measures and sample did not capture early adversity severe enough to result in lasting damage to the neurobiological systems involved in regulating diurnal cortisol output.

Limitations and Future Directions

Several additional limitations of the current study should be acknowledged. First, the daily measure of physical symptoms was drawn from the PILL, which has been validated in between-subjects analysis, rather than studies of within-subject fluctuations across days. In addition, items for the daily measures of stress and mood were drawn from previous daily diary research, but adjusted as necessary for a college student population. Thus, further research is needed to validate these measures, and to ascertain whether there might be additional stressful events or physical symptoms that are particularly relevant for mental and physical health among young adults. Second, as noted above, the undergraduate sample used is likely not representative of the general population in terms of early childhood experiences, and findings will therefore need to be replicated in community samples. Third, given that there was only one assessment per day, it is impossible to know that stressors always preceded negative affect and physical symptoms on a given day. Secondary analyses were run to attempt to address the question of timing, and showed that results remained the same when controlling for the previous day's physical symptoms or negative affect in predicting today's physical symptoms or negative affect. Thus, there is some evidence that the findings were not simply accounted for by physical and

emotional symptoms on a previous day. However, multiple assessments per day are needed to fully untangle the temporal associations between stress and negative physical and emotional outcomes. Fourth, brevity in daily diary measures is crucial for encouraging compliance. As a result, daily stressors in the proposed study were not assessed using gold standard semi-structured interviews, and were based on subjective, rather than objective, ratings of stress. Ratings of daily stress could therefore have been influenced by individual's affective states, and the threshold for reporting subjective experiences of stress might have varied across individuals. However, this limitation was somewhat addressed by using within-subjects analyses, for which fluctuations in stress are compared to each individual's mean across all 14 days. Finally, consistent with the overall population of students enrolled in psychology classes, our sample was approximately one third male and two thirds female. As a result, we did not have enough male participants to fully explore the potential moderating role of gender in analyses.

Despite these limitations, the present project addresses several gaps in our understanding of the long-term impact of early adversity on social functioning and health. The use of a daily diary format tested whether models of the effects of early adverse experiences and ongoing stress on emotional and physical health can be applied to a variety of naturally-occurring social interactions outside of the laboratory. Moreover, examining these questions in a young adult population showed that the negative effects of early adversity on social functioning and mood persist beyond childhood, and might therefore play a role in the long-term impact of early adversity on health. The use of a non-clinical sample also established that some negative emotional and physical effects of early adversity exist in the absence of confounding factors such as chronic mental or physical illness.

Future research should explore the extent to which problematic social behaviors, such as reassurance-seeking and aggression, might mediate the negative effects of early adversity on chronic stress within close relationships in adulthood. In addition, future research should further investigate the physiological mechanisms that underlie the long-term impact of early adversity and poor social functioning on health. In particular, there might be genetic or psychosocial factors that moderate the impact of early life adversity on the functioning of the HPA axis and immune system later in life, thereby creating risk for mental and physical health problems. Identification of these multiple mechanisms of the effects of early adversity on later health has the potential to inform the development of targeted psychosocial and biological interventions that could prevent the long-term consequences of childhood adversity and benefit populations afflicted by chronic physical or mental illness.

Chapter 5: General Discussion

The overarching goal of the present dissertation was to explore several mechanisms by which early adversity prospectively shapes physical health in adulthood. Exposure to early life stress is a relatively common phenomenon, with approximately one third of adults in community samples endorsing experiencing some type of physical, emotional, or sexual abuse or neglect (Scher et al., 2004). These early stressful experiences have been found to create risk for a variety of negative health outcomes later in life, including serious disease and death (Felitti et al., 1998; Power et al., 2005; Springer et al., 2007). Despite this clear link between stressful early experiences and health problems, the specific psychosocial and biological mechanisms by which early adversity affects later health remain unclear. The current set of studies was therefore designed to uncover how various early psychosocial experiences continue to shape individuals' emotional and social functioning beyond childhood, thereby creating risk for poor physical health in adulthood. Moreover, the dissertation sought to examine whether and how negative psychosocial experiences might “get under the skin” to affect biological outcomes decades later.

Summary of Results

Based on the findings from the three distinct papers presented above, it is possible to address the objectives stated in the introduction.

1. Does early adversity influence physical health in young adulthood?

Study 1 used longitudinal data from a community sample to show that cumulative experiences of early adversity, as measured by contemporaneous maternal report, predicted poor self-reported and interviewer-rated physical health in young adults. Results suggested that early adverse experiences led to ongoing stress in social and nonsocial (e.g., academic) contexts, as well as increased depressive symptoms, which in turn portended poor health. Study 3 examined

the day-to-day dynamics of these relationships on a within-person basis in a sample of college students. Results within this college sample suggested that early adversity did not predict higher reports of daily physical symptoms or moderate the relationship between daily stress and physical symptoms. Thus, the effects of exposure to early adversity on health may only be observed when measuring more global measures of overall perceived physical functioning status between subjects, rather than within-subject daily fluctuations in minor physical complaints. In addition, it could be that different measures of early adversity (and contemporaneous versus retrospective reports of adversity) might show different effects on later physical health.

2. Does the negative impact of childhood adversity on social functioning extend into adolescence and young adulthood? If so, what types of social dysfunction are present?

Study 1 showed that a cumulative measure of early adversity by age 5 predicted increased chronic and acute social stress (as well as nonsocial stress) during adolescence. Building upon these findings, Study 2, using a subset of the same sample as Study 1, examined best friends' psychopathology as a particular pathway by which early adversity might give rise to increased social stress later in life. Results showed that individuals who had experienced early adversity by age 5 had best friends with higher rates of psychopathology in young adulthood. Finally, Study 3 explored the effects of early adversity on specific social behaviors within a naturalistic setting, and showed that young adults who have experienced stressful family environments reported more instances of reassurance-seeking on a day-to-day basis. In addition, early adversity was marginally associated with increased rates of daily aggression and withdrawal. Taken together, these findings suggest that early life stress continues to influence social functioning beyond childhood and into adolescence and young adulthood, which could portend increased risk for emotional and physical problems.

3. How does problematic social functioning during adolescence and young adulthood play a role in the relationship between early adversity and physical health problems?

Study 1 used a contextual measure of objective chronic and episodic stress across a number of social domains to show that stressful social relationships at age 15 serve as a mechanism of the relationship between exposure to stress prior to age 5 and physical health in young adulthood. This was true even when co-varying for the effects of chronic stress in non-social domains (although non-social stress also negatively affected physical health). Study 3 used a daily diary methodology to examine whether individuals who have experienced early adversity might report increased physical symptoms in response to social stressors, which could imply increased risk for chronic physical complaints. Results suggested that early adversity did not moderate the negative effects of daily social stressors on physical complaints.

Thus, in between-subject analyses, stressful social relationships mediated the effects of early adversity on later physical health. However, in within-subjects analyses, daily stressful social situations did not interact with early adverse experiences to predict more minor physical complaints. Further research should test whether mediation or moderation models best fit the relationships among early adversity, interpersonal stress, and health. Moreover, these two studies examined different indicators of physical health (self-reported physical functioning on the SF-36 and interviewer-rated general health versus minor daily physical symptoms). It is important for future studies to determine the specific health outcomes influenced by early adversity and ongoing social dysfunction. In particular, research should examine physiological markers of health risk, such as inflammation, as well as fluctuations in symptoms of chronic disease (e.g., asthma).

4. How does early adversity affect mood, and what role does negative mood play in the effects of early life stress and social difficulties on later physical health?

Study 1 revealed that both early adversity by age 5 and ongoing social and non-social stress in adolescence gave rise to elevated depressive symptoms in young adulthood. The negative effects of ongoing social stress on physical health appeared to be largely accounted for by depressive symptoms, while non-social stress continued to have a direct effect on physical health over and above the effects of depressive symptoms. Study 2 examined a specific social pathway by which early adversity might influence negative affect. Results showed that individuals who experienced early adversity tended to have higher depressive symptoms partially as a result of close friendships with individuals with elevated symptomatology. Both of these findings are consistent with theories suggesting that early adversity might predict selection into problematic friendships that have the potential to generate ongoing stress and depression.

Finally, Study 3 examined these questions on a within-subjects level, and found that early adversity, as well as both daily social and non-social stress, predicted elevated negative affect on a daily basis. In contrast to Study 1, both social and non-social stress continued to predict increased physical complaints when co-varying for the significant effects of negative affect on physical health. Thus, both early adversity and ongoing stress gave rise to increased negative affect on a daily basis, and negative affect had a significant impact on daily physical health. However, the within-person effects of social stress on daily physical functioning could not be completely accounted for by negative affect.

5. Does early adversity lead to abnormal diurnal cortisol rhythm in young adulthood?

This question was examined in Study 3. Results suggested that scores on a measure of conflict, chaos, and neglect within an individual's family of origin were not associated with

abnormal diurnal cortisol rhythm in young adulthood. Few studies have examined the negative impact of early adversity on HPA axis functioning in healthy adults (for an exception, see Taylor et al., 2011). Thus further research is needed to determine how and for whom early adversity has a long-term impact on HPA axis functioning. It is possible that persistent alterations in diurnal cortisol slope might be seen only for individuals who have experienced severe neglect or abuse (e.g., Carlson & Earls, 1997; van der Vegt et al., 2009) or who have ongoing mental and physical health problems (e.g., Weissbecker et al., 2005).

Implications

There has been a recent interest in uncovering the mechanisms by which early life stress might create long-term risk for poor health and mortality. Much of the current research has examined whether early adversity leads to poorer social functioning and physiological alterations in children, with the assumption that these risk factors persist and contribute to long-term risk for disease. However, few studies have actually examined whether the negative psychosocial and physiological sequelae of early adversity are also observed in adulthood. The current project highlighted two key pathways that help to explain the effects of early adversity on health in adulthood: poor social functioning and negative mood.

A large body of evidence has suggested that exposure to early life stress leads to increased social difficulties during childhood, including increased problematic social behaviors (e.g., aggression; Teisl & Cicchetti, 2007) and higher rates of negative qualities within close relationships (Howe & Parke, 2001; McCloskey & Stuewig, 2001). The current set of studies expands upon these findings by showing that early adversity continues to predict poor social functioning in young adulthood. Using a longitudinal dataset, Study 1 indicated that early adversity gives rise to elevated stress within close family and peer relationships throughout

adolescence. Studies 2 and 3 highlighted two particular mechanisms by which early adversity might create increased stress within relationships later in life. These studies showed that young adults who have experienced early adversity tend to have best friends with elevated rates of psychopathology and also tend to exhibit higher rates of problematic social behaviors, such as reassurance-seeking, aggression, and withdrawal. Importantly, Studies 1 and 3 indicated that elevated rates of social stress are in turn linked to poorer health and increased physical complaints. However, it should be noted that both of these studies also showed a significant effect of non-social stress on physical health, in contrast to theories of stress specificity.

Thus, poor social functioning is one mechanism by which early adversity might continue to have a psychosocial impact on individuals throughout adolescence and adulthood, thereby creating risk for poor health later in life. Psychosocial interventions that address problematic social behaviors and difficulties in close relationships might therefore be particularly beneficial in reducing the negative health impact of early adversity. For example, several clinical interventions have been designed to target issues of insecure attachment in maltreated children, with an eye toward helping these children form healthier and more supportive relationships with family members and peers (Cicchetti & Toth, 1995). Current findings also suggest that it might be most effective for interventions to target different types of relationships during different developmental stages. For example, Study 2 showed that a high level of psychopathology in one's best friend predicted elevated depressive symptoms during young adulthood. Functioning in close peer relationships might be particularly important for mental and physical health during adolescence and young adulthood, when individuals are becoming more independent and begin to turn to friends more frequently for advice and support (van Lieshout et al., 1999). In contrast,

the attachment relationship with the mother might be more important during earlier stages of development (Cicchetti & Toth, 1995).

The current set of findings also highlighted the role of negative mood in the effects of early adversity on later health. A significant body of literature has linked exposure to early adversity to an impaired ability to regulate negative affect in the face of stress (Ballard et al., 1993; Davies & Cummings, 1998), as well as increased rates of depression (Batten et al., 2004; Hazel et al., 2008). At the same time, depression has been linked with indicators of poor physical health, such as inflammation, as well as increased morbidity from chronic illness (Kiecolt-Glaser & Glaser, 2002). Nevertheless, it remains unclear whether depression and other forms of negative affect might serve as mechanisms of the effects of early adversity on health. That is, past research has often failed to take into account the negative effects of early adversity on mood when examining the physical health impact of early adversity. The current project addressed this gap in the literature by examining the interplay among early adversity (and ongoing stress), negative mood, and health. Studies 1 and 2 showed that early adversity leads to increased rates of depression, at least in part through increased social stress and affiliation with disordered best friends. Moreover, Study 1 indicated that these elevated rates of depression in turn create risk for poor physical health. Similarly, Study 3 showed that individuals with higher rates of early adversity (as well as ongoing daily stress) have higher rates of negative affect on a daily basis, and higher negative affect also predicts elevated rates of daily physical complaints.

These findings support the notion that early adversity can have long-lasting mental health consequences; moreover, early adversity and ongoing stress might contribute to poor physical health in part through their effects on mental health. Clinical interventions designed to reduce negative mood, such as empirically supported treatments for depression, might therefore be

helpful in reducing the negative emotional and physical impact of early adversity. Future research should explore the specific behavioral (e.g., negative health behaviors) and physiological (e.g., inflammation) mechanisms that might underlie the comorbidity of negative mood and poor health in individuals who have experienced early adversity. A better understanding of these mechanisms would allow for more targeted interventions designed to prevent the co-occurring emotional and physical consequences of early adversity.

The current dissertation did not find any negative effects of stressful early family environments on diurnal cortisol slope in a sample of college students. This finding suggests that further research is required to more fully understand whether early adversity has a long-lasting impact on HPA axis functioning. As noted above, using a sample of college students from a top-tier university might have prevented us from recruiting individuals who show enduring abnormalities in the biological response to stress. It is also possible that for some individuals, the negative impact of early adversity on HPA axis functioning is not persistent, and diurnal cortisol slope returns to normal over the course of adolescence and adulthood. If so, it is necessary to explore the psychosocial and biological factors that might moderate the persistence of HPA axis dysregulation in the face of early adversity. For example, individuals who have been exposed to early adversity, and also have certain genetic vulnerabilities or a lack of supportive peer relationships in adolescence, might be more likely to show ongoing HPA axis dysregulation in adulthood than those who do not have these additional risk factors. Individuals who go on to develop emotional and physical disorders, such as chronic pain or depression, as a result of early adversity might also be more likely to show long-lasting HPA axis dysregulation. Finally, young adults who have experienced early adversity might show persistent dysregulation in other biological systems despite normal diurnal cortisol slope. The HPA axis has important effects on

the immune system and the hippocampus, as well as other neural regions. Thus, it is possible that the downstream effects of dysregulation in the HPA axis are able to be detected, even if diurnal cortisol levels are normal, and these physiological indicators might be better avenues for investigating the long-term health impact of early adversity.

Limitations and Future Directions

One key unresolved issue in the literature on the effects of early adversity on physical health involves the measurement of early life stress. Previous research in this area has used widely varying approaches to the measurement of early adversity, making it difficult to interpret differences in findings across studies. The current project used two methods for assessing early adversity. First, in a community sample, early adversity was measured using a composite of early life adversities associated with maternal psychopathology (e.g., marital discord, low income, parental criminality) occurring up to child age 5. Second, in a daily diary study in a college student sample, early adversity was measured using the Risky Families Questionnaire, a retrospective measure of harsh and chaotic family environments between the ages of 5 and 15. These measures were chosen to be consistent with evidence showing that early adversities frequently co-occur and should be assessed in clusters, rather than as individual adversities (Dong et al., 2004; Green et al., 2010; Kessler et al., 1997). Moreover, a cluster of adversities associated with family psychopathology (e.g., family violence, parental mental illness) has been shown to be a more potent predictor of later mental health problems than other clusters of adversity, such as abuse and neglect or interpersonal loss (Green et al., 2010). Nevertheless, more research is needed to determine the specific clusters of adversity that are the most important in predicting poor physical health. Moreover, research should examine whether there is any specificity in the psychological and physiological domains affected by different clusters of

early adversity. For example, neglect might lead to a different shift in biological functioning than physical abuse.

Relatedly, it is crucial to determine whether the negative health effects of early adversity depend on the timing of exposure. Past studies have examined the effects of exposure to early stress across a variety of developmental stages, from infancy to adolescence. Moreover, some studies have examined the effects of adversity during specific time periods (e.g., ages 7 to 13, Cicchetti et al., 2010), while others have used very broad age ranges to capture cumulative early adversity (e.g., up to age 18, Green et al., 2010). In the current project, two studies utilized contemporaneous assessment of early adversity during the first five years of life, while the third study examined retrospective reports of stressful family environments between ages 5 and 15. This broad conceptualization of “early” in the measurement of early life stress makes it difficult to compare findings regarding the negative physical effects of early adversity. It is likely that the duration and timing of early adversity plays an important role in whether it has persistent negative consequences for health. Consistent with this hypothesis, there is evidence showing that adversities can have different consequences for social (Manly, Kim, Rogosch, & Cicchetti, 2001) and neurobiological (Rao et al., 2010; Tottenham et al., 2009) development depending on the timing of exposure to the adversity. Thus, future research will need to more carefully examine how exposure to early adversity at different developmental stages might translate into the social and physiological deficits that create risk for poor health. Moreover, research will need to examine the severity of adversity required to interfere with normal developmental processes during each of these stages.

Finally, the current dissertation used mostly self-report and interview measures to assess physical health. In Study 1, physical health was measured using the physical functioning

subscale of the SF-36, as well as health-related impairment ratings from the UCLA Life Stress Interview. In Study 3, participants provided daily reports of their experiences with a range of minor physical symptoms (e.g., headaches). Future research might benefit from directly examining physiological markers of disease risk. As one example, Study 3 in the current dissertation examined diurnal cortisol slope as an indicator of HPA axis functioning and risk for poor health. Future studies should examine additional markers of risk for disease, such as inflammation or sympathetic nervous system (SNS) activity, in order to more fully understand the ways in which early adversity influences the complex interactions among biological systems in a way that creates risk for poor health.

More careful examination of the types of early adversity that create risk for poor health, as well as the moderating and mediating variables that play a role in these effects, would help to improve explanatory models of the course of physical and mental illness. Moreover, such research could aid in the development of targeted interventions designed to prevent the long-term emotional and physical consequences of early life stress.

Appendices

Appendix A. Psychology subject pool pre-screening items

Early life stress items to screen for a range of early adversity scores:

These are questions about your childhood and early adolescence (age 5 – 15). Please think over your family life during that time and answer these questions.

RF1. How often did a parent or other adult in the household make you feel that you were loved, supported, and cared for?

1	2	3	4	5
Not at all				Very often

RF2. How often did a parent or other adult in the household swear at you, insult you, put you down, or act in a way that made you feel threatened?

1	2	3	4	5
Not at all				Very often

RF3. How often did a parent or other adult in the household express physical affection for you, such as hugging, or other physical gestures of warmth and affection?

1	2	3	4	5
Not at all				Very often

RF4. How often did a parent or other adult in the household push, grab, shove, or slap you?

1	2	3	4	5
Not at all				Very often

RF5. In your childhood, did you live with anyone who was a problem drinker or alcoholic, or who used street drugs?

1	2	3	4	5
Not at all				Very often

Appendix B. Social behaviors

Reassurance seeking items

I sought reassurance from people I feel close to as to whether they really care about me
I found myself directly asking the people I feel close to how they *truly* feel about me
People I feel close to got 'fed up' with me for seeking reassurance from them about whether they really care about me
I made comments to others to try to elicit how they *really* feel about me
I posted something on social media or checked for responses to see whether others like me

Withdrawal items

I felt like being alone rather than spending time with others
I chose not to go to a party or event because I didn't feel like being around others
I avoided someone (e.g., a friend, professor, family member) when I saw them in person
I chose not to answer someone's phone call because I didn't feel like talking
I went to a social event but avoided interacting with others

Aggression items

I was mean to others
I got so mad that I yelled at or insulted someone
I spread rumors or gossiped about someone
I excluded someone from a study group, party, etc.
I lied to get what I wanted
I got so mad that I pushed, grabbed, or hit someone
I got so mad that I broke or threw something

Positive items

I sent or received an enjoyable letter/email/phone call from someone
I showed affection toward someone else
I was supportive of someone else
I went out socializing or spent pleasant or relaxing time with someone

Appendix C. Stressors

Social stressors

Ended a dating relationship

Someone (friend, significant other, family member, etc.) refused to provide help in response to a request for assistance

Had an argument/problem with significant other

Had an argument/problem with a friend

Had an argument/problem with a roommate

Had an argument/problem with family member

Had an argument/problem with a professor, or project group

Fight or argument among social group to which you belong

Was rejected or excluded from a group event (party, group project, etc.)

Was turned down asking someone for a date

Was criticized by someone (significant other, friend, professor, project group, etc.)

Friend or acquaintance teased or made fun of me

My friends were not available when I wanted to socialize

I was pressured by a parent or other family member about something (school, social life, etc.)

Non-social stressors

Had problems at work (e.g., didn't get the schedule you requested, couldn't find someone to fill in for you)

Had to study for a difficult exam

Did poorly on, or failed, an important exam or major project

Was late for an important meeting, exam, etc.

Failed to achieve an important school related goal that does not involve GPA (e.g., didn't get a leadership position in a school related group, didn't get into a required class)

Did not have enough money to do something or buy something

Lost money or something important

Property was damaged or stolen

Appendix D. Physical health

Physical symptoms

How much have the following problems bothered or disturbed you today from 0 (not at all) to 4 (extremely)? Do **not** count symptoms that are the result of intentional physical exercise.

Headache

Backache

Joint pain (knee, shoulder, ankles, hip)

Chest pain

Stiff or sore muscles

Nasal congestion/runny nose

Coughing

Sore throat

Sneezing spells

Heartburn

Stomachache/nausea

Abdominal pain

Constipation

Diarrhea

Skin rashes

Dizziness/faintness

Are you suffering from any injury, illness, or other physical condition that might be affecting your experiences with these problems today (e.g. menstrual period, flu, sports injury, hangover)?

Yes No

If so, what is the physical condition?

Appendix E. Daily mood

Daily mood

Indicate the extent to which you have felt this way during the past day from 1 (very slightly or not at all) to 5 (extremely).

Interested
Distressed
Excited
Upset
Strong
Guilty
Scared
Hostile
Blue
Enthusiastic
Proud
Irritable
Alert
Ashamed
Tired
Inspired
Nervous
Determined
Attentive
Jittery
Active
Afraid
Angry
Sad

Tables

Table 1.
Correlations among Individual Early Adversities and Health Outcomes (Study 1)

Study Variable	1	2	3	4	5	SF36 Physical Functioning	Interviewer- rated health
1. Parental separation	--					.06	.08*
2. Maternal depressive symptoms	.20**	--				.08*	.09*
3. Marital satisfaction	-.38**	-.42**	--			-.07 ⁺	-.05
4. Maternal stressful life events	.27**	.37**	-.41**	--		.07	.06
5. Family income	-.36**	-.23**	.29**	-.33**	--	-.09*	-.08*

Note. * $p \leq .05$, ** $p \leq .01$, + $p < .10$

Table 2.*Correlation Matrix of Early Adversity, Stress, and Physical Health Variables (Study 1)*

Study Variable	M	SD	Range	1	2	3	4	5
1. Early adversity (by age 5)	1.64	1.36	0-4	--				
2. Social Stress (age 15)	0	1.47	-3.4-7.1	.16**	--			
3. Non-social Stress (age 15)	0	1.47	-2.7-9.0	.13**	.19**			
4. SF-36 Physical Functioning (age 20)	89.97	18.08	0-100	.14**	.13**	.19**	--	
5. Interviewer-rated physical health (age 20)	2.31	0.57	1-4.5	.09*	.19**	.21**	.17**	--
6. Presence of chronic disease (age 20)	0.23	0.42	0-1	0.06	.20**	.12**	0.05	.24**

Note. * $p \leq .05$, ** $p \leq .01$

Table 3.
Regression Analyses Predicting Health from Early Adversity (Study 1)

	SF-36 Physical Functioning (N = 619)				Interviewer-rated Health (N = 697)				Presence of Chronic Disease (N = 464)				
	<i>b</i>	<i>SE</i>	β	ΔR^2	<i>b</i>	<i>SE</i>	β	ΔR^2	<i>B</i>	<i>SE</i>	Wald	OR	95% CI
<i>Step 1</i>				0.01				0.02					
Child illness	1.62	2.27	0.03		0.26**	0.07	0.15		0.83**	0.30	7.42	2.28	1.26-4.14
Gender	2.90*	1.44	0.08		0.03	0.04	0.03		1.1**	0.25	19.94	3.07	1.88-5.02
<i>Step 2</i>				0.02				0.02					
Child illness	.88	2.26	0.02		0.24**	0.07	0.14		0.79**	0.31	6.69	2.21	1.21-4.02
Gender	2.83*	1.43	0.08		0.03	0.04	0.03		1.13**	0.25	20.08	3.08	1.88-5.04
Early adversity	1.75**	0.53	0.13		0.03 ⁺	0.02	0.07		0.07	0.08	.74	1.07	.91-1.27

Note. * $p \leq .05$, ** $p \leq .01$, + $p \leq .06$

Table 4.

Correlation Matrix of Early Adversity, Peer Psychopathology, Depressive Symptoms, and Chronic Stress (Study 2)

Study Variable	M	SD	Range	1	2	3	4	5	6
1. Early adversity (by age 5)	1.59	1.40	0-5	--					
2. Peer internalizing symptoms (age 20)	12.36	8.12	0-47	.20**	--				
3. Peer externalizing symptoms (age 20)	10.12	6.45	0-29	.13*	.49**	--			
4. Peer personality symptoms (age 20)	26.70	12.89	1-63	.17**	.71**	.62**	--		
5. Depressive symptoms (age 20)	7.05	8.40	0-52	.17**	.17**	.09	.13*	--	
6. Depressive symptoms (ages 22-25)	7.64	8.52	0-45	.18**	.18*	.05	.20**	.55**	--
7. Chronic stress (age 20)	22.59	4.52	12-39.5	.23**	.11	.06	.14*	.48**	.32**

Note. * $p \leq .05$, ** $p \leq .01$

Table 5.
Sample Demographic Characteristics (Study 3)

	<i>M (SD)</i>	Range
Age	19.92 (1.97)	18-31
Family income ^a	4.23 (1.82)	1-7
BMI	21.90 (2.69)	16.47-28.89
Time in US ^b	4.25 (1.36)	0-5
RFQ average score	2.11 (.69)	1-4.46

a On a scale from 1 to 7; a score of 4 corresponds with "\$50,000 to \$99,999"

b On a scale from 0 to 5; a score of 4 corresponds with "15+ years"

Table 6.

Descriptive Statistics and Correlations with Risky Families Questionnaire for Baseline Measures (Study 3)

Variable	Descriptive Statistics		Correlation with RFQ	
	M (SD)	Range	<i>r</i>	<i>p</i>
Early Life Experiences				
CTQ	30.45 (8.00)	20--50	0.7	< .001
Emotional abuse	8.23 (3.37)	4--19	0.52	< .001
Emotional neglect	9.84 (3.95)	4--18	0.57	< .001
Physical abuse	5.95 (1.82)	1--13	0.39	< .001
Physical neglect	6.43 (1.78)	5--12	0.5	< .001
Early adversity checklist	2.40 (2.38)	0--10	0.54	< .001
Physical Health				
SF-36				
Physical roles	91.60 (22.28)	0--100	-0.19	< .05
General health	68.51 (17.70)	20--100	-0.13	0.15
Emotional Functioning				
CESD	14.70 (9.68)	1--47	0.27	< .01
PSWQ	50.63 (15.37)	20--79	0.19	< .05
Social Relationships				
UCLA Loneliness Questionnaire	43.53 (10.56)	23--68	0.21	< .05
Rejection Sensitivity	8.83 (3.72)	1.06--24.89	0.40	< .001
PBI--maternal care	25.63 (6.36)	1--34	-0.39	< .001
PBI--maternal overprotection	12.04 (8.01)	0-33	0.11	0.23
PBI--paternal care	22.73 (7.33)	3--36	-0.35	< .001
PBI--paternal overprotection	9.79 (8.19)	0--39	0.20	< .05
Current Perceived Stress	16.48 (6.49)	3--32	0.30	< .001

Table 7.
Descriptive Statistics for Daily Diary Measures (Study 3)

Variable	<i>M (SD)</i>	Most frequently endorsed
Social Behaviors		
Aggression	1.08 (2.12)	1. Was mean to others. 2. Spread rumors or gossiped about someone.
Reassurance-seeking	1.80 (2.46)	1. Posted something on social media or checked for responses to see whether others like me. 2. Sought reassurance from people I feel close to as to whether they really care about me.
Withdrawal	1.65 (2.38)	1. Felt like being alone rather than spending time with others. 2. Avoided someone
Positive	6.45 (4.49)	1. Showed affection toward someone else. 2. Sent or received an enjoyable letter/email/phone call from someone
Stressors		
Social	1.65 (2.82)	1. Someone teased or made fun of me. 2. I was criticized by someone.
Non-social	1.69 (2.18)	1. Had to study for a difficult exam. 2. Did not have enough money to do something.
Physical Symptoms	2.58 (2.37)	1. Headache 2. Stiff or sore muscles
Negative Affect		
Fear	6.27 (2.12)	
Hostility	4.53 (1.57)	
Sadness	3.26 (1.24)	

Table 8.*Effects of Early Adversity on Daily Social Behavior (Study 3)*

Predictor	Reassurance-seeking			Withdrawal			Aggression			Positive		
	<i>b</i>	<i>SE</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>p</i>
Overall												
Intercept												
Intercept	1.55	0.3	<.001	1.67	0.35	<.001	1.3	0.4	<.01	5.81	0.63	<.001
Gender	0.39	0.41	0.35	-0.04	0.43	0.92	-0.35	0.45	0.43	0.94	0.8	0.24
RFQ score	0.72	0.27	<.01	0.72	0.38	0.06	0.47	0.25	0.07	0.78	0.58	0.19

Table 9.*Effects of Daily Stress on Daily Physical Symptoms and Negative Affect (Study 3)*

Predictor	Physical Symptoms			Negative Affect		
	<i>b</i>	<i>SE</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>p</i>
<i>For Social Stress:</i>						
Overall Intercept						
Intercept	1.59	0.31	< .001	14.46	0.63	<0.001
Social Stress Person						
Mean	0.48	0.07	< .001	0.73	0.17	<0.001
Gender	0.31	0.36	0.39	0.71	0.76	0.35
Social Stress						
Intercept	0.18	0.05	< .001	0.44	0.08	<0.001
<i>For Non-social Stress:</i>						
Overall Intercept						
Intercept	1.59	0.32	< .001	14.62	0.64	< .001
Non-Social Stress						
Person Mean	0.50	0.11	< .001	0.66	0.16	< .001
Gender	0.24	0.37	0.52	0.58	0.79	0.46
Non-Social Stress						
Intercept	0.12	0.04	< .01	0.47	0.09	< .001

Table 10.*Effects of Daily Stress and Negative Affect on Daily Physical Symptoms (Study 3)*

Predictor	<i>b</i>	<i>SE</i>	<i>p</i>
<i>For Social Stress:</i>			
Overall Intercept			
Intercept	0.90	0.37	< .05
Social Stress Person Mean	0.44	0.08	< .001
Gender	0.27	0.35	0.45
Social Stress Intercept	0.16	0.05	≤ .001
Negative Affect Intercept	0.05	0.02	< .01
<i>For Non-social Stress:</i>			
Overall Intercept			
Intercept	0.71	0.37	0.06
Non-Social Stress Person Mean	0.46	0.11	< .001
Gender	0.20	0.36	0.57
Non-Social Stress Intercept	0.1	0.04	< .05
Negative Affect Intercept	0.06	0.02	< .001

Table 11.

The Role of Early Adversity in the Effects of Daily Stress on Daily Physical Symptoms and Negative Affect (Study 3)

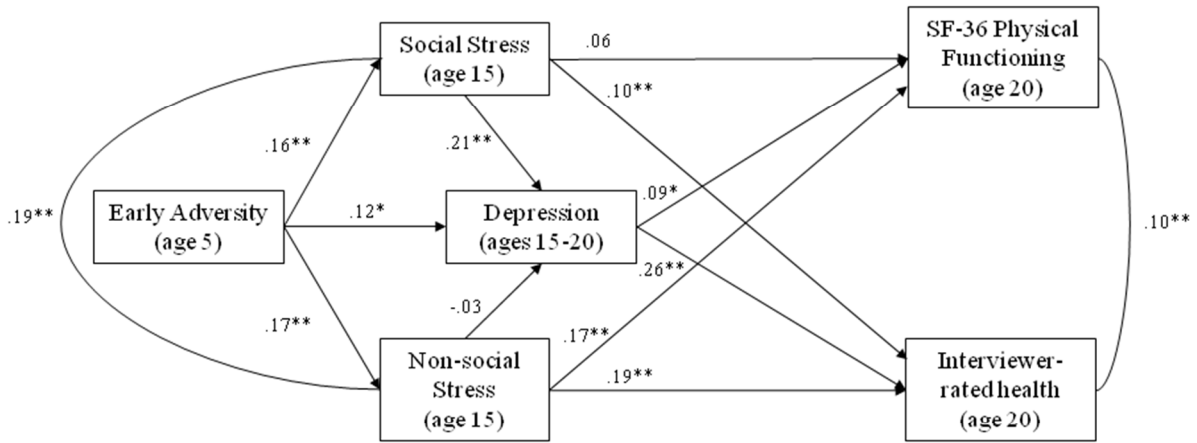
Predictor	Physical Symptoms			Negative Affect		
	<i>b</i>	<i>SE</i>	<i>p</i>	<i>b</i>	<i>SE</i>	<i>p</i>
<i>Main Effects of Early Adversity:</i>						
Overall Intercept						
Intercept	2.46	0.38	< .001	15.75	0.71	< .001
RFQ score	0.50	0.41	0.23	1.33	0.63	< .05
Gender	0.19	0.45	0.68	0.57	0.86	0.51
<i>Daily Social Stress x Early Adversity:</i>						
Overall Intercept						
Intercept	1.58	0.30	< .001	14.52	0.64	< .001
Social Stress Person Mean	0.49	0.06	< .001	0.7	0.16	< .001
RFQ score	-0.09	0.34	0.8	0.5	0.55	0.37
Gender	0.31	0.35	0.38	0.69	0.76	0.37
Social Stress						
Intercept	0.19	0.05	< .001	0.47	0.09	< .001
RFQ score	-0.03	0.06	0.57	-0.09	0.10	0.39
<i>Daily Non-social Stress x Early Adversity:</i>						
Overall Intercept						
Intercept	1.61	0.31	< .001	14.75	0.66	< .001
Non-Social Stress Person Mean	0.49	0.11	< .001	0.60	0.16	< .001
RFQ score	0.13	0.32	0.69	0.89	0.59	0.14
Gender	0.23	0.36	0.52	0.54	0.79	0.50
Non-Social Stress						
Intercept	0.12	0.05	≤ .01	0.50	0.10	< .001
RFQ score	0.03	0.05	0.61	-0.05	0.13	0.70

Table 12.*Effects of Time, Early Adversity, and Time by Early Adversity Interaction on Cortisol (Study 3)*

Predictor	<i>b</i>	<i>SE</i>	<i>p</i>
Overall intercept	3.67	0.39	< .001
Gender	0.13	0.07	0.08
Age	-0.04	0.02	< .05
Hours since waking	-0.10	0.01	< .001
RFQ	-0.02	0.05	0.70
Hours since waking x RFQ	-0.01	0.01	0.85

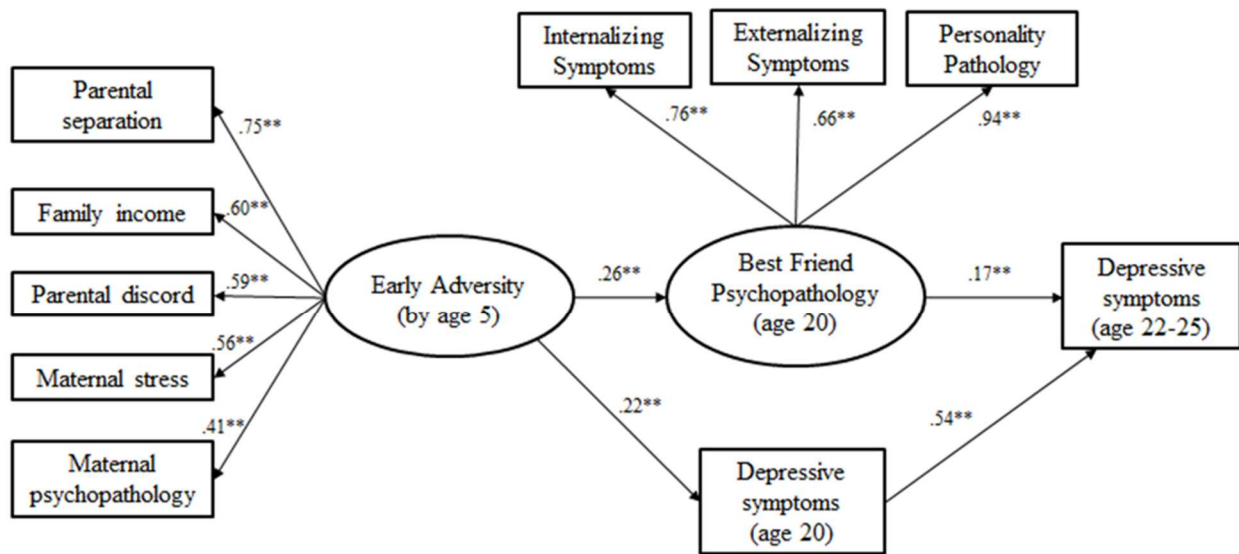
Figures

Figure 1. The effects of early adversity on SF-36 physical functioning and interviewer-rated health, via social stress, non-social stress, and depression (Study 1).



Note. * $p \leq .05$, ** $p \leq .01$

Figure 2. The effects of early adversity by age 5 on psychopathology in best friends' psychopathology at age 20, and the effects of best friends' psychopathology on target individuals' depressive symptoms 2 to 5 years later. Gender is controlled for in all paths, and youth depressive symptoms at age 20 are included as an additional mediational pathway to control for their effects on later depressive symptoms (Study 2).



* $p \leq .05$, ** $p \leq .01$

References

- Abercrombie, H. C., Giese-Davis, J., Sephton, S., Epel, E. S., Turner-Cobb, J. M., & Spiegel, D. (2004). Flattened cortisol rhythms in metastatic breast cancer patients. *Psychoneuroendocrinology, 29*(8), 1082-1092.
- Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 profile*. Burlington: University of Vermont Department of Psychiatry.
- Achenbach, T. M. (1997). *Manual for the Young Adult Self-Report and Young Adult Behavior Checklist*. Burlington: University of Vermont Department of Psychiatry.
- Adam, E.K. (2006). Transactions among adolescent trait and state emotion and diurnal and momentary cortisol activity in naturalistic settings. *Psychoneuroendocrinology, 31*, 664-679.
- Adam, E. K., & Kumari, M. (2009). Assessing salivary cortisol in large-scale, epidemiological research. *Psychoneuroendocrinology, 34*(10), 1423-1436.
- Avitsur, R., Powell, N., Padgett, D. A., & Sheridan, J. F. (2009). Social interactions, stress, and immunity. *Immunology and Allergy Clinics of North America, 29*, 285-293.
- Baker, S. R. (2006). Towards an idiographic understanding of the role of social problem solving in daily event, mood and health experiences: A prospective daily diary approach. *British Journal of Health Psychology, 11*(3), 513-531.
- Ballard, M., Cummings, E. M., & Larkin, K. (1993). Emotional and cardiovascular responses to adults' angry behavior and to challenging tasks in children of hypertensive and normotensive parents. *Child Development, 64*, 500-515.
- Batten, S. V., Aslan, M., Maciejewski, P. K., & Mazure, C. M. (2004). Childhood maltreatment

- as a risk factor for adult cardiovascular disease and depression. *Journal of Clinical Psychiatry*, 65(2), 249-254.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory-II (BDI-II)*. San Antonio, TX: Psychological Corporation.
- Bedford, A., & Foulds, G. (1978). *Manual of the Delusions-Symptoms-States Inventory (DSSI)*. NFER, Windsor.
- Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin*, 107(2), 238-246.
- Berkman, L. F., & Syme, S. L. (1979). Social networks, host resistance, and mortality: a nine-year follow-up study of Alameda County residents. *American journal of Epidemiology*, 109(2), 186-204.
- Bernet, C. Z. & Stein, M. B. (1999). Relationship of childhood maltreatment to the onset and course of major depression in adulthood. *Depression and Anxiety*, 9, 169-174.
- Bernstein, D. P., & Fink, L. (1998). *Childhood trauma questionnaire: A retrospective self-report: Manual*. Psychological Corporation.
- Bernstein, D. P., Fink, L., Handelsman, L., & Foote, J. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *The American Journal of Psychiatry*, 151(8), 1132-1136.
- Bifulco, A., Moran, P. M., Ball, C., & Lillie, A. (2002). Adult attachment style. II: Its relationship to psychosocial depressive-vulnerability. *Social Psychiatry and Psychiatric Epidemiology*, 37, 60-67.
- Black, B., & Hazen, N. L. (1990). Social status and patterns of communication in acquainted and unacquainted preschool children. *Developmental Psychology*, 26(3), 379-287.

- Bolger, N., DeLongis, A., Kessler, R. C., & Schilling, E. A. (1989). Effects of daily stress on negative mood. *Journal of Personality and Social Psychology*, 57(5), 808-818.
- Bolger, K. E., & Patterson, C. J. (2001). Developmental pathways from child maltreatment to peer rejection. *Child Development*, 72, 549–568.
- Brown, G. W., Craig, T. K., Harris, T. O., & Handley, R. V. (2008). Parental maltreatment and adulthood cohabiting partnerships: A life-course study of adult chronic depression—4. *Journal of Affective Disorders*, 110, 115-125.
- Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. *Testing Structural Equation Models*, 154, 136–162.
- Bugental, D. B., Martorell, G. A., & Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones & Behavior*, 43, 237-244.
- Buote, V. M., Pancer, S. M., Pratt, M. W., Adams, G., Birnie-Lefcovitch, S., Polivy, J., & Wintre, M. G. (2007). The importance of friends, friendship and adjustment among 1st-year university students. *Journal of Adolescent Research*, 22(6), 665-689.
- Buss, A. H., & Warren, W. L. (2000). *The Aggression Questionnaire manual*. Los Angeles: Western Psychological Services.
- Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Research*, 28(2), 193-213.
- Caplan, R. D., Cobb, S., & French, Jr., J. R. (1979). White collar work load and cortisol: Disruption of a circadian rhythm by job stress? *Journal of Psychosomatic Research*, 23, 181-192.
- Carels, R. A., Musher-Eizenman, D., Cacciapaglia, H., Pérez-Benítez, C. I., Christie, S., &

- O'Brien, W. (2004). Psychosocial functioning and physical symptoms in heart failure patients: A within-individual approach. *Journal of Psychosomatic Research, 56*(1), 95-101.
- Carlson, M. & Earls, E. (1997). Psychological and neuroendocrinological sequelae of early social deprivation in institutionalized children in Romania. *Annals of the New York Academy of Sciences, 807*, 419-428.
- Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., et al. (2007). Decreased ACTH and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry, 62*(10), 1080-1087.
- Carpenter, L. L., Shattuck, T. T., Tyrka, A. R., Geraciotti, T. D., & Price, L. H. (2011). Effect of childhood physical abuse on cortisol stress response. *Psychopharmacology, 214*, 367-375.
- Çelik, Ç., & Odacı, H. (2012). The effect of experience of childhood abuse among university students on self-perception and submissive behavior. *Children and Youth Services Review, 34*(1), 200.
- Cicchetti, D., & Toth, S. L. (1995). A developmental psychopathology perspective on child abuse and neglect. *Journal of the American Academy of Child & Adolescent Psychiatry, 34*(5), 541-565.
- Cohen, S., Doyle, W. J., Skoner, D. P., Rabin, B. S., Gwaltney, J. M. (1997). Social ties and susceptibility to the common cold. *Journal of the American Medical Association, 277*, 1940–1944.
- Cohen, S., Schwartz, J. E., Epel, E., Kirschbaum, C., Sidney, S., & Seeman, T. (2006).

- Socioeconomic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Psychosomatic Medicine*, 68(1), 41-50.
- Cohen, S., & Williamson, G. M. (1991). Stress and infectious disease in humans. *Psychological Bulletin*, 109(1), 5-24.
- Colman, R. A. & Widom, C. S. (2004). Childhood abuse and neglect and adult intimate relationships: A prospective study. *Child Abuse & Neglect*, 28, 1133-1151.
- Conaway, L. P. & Hansen, D. J. (1989). Social behavior of physically abused and neglected children: A critical review. *Clinical Psychology Review*, 9, 627-652.
- Conway T. L., Vickers R. R., Ward H. W., Rahe R. H. (1981). Occupational stress and variation in cigarette, coffee and alcohol consumption. *Journal of Health & Social Behavior*, 22, 155-165.
- Covault, J., Tennen, H., Armeli, S., Conner, T. S., Herman, A. I., Cillessen, A. H. N., & Kranzler, H. R. (2007). Interactive effects of the serotonin transporter 5-HTTLPR polymorphism and stressful life events on college student drinking and drug use. *Biological Psychiatry*, 61(5), 609–616.
- Criss, M. M., Pettit, G. S., Bates, J. E., Dodge, K. A., & Lapp, A. L. (2002). Family adversity, positive peer relationships, and children's externalizing behavior: A longitudinal perspective on risk and resilience. *Child Development*, 73(4), 1220-1237.
- Crockenberg, S. & Lourie, A. (1996). Parents' conflict strategies with children and children's conflict strategies with peers. *Merrill-Palmer Quarterly*, 42(4), 495-518.
- Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. *Proceedings of the National Academy of Sciences*, 104, 1319-1324.

- Davies, P. T., & Cummings, E. M. (1998). Exploring children's emotional security as a mediator of the link between marital relations and child adjustment. *Child Development, 69*, 124–139.
- Davis, D. A., Luecken, L. J., & Zautra, A. J. (2005). Are reports of childhood abuse related to the experience of chronic pain in adulthood: A meta-analytic review of the literature. *Clinical Journal of Pain, 21*, 398-405.
- Deinzer, R., Kleineidam, C., Stiller-Winkler, R., Idel, H., & Bachg, D. (2000). Prolonged reduction of salivary immunoglobulin A (sIgA) after a major academic exam. *International Journal of Psychophysiology, 37*, 219-232.
- DeLongis, A., Folkman, S., & Lazarus, R. S. (1988). The impact of daily stress on health and mood: Psychological and social resources as mediators. *Journal of Personality and Social Psychology, 54*(3), 486-495
- Dickerson, S., Gable, S. L., Irwin, M., Aziz, N., & Kemeny, M. (2009). Social-evaluative threat and proinflammatory cytokine regulation: An experimental laboratory investigation. *Psychological Science, 20*, 1237-1244.
- Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: Shame, physiology, and health. *Journal of Personality, 72*(6), 1191-1216.
- Dickerson, S. & Kemeny, M. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355-391.
- Dodge, K. A., Bates, J. E., & Pettit, G. S. (1990). Mechanisms in the cycle of violence. *Science, 250*, 1678–1683.
- Doherty, W. J., & Allen, W. (1994). Family functioning and parental smoking as predictors of

- adolescent cigarette use: A six-year prospective study. *Journal of Family Psychology*, 8(3), 347-353.
- Dong, M., Anda, R. F., Felitti, V. J., Dube, S. R., Williamson, D. F., Thompson, T. J., Loo, C. M., & Giles, W. H. (2004). The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect*, 28, 771-784.
- Downey, G., & Coyne, J. (1990). Children of depressed parents: An integrative review. *Psychological Bulletin*, 108, 50-76.
- Downey, G., & Feldman, S. I. (1996). Implications of rejection sensitivity for intimate relationships. *Journal of Personality and Social Psychology*, 70(6), 1327-1343.
- Dozier, M., Manni, M., Gordon, M. K., Peloso, E., Gunnar, M. R., Stovall-McClough, K. C., & Levine, S. (2006). Foster children's diurnal production of cortisol: An exploratory study. *Child Maltreatment*, 11(2), 189-197.
- Drossman, D. A., Talley, N. J., Leserman, J., Olden, K. W., & Barreiro, M. A. (1995). Sexual and physical abuse and gastrointestinal illness: Review and recommendations. *Annals of Internal Medicine*, 123, 782-794.
- Dube, S. R., Fairweather, D., Pearson, W. S., Felitti, V. J., Anda, R. F., & Croft, J. B. (2009). Cumulative childhood stress and autoimmune diseases in adults. *Psychosomatic Medicine*, 71(2), 243-250.
- Edge, M. D., Ramel, W., Drabant, E. M., Kuo, J. R., Parker, K. J., & Gross, J. J. (2009). For better or worse? Stress inoculation effects for implicit but not explicit anxiety. *Depression and anxiety*, 26(9), 831-837.
- Egeland, B., & Sroufe, A. (2006). Developmental sequelae of maltreatment in infancy. *New Directions for Child and Adolescent Development*, 118(11), 77-92.

- Ellis, W. E. & Zarbatany, L. (2007). Peer group status as a moderator of group influence on children's deviant, aggressive, and prosocial behavior. *Child Development, 78*, 1240-1254.
- Evans, P. D., & Edgerton, N. (2011). Mood states and minor illness. *British Journal of Medical Psychology, 65*(2), 177-186.
- Fantuzzo, J. W., Weiss, A. D., Atkins, M., Meyers, R., & Noone, M. (1998). A contextually relevant assessment of the impact of child maltreatment on the social competencies of low-income urban children. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 1201-1208.
- Feldman, S., & Downey, G. (1994). Rejection sensitivity as a mediator of the impact of childhood exposure to family violence on adult attachment behavior. *Development and Psychopathology, 6*, 231-247.
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventative Medicine, 14*, 245-258.
- Finkelhor, D., & Baron, L. (1986). Risk factors for child sexual abuse. *Journal of Interpersonal Violence, 1*(1), 43-71.
- Finkelhor, D., Ormrod, R. K., & Turner, H. A. (2007). Poly-victimization: A neglected component in child victimization. *Child Abuse & Neglect, 31*, 7-26.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). *Structured Clinical Interview for DSM-IV Axis I Disorders: Patient Edition (SCIDI/P. Version 2.0)*. New York, NY: New York State Psychiatric Institute.

- Ford, E., Clark, C., & Stansfeld, S. A. (2011). The influence of childhood adversity on social relations and mental health at mid-life. *Journal of Affective Disorders, 133*, 320-327.
- Frerichs, R. R., Aneshensel, C. S., Yokopenic, P. A., & Clark, V. A. (1982). Physical health and depression: An epidemiologic survey. *Preventive Medicine, 11*, 639-646.
- Gallaty, K. & Zimmer-Gembeck, M. J. (2008). The daily social and emotional worlds of adolescents who are psychologically maltreated by their romantic partners. *Journal of Youth and Adolescence, 37*, 310-323.
- Gil, K. M., Carson, J. W., Porter, L. S., Scipio, C., Bediako, S. M., & Orringer, E. (2004). Daily mood and stress predict pain, health care use, and work activity in African American adults with sickle-cell disease. *Health Psychology, 23*(3), 267-274.
- Gilbert, P. (2000). Varieties of submissive behavior as forms of social defense: Their evolution and role in depression. In L. Sloman & P. Gilbert (Eds.), *Subordination and defeat: An evolutionary approach to mood disorders and their therapy*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Gilbert, R., Widom, C. S., Browne, K., Fergusson, D., Webb, E., & Janson, S. (2009). Child maltreatment 1. Burden and consequences of child maltreatment in high-income countries. *Lancet, 373*(9657), 68-81.
- Gold, P. W., Goodwin, F. K., Chrousos, G. P. (1988). Clinical and biochemical manifestations of depression in relation to the neurobiology of stress. *New England Journal of Medicine, 319*, 413-420.
- Gonzalez, A., Jenkins, J. M., Steiner, M., & Fleming, A. S. (2009). The relation between early life adversity, cortisol awakening response and diurnal salivary cortisol levels in postpartum women. *Psychoneuroendocrinology, 34*, 76-86.

- Gottman, J., Gonso, J., & Rasmussen, B. (1975). Social interaction, social competence, and friendship in children. *Child Development, 46*, 709-718.
- Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychopathology in the National Comorbidity Survey Replication (NCS-R) I: Associations with first onset of DSM-IV disorders. *Archives of General Psychiatry, 67*(2), 1-21.
- Gude, T., Moum, T., Kaldestad, E., & Friis, S. (2000). Inventory of interpersonal problems: A three-dimensional balanced and scalable 48-item version. *Journal of Personality Assessment, 74*, 296-310.
- Gunnar, M. R., Morison, S. J., Chisholm, K., Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Developmental Psychopathology, 13*, 611-628.
- Gunnar, M. R. & Vasquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology, 13*, 515-538.
- Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology, 1*, 293-319.
- Hammen, C., Hazel, N., Brennan, P., & Najman, J. (2012). Intergenerational transmission and continuity of stress and depression: Depressed women and their offspring in 20 years of follow-up. *Psychological Medicine, 42*, 931-942.
- Hankin B. L. (2005). Childhood maltreatment and psychopathology: Prospective tests of attachment, cognitive vulnerability, and stress as mediating processes. *Cognitive Therapy and Research, 29*, 645-671.
- Hart, C. H., Nelson, D. A., Robinson, C. C., Olsen, S. F., & McNeilly-Choque, M. K. (1998).

- Overt and relational aggression in Russian nursery-school-age children: Parenting style and marital linkages. *Developmental Psychology*, 34(4), 687-697.
- Hazel, N. A., Hammen, C., Brennan, P. A., & Najman, J. (2008). Early childhood adversity and adolescent depression: The mediating role of continued stress. *Psychological Medicine*, 38, 581-589.
- Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, 25, 1-35.
- Heim, C. & Nemeroff, C. B. (2001). The role of childhood trauma in the neurobiology of mood and anxiety disorders: Preclinical and clinical studies. *Biological Psychiatry*, 49, 1023-1039.
- Hill, E. M., Young, J. P., & Nord, J. L. (1994). Childhood adversity, attachment security, and adult relationships: A preliminary study. *Ethology and Sociobiology*, 15(5), 323-338.
- Hogue, A. & Steinberg, L. (1995). Homophily of internalized distress in adolescent peer groups. *Developmental Psychology*, 31(6), 897-906.
- Holt-Lunstad, J., Uchino, B. N., Smith, T. W., Olson-Cerny, C., & Nealey-Moore, J. B. (2003). Social relationships and ambulatory blood pressure: Structural and qualitative predictors of cardiovascular function during everyday social interactions. *Health Psychology*, 22, 288-297.
- House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science*, 241, 540-545.
- Howe, R. T. & Parke, R. D. (2001). Friendship quality and sociometric status: Between-group differences and links to loneliness in severely abused and nonabused children. *Child Abuse & Neglect*, 25, 585-606.

- Howes, C. & Espinosa, M. P. (1985). The consequences of child abuse for formation of relationships with peers. *Child Abuse & Neglect, 9*, 397-404.
- Howes, M. J., Hokanson, J. E., & Loewenstein, D. A. (1985). Induction of depressive affect after prolonged exposure to a mildly depressed individual. *Journal of Personality and Social Psychology, 49*(4), 1110-1113.
- Hu, L. & Bentler, P. M. (1998). Fit indices in covariance structural equation modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods, 3*, 424-453.
- Hyder, S. E., Rieder, R. O., Williams, J. B., Spitzer, R. L., Hendler, J., & Lyons, M. (1988). The Personality Diagnostic Questionnaire: development and preliminary results. *Journal of Personality Disorders, 2*(3), 229-237.
- Johnson, V. & Pandina, R. J. (1991). Effects of the family environment on adolescent substance use, delinquency, and coping styles. *The American Journal of Drug and Alcohol Abuse, 17*, 71-88.
- Joiner, T. E. (1994). Contagious depression: Existence, specificity to depressed symptoms, and the role of reassurance seeking. *Journal of Personality and Social Psychology, 67*(2), 287-296.
- Joiner, T. E., Alfano, M. S., & Metalsky, G. I. (1992). When depression breeds contempt: Reassurance seeking, self-esteem, and rejection of depressed college students by their roommates. *Journal of Abnormal Psychology, 101*(1), 165-173.
- Kamarck, T. W., Shiffman, S., Smithline, L., Goodie, J., Thompson, H., Ituarte, P. H., et al. (1998). The Diary of Ambulatory Behavioral States: A new approach to the assessment of ambulatory cardiovascular activity. In D. Krantz & A. Baum (Eds.), *Perspectives in*

- behavioral medicine: Technology and methodology in behavioral medicine* (pp. 161–193). Hillsdale, NJ: Erlbaum.
- Karlamangla, A. S., Friedman, E. M., Seeman, T. E., Stawski, R. S., & Almeida, D. M. (2013). Daytime trajectories of cortisol: Demographic and socioeconomic differences—Findings from the National Study of Daily Experiences. *Psychoneuroendocrinology*, *38*(11), 2585–2597.
- Kaufman, J., & Cicchetti, D. (1989). Effects of maltreatment on school-age children's Ssocioemotional development: Assessments in a day-camp setting. *Developmental Psychology*, *25*(4), 516–524.
- Kaufman, J., Yang, B. Z., Douglas-Palumberi, H., Crouse-Artus, M., Lipschitz, D., Krystal, J. H., & Gelernter, J. (2007). Genetic and environmental predictors of early alcohol use. *Biological Psychiatry*, *61*(11), 1228–1234.
- Keeping, J. D., Najman, J. M., Morrison, J., Western, J. S., Andersen, M. J., & Williams, G. M. (1989). A prospective longitudinal study of social, psychological and obstetric factors in pregnancy: response rates and demographic characteristics of the 8556 respondents. *BJOG: An International Journal of Obstetrics & Gynaecology*, *96*(3), 289–297.
- Kendler, K. S., Kuhn, J., & Prescott, C. A. (2004). The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression. *American Journal of Psychiatry*, *161*(4), 631–636.
- Kerns, K. A., Klepac, L., & Cole, A. K. (1996). Peer relationships and preadolescents' perceptions of security in the child–mother relationship. *Developmental Psychology*, *32*, 457–466.
- Kessler, R. C., Davis, C. G., & Kendler, K. S. (1997). Childhood adversity and adult psychiatric

- disorder in the US National Comorbidity Survey. *Psychological Medicine*, 27, 1101-1119.
- Kessler, R. C., & Magee, W. J. (1993). Childhood adversities and adult depression: basic patterns of association in a US national survey. *Psychology Medicine*, 23, 679-679.
- Kessler, R. C. & Magee, W. J. (1994). Childhood family violence and adult recurrent depression. *Journal of Health and Social Behavior*, 35, 13-27.
- Kiecolt-Glaser, J. K. & Glaser, R. (2002). Depression and immune function: Central pathways to morbidity and mortality. *Journal of Psychosomatic Research*, 53, 873-876.
- Kim, J., & Cicchetti, D. (2010). Longitudinal pathways linking child maltreatment, emotion regulation, peer relations, and psychopathology. *Journal of Child Psychology and Psychiatry*, 51(6), 706-716.
- Kong, S. & Bernstein, K. (2009). Childhood trauma as a predictor of eating psychopathology and its mediating variables in patients with eating disorders. *Journal of Clinical Nursing*, 18, 1897-1907.
- Kroenke, K., Spitzer, R. L., Williams, J. B. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, 16, 606-613.
- Kunz-Ebrecht, S. R., Kirschbaum, C., Marmot, M., & Steptoe, A. (2004). Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoneuroendocrinology*, 29(4), 516-528.
- Lansford, J. E., Miller-Johnson, S., Berlin, L. J., Dodge, K. A., Bates, J. E., & Pettit, G. S. (2007). Early physical abuse and later violent delinquency: A prospective longitudinal study. *Child Maltreatment*, 12(3), 233-245.
- Larkin, K.T., Frazer, N.L., Semenchuk, E.M. (1996). Physiological, affective, and behavioral

- responses to interpersonal conflict among males from families with difference levels of cohesion and adaptability. *Journal of Psychopathology and Behavioral Assessment*, 18(3), 239-254.
- Larkin, K.T., Frazer, N.L., Wheat, A.L. (2011). Responses to interpersonal conflict among young adults: influences of family of origin. *Personal Relationships*, 18, 657-667.
- Lehman, B. J., Taylor, S. E., Kiefe, C. I., & Seeman, T. E. (2005). Relation of childhood socioeconomic status and family environment to adult metabolic functioning in the CARDIA study. *Psychosomatic Medicine*, 67(6), 846-854.
- Levy, R. L., Cain, K. C., Jarrett, M., & Heitkemper, M. M. (1997). The relationship between daily life stress and gastrointestinal symptoms in women with irritable bowel syndrome. *Journal of Behavioral Medicine*, 20, 177-193.
- Luecken, L.J. (1998). Childhood attachment and loss experiences affect adult cardiovascular and cortisol functioning. *Psychosomatic Medicine*, 60, 763-772.
- Luecken, L. J., & Appelhans, B. M. (2006). Early parental loss and salivary cortisol in young adulthood: the moderating role of family environment. *Development and Psychopathology*, 18(1), 295-308.
- Luecken, L.J., Rodriguez, A.P., Appelhans, B.M. (2005). Cardiovascular stress responses in young adulthood associated with family-of-origin relationship experiences. *Psychosomatic Medicine*, 67, 514-521.
- MacLean, P. D. (1990). *The Triune Brain in Evolution*. New York: Plenum.
- Maes, M. (2008). The cytokine hypothesis of depression: inflammation, oxidative & nitrosative stress (IO&NS) and leaky gut as new targets for adjunctive treatments in depression. *Neuroendocrinology Letters*, 29(3), 287-291.

- Manly, J. T., Kim, J. E., Rogosch, F. A., & Cicchetti, D. (2001). Dimensions of child maltreatment and children's adjustment: Contributions of developmental timing and subtype. *Development and Psychopathology, 13*(04), 759-782.
- Matthews, K., Schwartz, J., Cohen, S., & Seeman, T. (2006). Diurnal cortisol decline is related to coronary calcification: CARDIA study. *Psychosomatic Medicine, 68*, 657-661.
- McCarthy, G., & Taylor, A. (1999). Avoidant/ambivalent attachment style as a mediator between abusive childhood experiences and adult relationship difficulties. *Journal of Child Psychology and Psychiatry, 40*(3), 465-477.
- McCloskey, L. A. & Stuewig, J. (2001). The quality of peer relationships among children exposed to family violence. *Development and Psychopathology, 13*, 83-96.
- McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*(3), 171-179.
- McHorney, C. A., Ware, Jr., J. E., & Raczek, A. E. (1993). The MOS 36-item Short-Form Health Survey (SF-36): II. Psychometric and clinical tests of validity in measuring physical and mental health constructs. *Medical Care, 31*, 247-263.
- Mercer, S. H. & Derosier, M. E. (2010). Selection and socialization of internalizing problems in middle childhood. *Journal of Social and Clinical Psychology, 29*, 1031-1056.
- Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State worry questionnaire. *Behaviour Research and Therapy, 28*(6), 487-495.
- Miller, G. E., Rohleder, N., Cole, S. W. (2009). Chronic interpersonal stress predicts activation of pro- and anti-inflammatory signaling pathways 6 months later. *Psychosomatic Medicine, 71*, 57-62.

- Morales, J. R., & Crick, N. (1999, April). *Hostile attribution and aggression in adolescent peer and romantic relationships*. Poster session presented at the biennial meeting of the Society for Research in Child Development, Albuquerque, NM.
- Mounts, N. A. & Steinberg, L. (1995). An ecological analysis of peer influence on adolescent grade point average and drug use. *Developmental Psychology*, 31(6), 915-922.
- Mroczek, D. K., & Almeida, D. M. (2004). The effect of daily stress, personality, and age on daily negative affect. *Journal of Personality*, 72(2), 355-378.
- Mullen, P. E., Martin, J. L., Anderson, J. C., Romans, S. E., & Herbison, G. P. (1996). The long-term impact of the physical, emotional, and sexual abuse of children: a community study. *Child Abuse & Neglect*, 20(1), 7-21.
- Muller, R. T., Thornback, K., & Bedi, R. (2012). Attachment as a mediator between childhood maltreatment and adult symptomatology. *Journal of Family Violence*, 27, 243-255.
- Muthén, L. K., & Muthén, B. O. (1998–2007). Mplus User's Guide, 5th edition. Los Angeles,, CA: Author.
- Nicolson, N. A. (2004). Childhood parental loss and cortisol levels in adult men. *Psychoneuroendocrinology*, 29, 1012-1018.
- Nicolson, N. A., Davis, M. C., Kruszewski, D., & Zautra, A. J. (2010). Childhood maltreatment and diurnal cortisol patterns in women with chronic pain. *Psychosomatic Medicine*, 72, 471-480.
- Ornduff, S. R., Kelsey, R. M., & O'Leary, K. D. (2001). Childhood physical abuse, personality, and adult relationship violence: A model of vulnerability to victimization. *American Journal of Orthopsychiatry*, 71(3), 322-331.
- Paik, I. H., Toh, K. Y., Lee, C., Kim, J. J., & Lee, S. J. (2000). Psychological stress may induce

- increased humoral and decreased cellular immunity. *Behavioral Medicine*, 26(3), 139-141.
- Pariante, C. M. & Lightman, S. L. (2008). The HPA axis in major depression: Classical theories and new developments. *Trends in Neurosciences*, 31, 464-468.
- Parker, G. (1983) Parental Overprotection: A Risk Factor in Psychosocial Development. New York, NY: Grune & Stratton.
- Parker, J. G., Rubin, K. H., Erath, S., Wojslawowicz, J. C., & Buskirk, A. (2006). Peer relationships, child development, and adjustment: A developmental psychopathology perspective. In D. Cicchetti (Ed.), *Developmental Psychopathology: Vol. 2. Risk, disorder, and adaptation* (419–93). New York: Wiley.
- Parker, G., Tupling, H., & Brown, L. B. (2011). A parental bonding instrument. *British Journal of Medical Psychology*, 52(1), 1-10.
- Pearlin, L. I. (1989). The sociological study of stress. *Journal of Health and Social Behavior*, 30, 241-256.
- Pennebaker, J. W. (1982). *The Psychology of Physical Symptoms*. Springer.
- Pettit, G. S., Bates, J. E., & Dodge, K. A. (1997). Supportive parenting, ecological context, and children's adjustment: A seven-year longitudinal study. *Child Development*, 68, 908-923.
- Phillips, N. K., Hammen, C. L., Brennan, P. A., Najman, J. M., & Bor, W. (2005). Early diversity and the prospective prediction of depressive and anxiety disorders in adolescents. *Journal of Abnormal Child Psychology*, 33(1), 13-24.
- Posener, J. A., Schildkraut, J. J., Samson, J. A., & Schatzberg, A. F. (1996). Diurnal variation of plasma cortisol and homovanillic acid in healthy subjects. *Psychoneuroendocrinology*, 21, 33-38.

- Power, C., Hypponen, E., & Smith, G. D. (2005). Socioeconomic position in childhood and early adult life and risk of mortality: A prospective study of the mothers of the 1958 British Birth Cohort. *American Journal of Public Health, 95*, 1-7.
- Prinstein, M. J., Cheah, C. S., Borelli, J. L., Simon, V. A., & Aikins, J. W. (2005). Adolescent girls' interpersonal vulnerability to depressive symptoms: A longitudinal examination of reassurance-seeking and peer relationships. *Journal of Abnormal Psychology, 114*, 676-88.
- Prinstein, M. J., Meade, C. S., & Cohen, G. L. (2003). Adolescent oral sex, peer popularity, and perceptions of best friends' sexual behavior. *Journal of Pediatric Psychology, 28*(4), 243-249.
- Pruessner, J. C., Hellhammer, D. H., & Kirschbaum, C. (1999). Burnout, perceived stress, and cortisol responses to awakening. *Psychosomatic Medicine, 61*, 197-204.
- Raison, C. L. & Miller, A. H. (2003). When not enough is too much: The role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. *American Journal of Psychiatry, 160*, 1554-1565.
- Rao, H., Betancourt, L., Giannetta, J. M., Brodsky, N. L., Korchykowski, M., Avants, B. B., ... & Farah, M. J. (2010). Early parental care is important for hippocampal maturation: evidence from brain morphology in humans. *Neuroimage, 49*(1), 1144-1150.
- Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical Linear Models: Applications and Data Analysis Methods, Second Edition*. Newbury Park, CA: Sage.
- Reiche, E. M., Nunes, S. O., & Morimoto, H. K. (2004). Stress, depression, the immune system, and cancer. *The Lancet Oncology, 5*, 617-625.

- Repetti, R.L., Robles, T.F., & Reynolds, B. (2011). Allostatic processes in the family. *Development and Psychopathology, 23*, 921-938.
- 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*, 330-366.
- Robbins, P. R., Meyersburg, H. A., & Tanck, R. H. (1974). Interpersonal stress and physical complaints. *Journal of Personality Assessment, 38*(6), 578-585.
- Rosenquist, J. N., Fowler, J. H., & Christakis, N. A. (2010). Social network determinants of depression. *Molecular Psychiatry, 16*(3), 273-281.
- Russell, D., Peplau, L. A., Cutrona, C. E. (1980). The revised UCLA Loneliness Scale: Concurrent and discriminant validity evidence. *Journal of Personality and Social Psychology, 39*, 472-480.
- Sahl, J. C., Cohen, L. H., & Dasch, K. B. (2009). Hostility, interpersonal competence, and daily dependent stress: A daily model of stress generation. *Cognitive Therapy and Research, 33*(2), 199-210.
- Sánchez, M. M., Noble, P. M., Lyon, C. K., Plotsky, P. M., Davis, M., Nemeroff, C. B., & Winslow, J. T. (2005). Alterations in diurnal cortisol rhythm and acoustic startle response in nonhuman primates with adverse rearing. *Biological Psychiatry, 57*(4), 373-381.
- Scaramella, L. V., Conger, R. D., Simons, R. L., & Whitbeck, L. B. (1998). Predicting risk for pregnancy by late adolescence: A social contextual perspective. *Developmental Psychopathology, 34*, 1233-1245.
- Scher, C. D., Forde, D. R., McQuaid, J. R., & Stein, M. B. (2004). Prevalence and demographic

- correlates of childhood maltreatment in an adult community sample. *Child Abuse & Neglect*, 28, 167-180.
- Schilling, E. A., Aseltine, R. H., & Gore, S. (2007). Adverse childhood experiences and mental health in young adults: A longitudinal survey. *BMC Public Health*, 7(30), 1-10.
- Scholz, U., Kliegel, M., Luszczynska, A., & Knoll, N. (2012). Associations between received social support and positive and negative affect: evidence for age differences from a diary-study. *European Journal of Ageing*, 9(4), 361-371.
- Seidlitz, L., & Diener, E. (1993). Memory for positive versus negative life events: Theories for the differences between happy and unhappy persons. *Journal of Personality and Social Psychology*, 64(4), 654-663.
- Selye, H. (1956). *The Stress of Life*. New York, NY: McGraw-Hill.
- Sephton, S. E., Sapolsky, R. M., Kraemer, H. C., & Spiegel, D. (2000). Diurnal cortisol rhythm as a predictor of breast cancer survival. *Journal of the National Cancer Institute*, 92(12), 994-1000.
- Shahar, G., Henrich, C. C., Reiner, I. C., & Little, T. D. (2003). Development and initial validation of the brief adolescent life event scale (BALES). *Anxiety, Stress & Coping*, 16(1), 119-128.
- Shaver, P. R., Schachner, D. A., Mikulincer, M. (2005). Attachment style, excessive reassurance seeking, relationship processes, and depression. *Personality and Social Psychology Bulletin*, 31, 343-359.
- Slavich, G. M., O'Donovan, A., Epel, E. S., & Kemeny, M. E. (2010). Black sheep get the blues: A psychobiological model of social rejection and depression. *Neuroscience & Biobehavioral Reviews*, 35, 39-45.

- Slopen, N., Koenen, K. C., & Kubzansky, L. D. (2012). Childhood adversity and immune and inflammatory biomarkers associated with cardiovascular risk in youth: A systematic review. *Brain, Behavior, & Immunity, 26*, 239-250.
- Snijders, T. A. B., & Bosker, R. J. (1999). *Multilevel Analysis*. Sage Publications London, UK.
- Spanier, G. B. (1976). Measuring dyadic adjustment: New scales for assessing the quality of marriage and similar dyads. *Journal of Marriage and the Family, 38*, 15–28.
- Springer, K. W., Sheridan, J., Kuoc, D., & Carnes, M. (2007). Long-term physical and mental health consequences of childhood physical abuse: Results from a large population based sample of men and women. *Child Abuse & Neglect, 31(5)*, 517-530.
- Starr, L., Hammen, C., Conway, C. C., Raposa, E. B., & Brennan, P. A. (*in press*). Sensitizing effect of early adversity on depressive reactions to later proximal stress: Moderation by 5-HTTLPR and CRHR1 in a 20-year longitudinal study. *Development and Psychopathology*.
- Steptoe, A., Wardle, J., Pollard, T., Canaan, L., & Davies, G. (1996). Stress, social support and health-related behavior: A study of smoking, alcohol consumption and physical exercise. *Journal of Psychosomatic Research, 41*, 171-180.
- Stevens, E. A., & Prinstein, M. J. (2005). Peer contagion of depressogenic attributional styles among adolescents: A longitudinal study. *Journal of Abnormal Child Psychology, 33(1)*, 25-37.
- Stronach, E. P., Toth, S. L., Rogosch, F., Oshri, A., Manly, J. T., & Cicchetti, D. (2011). Child maltreatment, attachment security, and internal representations of mother and mother-child relationships. *Child Maltreatment, 16(2)*, 137-145.
- Styron, T. & Janoff-Bulman, R. (1997). Childhood attachment and abuse: Long-term effects on

- adult attachment, depression, and conflict resolution. *Child Abuse & Neglect*, *21*, 1015-1023.
- Swenson, L. M., Nordstrom, A., & Hiester, M. (2008). The role of peer relationships in adjustment to college. *Journal of College Student Development*, *49*, 551-567.
- Taylor, S. E., Karlamangla, A. S., Friedman, E. M., & Seeman, T. E. (2011). Early environment affects neuroendocrine regulation in adulthood. *Social Cognitive & Affective Neuroscience*, *6*(2), 244-251.
- Taylor, S. E., Lehman, B. J., Kiefe, C. I., & Seeman, T. E. (2006). Relationship of early life stress and psychological functioning to adult C-reactive protein in the coronary artery risk development in young adults study. *Biological Psychiatry*, *60*, 819-824.
- Taylor, S. E., Lerner, J. S., Sage, R. M., Lehman, B. J., & Seeman, T. E. (2004). Early environment, emotions, responses to stress, and health. *Journal of Personality*, *72*(6), 1365-1394.
- Teisl, M. & Cicchetti, D. (2007). Physical abuse, cognitive and emotional processes, and aggressive/disruptive behavior problems. *Social Development*, *17*, 1-23.
- Tottenham, N., Hare, T. A., Quinn, B. T., McCarry, T. W., Nurse, M., Gilhooly, T., ... & Casey, B. J. (2010). Prolonged institutional rearing is associated with atypically large amygdala volume and difficulties in emotion regulation. *Developmental Science*, *13*(1), 46-61.
- Trickett, P. K., & McBride-Chang, C. (1995). The developmental impact of different forms of child abuse and neglect. *Developmental Review*, *15*, 311-337.
- Troxel, W. M., Matthews, K. A., Gallo, L. C., & Kuller, L. H. (2005). Marital quality and occurrence of the metabolic syndrome in women. *Archives of Internal Medicine*, *165*, 1022-1027.

- Turner H. A., Butler M. J. (2003). Direct and indirect effects of childhood adversity on depressive symptoms in young adults. *Journal of Youth and Adolescence*, 32, 89–103.
- Turner, R. J. & Lloyd, D. A. (1995). Lifetime traumas and mental health: The significance of cumulative adversity. *Journal of Health and Social Behavior*, 36, 360–376.
- Turner, R. J., Wheaton, B., & Lloyd, D. A. (1995). The epidemiology of social stress. *American Sociological Review*, 104-125.
- Uhrlass D. J. & Gibb B. E. (2007). Childhood emotional maltreatment and the stress generation model of depression. *Social and Clinical Psychology*, 26, 119–130.
- Valentiner, D. P., Holahan, C. J., & Moos, R. H. (1994). Social support, appraisals of event controllability, and coping: An integrative model. *Journal of Personality and Social Psychology*, 66(6), 1094-1102.
- Van der Vegt, E. J., van der Ende, J., Kirschbaum, C., Verhulst, F. C., & Tiemeier, H. (2009). Early neglect and abuse predict diurnal cortisol patterns in adults: A study of international adoptees. *Psychoneuroendocrinology*, 34, 660-669.
- Van Lieshout, C. F., Cillessen, A. H., & Haselager, G. J. (1999). Interpersonal support and individual development. In W. A. Collins & B. P. Laursen (Eds.), *Relationships as Developmental Contexts* (37-55). Psychology Press.
- Van Zalk, M. H. W., Kerr, M., Branje, S. J. T., Stattin, H., & Meeus, W. H. J. (2010). It takes three: Selection, influence, and de-selection processes of depression in adolescent friendship networks. *Developmental Psychology*, 46, 927-38.
- Walker, L. S., Garber, J., Smith, C. A., Van Slyke, D. A., & Claar, R. L. (2001). The relation of daily stressors to somatic and emotional symptoms in children with and without recurrent abdominal pain. *Journal of Consulting and Clinical Psychology*, 69(1), 85-91.

- Ware, J. E., & Gandek, B. (1998). Overview of the SF-36 health survey and the international quality of life assessment (IQOLA) project. *Journal of Clinical Epidemiology*, 51(11), 903-912.
- Ware, Jr., J. E. & Sherbourne, C. D. (1992). The MOS 36-Item Short-Form Health Survey (SF-36): I. Conceptual framework and item selection. *Medical Care*, 30, 473-483.
- Ware, Jr., J. E., Snow, K. K., & Kosinski, M. SF-36 Health Survey: Manual and Interpretation *Guide*. Quality Metric Incorporated; Lincoln, RI: 2000.
- Watson, D., & Clark, L. A. (1999). The PANAS-X: Manual for the positive and negative affect schedule-expanded form.
- Weiner, H. (1992). *Perturbing the Organism: The biology of stressful experience*. Chicago: University of Chicago Press.
- Weissbecker, I., Floyd, A., Dedert, E., Salmon, P., & Sephton, S. (2005). Childhood trauma and diurnal cortisol disruption in fibromyalgia syndrome. *Psychoneuroendocrinology*, 31, 312-324.
- Wickrama, K. S., Conger, R. D., & Abraham, W. T. (2005). Early adversity and later health: The intergenerational transmission of adversity through mental disorder and physical illness. *The Journals of Gerontology*, 60, S125-S129.
- Widom, C. S., & White, H. R. (1997). Problem behaviours in abused and neglected children grown up: prevalence and co-occurrence of substance abuse, crime and violence. *Criminal Behaviour and Mental Health*, 7(4), 287-310.
- Winer, Jeffrey P. (2013). Associations of childhood family adversity and pubertal timing with depressive symptomatology in adulthood. *Masters Theses*. Paper 1094.
<http://scholarworks.umass.edu/theses/1094>

- Wisner Fries, A. B., Shirtcliff, E. A., & Pollak, S. D. (2008). Neuroendocrine dysregulation following early social deprivation in children. *Developmental Psychobiology*, *50*(6), 588-599.
- Wonderlich, S. A., Rosenfeldt, S., Crosby, R. D., Mitchell, J. E., Engel, S. G., Smyth, J., & Miltenberger, R. (2007). The effects of childhood trauma on daily mood lability and comorbid psychopathology in bulimia nervosa. *Journal of Traumatic Stress*, *20*(1), 77-87.
- Yeh, E. T., & Willerson, J. T. (2003). Coming of age of C-reactive protein. *Circulation*, *107*(3), 370-371.
- Zautra, A. J., Guarnaccia, C. A., & Dohrenwend, B. P. (1986). Measuring small life events. *American Journal of Community Psychology*, *14*(6), 629-655.