How Childhood Environments and Parental Vigilance Shape Health Across the Lifespan

by

KELSEY L. CORALLO

(Under the Direction of Katherine B. Ehrlich)

ABSTRACT

Childhood environments matter for health: Greater risk exposure (e.g., living in poverty; experiencing maltreatment) predicts poor health throughout the lifespan. However, many individuals who grow up in risky environments remain healthy and free from chronic illness. Supportive relationships, particularly parent-child relationships, promote healthy development in risky contexts and buffer children and adults from experiencing negative physical and mental health outcomes. However, characteristics that constitute responsive and supportive parenting may differ depending on the environmental settings in which families are embedded. Previous research has shown that high parental vigilance, or parenting behaviors that seek to monitor, surveille, and even control children and adolescents, is associated with positive psychosocial adjustment outcomes for individuals in high-risk contexts. Conversely, among low-risk samples, high parental vigilance is related to negative outcomes, or unrelated to outcomes altogether. Therefore, the research presented in this dissertation seeks to expand upon the body of literature that examines the direct role of parental vigilance on health in risky contexts, and the moderating role of parental vigilance in the relation between childhood risk and health. Findings generally support the notion that the effects of parental vigilance are indeed context-dependent and suggest that efforts to promote high-quality parenting should consider families' environmental setting.

INDEX WORDS: Childhood experiences, Childhood risk, Childhood environment, Parental monitoring, Parental control, Parental vigilance, Physical health, Behavioral health

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DEDICATION

I would like to dedicate this document to my pre-kindergarten students and their families. You all showed me that I have the strength, patience, and perseverance to do hard things!

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CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

Childhood stressors have long-lasting ties to physical and emotional wellbeing, such that growing up in a high-risk setting (e.g., experiencing poverty or lack of resources, family chaos, and/or hazardous home or neighborhood environments) is associated with chronic health conditions throughout the lifespan (e.g., Allen et al., 2014; Ellen et al., 2001; Miller et al., 2011). Over two decades ago, the Adverse Childhood Experiences (ACEs) study explored the association between the number of adversities individuals experienced in childhood (e.g., physical, sexual, and psychological abuse; household dysfunction) and the likelihood of physical and mental illnesses in adulthood (Felitti et al., 1998). Results revealed a relation between ACEs and the likelihood of experiencing health conditions such as severe obesity, cardiovascular disease, cancer, lung disease, skeletal fractures, and liver disease among individuals who reported four or more adverse events (Felitti et al., 1998). ACEs were also related to poor health behaviors (e.g., drug and alcohol use), depressive episodes, and suicide attempts (Felitti et al., 1998).

Several frameworks—developed before and in the years following the seminal ACEs study—propose mechanisms by which childhood adversity leads to poor health in adulthood. One framework that explains this process is allostatic load, which posits that social and psychological stress cause wear-and-tear to the body's physiological set-points (known as allostasis) over time; eventually, the stress-induced changes to allostasis and resultant "allostatic load" overwhelms and makes the brain and body more susceptible to acute and chronic illness

(e.g., Beckie, 2012; McEwen, 1998a; McEwen, 1998b; McEwen & Gianaros, 2011; McEwen & Stellar, 1993). Similarly, the biological embedding framework developed by Miller et al. (2011) suggests that childhood stressors are "biologically embedded" such that they beget alterations and disruptions to the stress-response (e.g., the hypothalamic-pituitary-adrenal and sympathetic-adreno-medullar axes) and immune systems and dysregulate hormone production; these physical maladjustments result in chronic diseases of aging (e.g., diabetes, heart disease, and some cancers).

Additionally, growing up in a toxic or under-resourced environment in childhood may serve as a direct predictor of poor health conditions and behaviors that contribute to the prevalence of chronic disease. For example, neighborhood disrepair and air pollution are associated with asthma prevalence in childhood and adulthood (Gold & Wright, 2005; Wright & Subramanian, 2006), and poverty and lack of access to a healthy diet are associated with higher obesity rates (Ludwig et al., 2011; Diez Roux & Mair, 2010). These health conditions are often precursors to, and co-morbid with, other chronic diseases, such as lung disease and diabetes (e.g., Christine et al., 2015; Diez Roux & Mair, 2010). Additionally, individuals in risky environments may engage in poor health behaviors (e.g., smoking and substance use) to cope with stressors (e.g., Wills, 1986; Wills, 1990; Kassel et al., 2003). In turn, these maladaptive behaviors give rise to greater incidences of chronic diseases such as heart disease and cancer (Schulte & Hser, 2013).

Taken together, adverse experiences (e.g., maltreatment and family dysfunction) as well as risky home and neighborhood environments in childhood are thought to leave a biological residue that can result in physical and emotional illness later in life. However, many children who experience adversity or grow up in dangerous home or neighborhood environments remain

physically and emotionally healthy in adulthood. Factors that promote healthy development regardless of environment (known as "promotive factors") and factors that specifically offset the risk for negative outcomes among individuals who experience childhood adversity (i.e., "protective factors") reduce the risk for negative long-term health consequences associated with ACEs and risky home and neighborhood environments (Garmezy, 1993; Luthar et al., 2000; Masten, 2014). For example, emotion regulation, supportive social relationships, and receiving a high-quality education are promotive factors—they lead to positive outcomes in any environmental context.

Although supportive and nurturing parenting are promotive, such that all children benefit from high-quality parent-child relationships no matter their circumstances, characteristics that constitute a supportive, nurturing parent may vary depending on context (e.g., socioeconomic or cultural conditions; Omer et al., 2016; Soenens et al., 2015). Vigilant parenting, or parenting behaviors that seek to monitor, surveille, or even control children (e.g., Varner et al., 2018), has been found to be both protective (i.e., buffer against negative outcomes) and have compensatory effects (i.e., promote positive outcomes; Zimmerman et al., 2013) in high-risk settings. To elaborate, in low-resourced environments where children are exposed to dangerous adults or peers and can easily access harmful substances (e.g., illicit drugs use or alcohol), parental control and vigilance may offset delinquency and indicate parents' efforts to protect their children from danger (e.g., Furstenberg, 1993; Pinderhughes et al., 2007; Roche, 2007). However, in high-resourced environments where children's access to precarious social situations and substances is low, parental control and vigilance may reflect parents' efforts to inhibit children's autonomy unnecessarily (Omer et al., 2016; Soenens et al. 2015). Therefore, high levels of parental control and vigilance are a protective factor that can offset behaviors that lead to poor health in

dangerous or risky contexts but may be maladaptive in the absence of social and environmental threats.

Indeed, previous literature has shown that parental vigilance offsets negative short-term psychosocial development outcomes and halts upwards trajectories of delinquency in young adulthood. For example, greater parental control is associated with better social functioning and lower levels of externalizing behaviors among high-risk samples of adolescents (Mason et al., 1996; McElhaney & Allen, 2001; Galambos et al. 2003). Additionally, parental monitoring, or efforts to surveille children's whereabouts and activities, in adolescence is associated with a lower likelihood of engaging in risky health behaviors and substance use both throughout adolescence and into young adulthood (e.g., Donaldson et al., 2016; Ethier et al. 2016; Lee et al., 2014; Varner et al., 2018).

Conversely, parental monitoring and control are unrelated to, and sometimes maladaptive for, children's development in lower-risk settings. Studies have found that high levels of parental control are associated with greater depressive symptoms and social functioning issues among adolescents from lower-risk backgrounds (Alloy et al., 2006; McElhaney & Allen, 2001). Additionally, aspects of parental monitoring (specifically, parents' unwarranted solicitation of children's whereabouts and activities), have been shown to be unrelated to children's adjustment outcomes (e.g., Stattin & Kerr, 2000; Kerr & Stattin, 2000), and further, negative reactions to parental monitoring have been found to be associated with greater depressive symptomatology (Laird et al., 2018). These studies suggest that the potential for parental vigilance to benefit children's development is nuanced and depends on contextual factors.

Collectively, researchers have explored psychosocial outcomes related to parental vigilance as a function of context for individuals in adolescence and young adulthood, but no

research has explored long-term physical and mental health implications. Therefore, the work presented in this dissertation seeks to address the gaps in the literature in a series of three studies: Studies 1 and 3 explore whether indicators of heightened parental vigilance (e.g., parental control, monitoring, and/or limit-setting) moderates the association between childhood risk and health outcomes in adulthood, whereas Study 2 examines whether high levels of parental knowledge in one generation promotes positive developmental outcomes among a future generation of high-risk youth (and further, whether families' socioeconomic context moderates this association).

To elaborate, Study 1 explores whether adults' retrospective reports of parental control modified the association between recalled neighborhood risk in childhood and susceptibility to the common cold using a viral challenge study design. Study 2 uses data from a study with African American families spanning three generations of individuals to determine whether parents' monitoring behaviors are intergenerationally transmitted to influence the third generation's psychosocial adjustment outcomes, and if these associations vary as a function of socioeconomic risk. Finally, Study 3 examines the prospective associations among childhood environmental risk and physical and emotional health in adulthood, and whether participants' reports of parental vigilance play a role in these associations.

In general, we predicted that high levels of parental vigilance would serve as a protective factor and offset projected negative long-term health consequences associated with childhood risk and be associated with poorer health (or unrelated to health) among individuals who experienced lower levels of risk in childhood (Studies 1 and 3). We also predicted that greater parental vigilance would be associated with better psychosocial adjustment outcomes (fewer depressive symptoms and lower anxiety, risk-taking behaviors, and poor self-control) among

youth in risky contexts, and that vigilance would be particularly protective among youth whose families experienced high levels of socioeconomic risk (Study 2).

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CHAPTER 2

RECALLED NEIGHBORHOOD ENVIRONMENTS, PARENTAL CONTROL, AND CYTOKINE-MEDIATED RESPONSE TO VIRAL CHALLENGE [1]

[1] Corallo, K. L., Lyle, S. M., Murphy, M. L. M., vanDellen, M., Ehrlich, K. B. 2021. *Psychosomatic Medicine*. 83:843-851. Reprinted here with permission of the publisher.

Abstract

Objective: Neighborhood risk in childhood is associated with poor health across the lifespan. However, many people who are reared in risky neighborhoods remain healthy in adulthood. In the context of high-risk neighborhoods, controlling parenting practices might promote better physical health outcomes later in life. The current study utilized a viral challenge paradigm to examine whether parental control throughout childhood moderated the association between recalled neighborhood risk and cytokine-mediated cold susceptibility.

Methods: A sample of 209 healthy adults completed questionnaires to assess recalled neighborhood risk and parental control over the first 15 years of life, were exposed to a common cold virus, and were quarantined for six days. Researchers assessed nasal proinflammatory cytokine production and objective markers of illness. Participants were diagnosed with a clinical cold if they met infection and objective illness criteria.

Results: A significant Neighborhood Risk × Parental Control interaction emerged to predict proinflammatory cytokine production. Further, parental control moderated the cytokine-mediated association between neighborhood risk and cold diagnosis (index = -.073, 95% CI [-.170, -.016]), likelihood of infection (index = -.071, 95% CI [-.172, -.015]), and meeting objective symptom criteria (index = -.074, 95% CI [-.195, -.005]). Specifically, there was a negative association between neighborhood risk and objective cold diagnosis and infection status at higher levels of parental control, but a nonsignificant association at lower levels of parental control.

Conclusion: Findings suggest that the degree to which recalled neighborhood risk is related to adult health varies as a function of parental control throughout childhood.

Keywords: cold susceptibility, cytokines, infection, rhinovirus, parental control, neighborhood risk

Introduction

Risky neighborhoods in childhood, broadly characterized by poor-quality physical and social characteristics (e.g., uncleanliness, pollution, high traffic volume, noisiness, limited access to public services and amenities, and compromised safety due to high rates of crime and violence) have been linked to poor lifespan physical health outcomes (1-4). For example, census tract measures of neighborhood-level socioeconomic disadvantage are associated with larger body mass indexes (BMI) and lower basal cortisol levels among adolescents (5) and poor neighborhood conditions predict harmful inflammatory outcomes among children with asthma (6,7). Further, recalled neighborhood physical and safety conditions throughout childhood are associated with increased latent virus reactivation in adulthood (2). Characteristics associated with neighborhood poverty, such as pollution and housing disrepair, are associated with allostatic load (8-10) and exposure to neighborhood crime and violence in childhood predicts engagement in poor health behaviors (1). Collectively, this evidence suggests neighborhood physical and social characteristics, assessed via self-reports and community data, are associated with health outcomes.

Early neighborhood risk may influence physical health through disruptions to the neuroendocrine, immune, and stress-response systems (1,8-11). In addition, individuals reared in unsafe neighborhoods often engage in risky health behaviors (e.g., drug use; (1)). In turn, alterations to biological systems and engagement in poor health behaviors may give rise to heightened susceptibility to acute and chronic illnesses (12-14). However, despite robust associations among indicators of neighborhood risk and health, many individuals who grow up in risky neighborhoods remain healthy. Protective factors, such as high-quality parenting, have been shown to mitigate negative consequences of adverse childhood environments (15-17).

While early research focused on the benefits of supportive and autonomy-promoting parenting (18-20), recent work suggests that social contexts shape the extent to which parenting behaviors are beneficial for child development (21-23). For example, in risky neighborhoods, greater parental control (i.e., attempts to govern children's whereabouts and activities) and undermining of youths' autonomy (i.e., restricting children from making decisions) may reflect parents' intentions to shield children from risky neighborhood and social conditions (24,25). For youth in high-risk contexts, parental control may be positively associated with social functioning and academic achievement (22,26). However, in low-risk settings, greater parental control and undermining of children's autonomy is negatively associated with children's development (22,27), suggesting the influence of control-oriented parenting behaviors is context-dependent.

Importantly, in addition to proximal influences, parental control has long-term implications for individuals' psychosocial outcomes. For example, among adolescents with greater delinquent peer affiliation, parental control protects against future engagement in problematic behaviors (28,29). Additionally, parental monitoring (i.e., supervision and knowledge of children's day-to-day experiences)—a parenting construct widely recognized as separate but related to parental control (30)—has lasting ties to positive adjustment for individuals reared in risky environments (31-33). Conversely, prospective reports of high parental control (particularly in the absence of parental warmth and care) is associated with ongoing depressive symptomatology among low-risk samples (27,34). Taken together, these findings suggest that in risky environments, parents' efforts to prevent youth from engaging in delinquent behaviors have persistent benefits, whereas parental control in low-risk environments may engender emotional distress. However, less is known about long-term *physical* health implications associated with parental control in different childhood contexts.

Compelling evidence points to inflammatory processes as key mediators that link early life experiences to many future health problems (13). As such, study designs that leverage quasi-experimental immunologic challenges, such as viral challenge paradigms, can be informative for understanding potential mechanisms through which childhood factors may influence adult health. In a common cold viral challenge paradigm, healthy adults report on various psychosocial factors and are then exposed to a relatively benign cold virus. Volunteers are subsequently quarantined for five to six days to determine who becomes infected with the virus, and who among those infected develops a clinical illness (35). Infection occurs when the virus replicates within the host, prompting both an innate and an adaptive immune response to eradicate the virus. Only a fraction of individuals who become infected with a cold virus develop clinical signs of a cold (e.g., increased mucus, nasal congestion). These signs of illness have been shown to be driven primarily by an over-exaggerated local inflammatory response to viral infection, rather than by the cold virus itself (36,37).

In the case of a common cold, an over-exaggerated local inflammatory response to infection results in unpleasant symptoms but is otherwise generally benign. However, tendencies toward excessive inflammation are known to contribute to the pathogenesis and progression of many chronic illnesses (38). Thus, viral challenge experiments provide a low-risk, highly controlled framework for determining how psychosocial stressors relate to inflammatory-linked processes and outcomes in a disease context. Psychosocial factors may modulate clinical illness risk by increasing the likelihood of becoming infected with the virus (suggesting impaired host immunity) or by promoting severe signs of illness in response to viral infection (suggesting an overly exaggerated host proinflammatory response to infection), or both. When examined

together, these outcomes can provide important insights into the mechanisms through which psychosocial variables relate to risk for more serious illnesses (39,40).

The current study utilized open-access viral challenge data from the Common Cold Project (www.commoncoldproject.com) to examine whether perceived parental control in childhood and adolescence moderated the association between recalled neighborhood risk throughout childhood and inflammation-mediated response to viral exposure in adulthood (Figure 2.1; (41)). We hypothesized that the extent to which recalled neighborhood risk was related to inflammation-mediated cold susceptibility would be dependent on participants' receipt of parental control. We predicted that individuals who grew up in a risky neighborhood would be less likely to meet criteria for viral infection and clinical illness if they reported greater levels of parental control.

Method

Participants

The data for this study (Pittsburgh Cold Study 3; PCS3) were collected by the Laboratory for the Study of Stress, Immunity, and Disease at Carnegie Mellon University under the directorship of Sheldon Cohen, PhD, and were accessed via the Common Cold Project website (www.commoncoldproject.com; grant number NCCIH AT006694) (41). Participants included 213 adults ages 18 to 55 recruited between 2007 and 2011 from the metropolitan Pittsburgh, Pennsylvania area through newspaper advertisements. To participate, individuals were required to be in good health (as determined by medical history and physician examination). For full, detailed information on inclusion and exclusion criteria, please see https://www.cmu.edu/common-cold-project/human-subjects/index.html. Participants received $1000 in compensation for completing the study. The Institutional Review Boards at Carnegie

Mellon University and the University of Pittsburgh approved the study, and all participants provided informed consent. For examples of previously published analyses using PCS3 data, please see references 2,42-45.

Procedure

Participants completed pre-study questionnaires to assess demographic information, psychosocial constructs, and health behaviors. Two to three days prior to the study, participants provided blood samples to assess pre-challenge antibody titers to rhinovirus 39 (RV39). Participants were subsequently quarantined in a hotel room for six days. On the first day, they were exposed to standardized dose of RV39 via nasal drops and were monitored for the following five days to assess viral infection and objective markers of illness. Participants attended a follow-up visit approximately 28 days after initial viral exposure to evaluate post-challenge antibody titers used for determining RV39 infection status.

Measures

Recalled Neighborhood Risk

The Places You've Lived Interview (PLI; (2)) is a 13-item questionnaire designed to assess recalled safety, physical, and social characteristics of the neighborhood environment based separately on when participants were 5, 10, and 15 years of age. The 3-item Neighborhood Safety scale asked participants to reflect on whether their childhood neighborhood was safe; items were "Was your street considered safe?", "How often did you observe violent acts on your street?", and "How often did you see people using drugs or drinking alcohol on your street?" (α = 0.64-0.67; M = 1.53, SD = 0.58). The four-item Social Environment scale evaluated the quality of interactions with neighbors, friends, and adults; example items include "Did you have friends in the neighborhood?" and "Were there any adult neighbors who might watch out for you?" (α =

0.71-0.76; M = 1.82, SD = 0.59). The 6-item Physical Environment scale included items about litter, noisiness, and other conditions of the neighborhood; example items include "Was the condition of the street very poor?" and "Was the street very noisy?" (α = 0.76-0.82; M = .24, SD = 0.22). Response options for the neighborhood safety and social environment scales ranged from *all the time (1)* to *never (4)*. For the physical environment items, response options were *yes (1)*/*no (0)*. Participants' average responses across all three ages were z-scored for each scale. The z-scores for the three scales across all ages were significantly interrelated (r range: .24 – .78, p's < .001; α = 0.70) and averaged to form a composite for recalled neighborhood risk. Higher scores indicated riskier childhood neighborhood environments.

Parental Control

The Overprotection Dimension of the Parental Bonding Instrument (PBI; (46)) assessed recalled parental control throughout the first 15 years of life. This subscale included 6 items with response options ranging from *very like (1)* to *very unlike (4).* Participants were prompted with the following statement: "During the first 15 years of my life, my parents…" for the following 6 items related to parental control: "were overprotective of me," "tried to control everything I did," "tended to baby me," "let me do things I liked doing," "liked me to make my own decisions," and "let me decide things on my own." The latter three items were reverse scored, and participants' responses were averaged so that higher scores indicated greater perceived parental control (α = 0.68; M = 2.13, SD = 0.56).

Objective Disease Outcomes

Infection Status**.** Infection was present if there was recovery of the challenge virus in nasal secretions on any of the post-exposure quarantine days, or at least a 4-fold increase in antibody titers from the pre-challenge to 28-day post-challenge assessments.

Objective Symptom Criteria**.** Clinical cold diagnoses were determined using previously validated criteria (35) utilizing objective measures of the following two symptoms throughout the five post-exposure days of quarantine: 1) mucus production and 2) nasal mucociliary clearance function. Total baseline-adjusted mucus weight (in grams) was measured by asking participants to use pre-weighed tissues (in grams) to collect their mucus secretions, which they sealed in pre-weighed bags (in grams). The total mucus weight was determined by subtracting the bag and tissue weight from the total weight (M = 10.6, SD = 18.2; (47)). Average nasal mucociliary clearance function was determined by the amount of time (in minutes) it took for 20 µL of a sweetened dye solution placed in participants' nasal turbinate to produce a taste in their mouth (M = 3.82; SD = 3.88; (48)) across each post-exposure day of quarantine.

Objective Cold Diagnosis. A clinical cold diagnosis was made if a participant (a) showed evidence of viral infection, and (b) showed sufficiently severe illness signs defined as either (i) a total baseline-adjusted mucus weight of at least 10 g or (ii) an average baseline-adjusted nasal mucociliary clearance time of at least 7 minutes (35).

Inflammatory Markers

Nasal Proinflammatory Cytokine Production. Due to previous evidence that the development of clinical illness following infection with a cold-causing virus is a cytokine-mediated process (36,40,44), we created a composite of the nasal proinflammatory cytokines interleukin-(IL-)6, IL-1β, and tumor necrosis factor alpha (TNF-α). Participants' daily quarantine nasal wash fluid was assayed for IL-6 (M = 58.3, SD = 80.6), IL-1β (M = 31.6, SD = 44.0), and TNF-α (M = 6.00, SD = 9.72) in pg/mL via commercially available enzyme-linked immunosorbent assays (Endogen) performed using the manufacturer's instructions. Nasal cytokine response to viral exposure was determined by log-transforming the area under the curve

(AUC) for the 5 post-exposure days in quarantine, adjusted for the baseline cytokines. Nasal secretion levels of IL-6, IL-1β, TNF-α were highly interrelated (*r* range: .55-.66, *p*s < .001), and therefore a composite measure was created by summing standardized IL-6, IL-1β, and TNF-α scores (see references 44,49).

Standard Control Variables

Based on standard analytic procedures utilizing Common Cold Project viral challenge data published over the past three decades (e.g., 35,40), seven "standard covariates" were included in all analyses: age, sex (male = 0; female = 1), race (White = 0; non-White = 1), socioeconomic status (represented by years of education), BMI, season (spring was the reference category), and pre-challenge antibody titers to RV39 (coded as < 4 = 0 and ≥ 4 = 1).[2]

Psychological Control Variables

Previous evidence suggests that contemporaneous perceived stress, negative and positive affectivity, and personality characteristics (e.g., neuroticism) can bias recalled experiences (50). We ran follow-up analyses including these factors as covariates. Perceived stress was assessed via the 10-item Perceived Stress Scale (PSS), on which participants rated how often they felt or thought a certain way in relation to stressful/upsetting events over the past month (*never [0] to very often [4]*). Responses were summed and higher scores indicated greater perceived stress (*M* = 12.0, *SD* = 5.67). Trait positive and negative affect were assessed via the Positive and Negative Affect Schedule (PANAS) – Expanded Form (51). Participants rated how they generally felt on a scale from *very slightly or not at all (1*) to *extremely (5)*. The sum of 10 items assessed positive

[2]Prospective volunteers were screened for RV39 10-12 weeks prior to viral exposure and were ineligible to participate if they had a positive test at that point. However, if they subsequently tested positive when assessed several days prior to the administration of the challenge virus, they were not excluded. Instead, antibody titers were entered as a covariate as described above. For more details, please visit https://www.cmu.edu/common-cold-project/pittsburgh-cold-study-3/trial-outline.html.

affect (M = 34.9, SD = 7.08), and the sum of an additional 10 items assessed negative affect (M = 16.4, SD = 5.61). Neuroticism was assessed via the 10-item 'emotional stability' subscale of the International Personality Item Pool (IPIP) Big-Five Factor Markers (52); items were scored on a scale ranging from *very inaccurate (1)* to *very accurate (5)*. Responses were summed so that higher scores indicated greater neuroticism (M = 25.2, SD = 7.57). Because trait negative affect and neuroticism were highly correlated (r = .71, p < .001), they were standardized and summed to create a composite measure of negative affectivity.

Data Analytic Plan

Data were analyzed using IBM SPSS Statistics Version 25 and PROCESS Version 2.16 (53). First, a moderation model assessed the Recalled Neighborhood Risk × Parental Control interaction to predict nasal inflammatory cytokine production (i.e., the first leg of the Figure 2.1 model). Next, percentile bootstrapping techniques based on 50,000 resamples (seed value = 3276459) were used to test moderated mediation models. These models assessed the indirect effect of recalled neighborhood risk on cold susceptibility through cytokine production as a function of perceived parental control (see Figure 1).

The index of moderated mediation is used to determine whether an indirect effect (i.e., the indirect effect of neighborhood risk on cold susceptibility through cytokine production) depends on the level of the moderator (i.e., parental control). If the confidence interval associated with the index of moderated mediation does not include 0, there is evidence that the mediation is moderated (53). For models with a significant index of moderated mediation, conditional indirect effects for the moderator were probed at the mean and ±1 *SD*.

Results

Of the original 213 PCS3 participants, complete data were available for 209 individuals. Of these individuals, 156 (74.6%) met criteria for infection, and 63 (30.1%) met criteria for a clinical illness. Table 2.1 presents sample characteristics and demographic information for the entire sample as well as just those who were infected and just those who met illness criteria. These rates are nearly identical to rates reported in previous publications from different subsets of these data; see references 2,42-45.

Intercorrelations and descriptive statistics for the principal variables are presented in Table 2.2. Neighborhood risk and parental control were not correlated with proinflammatory cytokine production or any of the objective illness outcomes (*r range:* -.01 to -.10, *p*s > .10). However, cytokine production was significantly correlated with all illness outcomes.

Neighborhood Risk, Parental Control, and Nasal Proinflammatory Cytokine Production

Regression analyses (Table 2.3) revealed no conditional effects of neighborhood risk or parental control on cytokine production (see Table 2.3, Model 2). However, the Neighborhood Risk × Parental Control interaction predicted nasal proinflammatory cytokine production (see Table 2.3, Model 3). At values ≥ 2.48 on the parental control scale (represented by 29.7% of the sample, or 62 individuals), there was a significant negative association between recalled neighborhood risk and cytokine production. Figure 2 illustrates the simple slopes for mean +/- 1 *SD* values of parental control.[3]

Post-hoc exploration of the Johnson-Neyman regions of significance when the moderator is flipped from parental control to neighborhood risk indicated that the interaction was also significant at values below -1.07 and above 0.87 on the neighborhood risk composite, meaning

[3]Please see supplemental materials for a scatterplot that displays the association between neighborhood risk and nasal pro-inflammatory cytokine production at high and low levels of parental control (Figure 3).

that at very low (2.87% of the sample) and high (14.4% of the sample) levels of neighborhood risk, nasal proinflammatory cytokine production varied as a function of parental control. The interaction plot (Figure 2.2) indicates that at the lower end of neighborhood risk, individuals who reported greater (vs. lower) levels of parental control were more likely to mount a proinflammatory response to viral exposure. Conversely, at higher levels of neighborhood risk, individuals who reported greater (vs. lower) levels of parental control were less likely to mount a proinflammatory response.

Given evidence for a significant Neighborhood Risk × Parental Control interaction in the prediction of nasal proinflammatory cytokine production and evidence for associations between cytokine production and illness outcomes, we proceeded to conduct moderated mediation models to test the Recalled Neighborhood Risk × Parental Control interaction as a predictor of cytokine-mediated objective cold diagnosis, infection status, and illness criteria.

Neighborhood Risk × Parental Control to Predict Cytokine-Mediated Cold Outcomes

Clinical Cold Diagnosis

Analyses revealed a significant index of moderated mediation in the prediction of clinical cold diagnosis (Table 2.4). In other words, parental control moderated the indirect association between neighborhood risk and clinical cold diagnosis through proinflammatory cytokine production. At -1 *SD* and the mean of parental control, neighborhood risk was not associated with cytokine-mediated cold diagnoses. However, at +1 *SD* of parental control, neighborhood risk was negatively associated with cytokine-mediated cold diagnoses. Thus, individuals who reported greater parental control were more likely to be diagnosed with a clinical illness if they were from lower-risk neighborhoods than if they were from higher-risk neighborhoods.

Infection Status. There was a significant index of moderated mediation in predicting the likelihood of viral infection (Table 2.4). Again, the conditional indirect effects for individuals who were at -1 *SD* and at the mean of parental control were non-significant. However, the conditional indirect effect for individuals who were +1 *SD* in parental control was significant. Like the above findings for clinical cold diagnoses, there was a negative association between recalled neighborhood risk and cytokine-mediated infection status at greater levels of parental control.

Objective Illness Criteria. There was a significant index of moderated mediation in predicting the likelihood of meeting objective illness criteria among individuals who met infection criteria (n = 156; Table 2.4). However, the conditional indirect effects of neighborhood risk on objective illness criteria through cytokine production did not meet statistical significance at ±1 *SD* of parental control.

Alternative Explanations

To rule out the possibility that contemporaneous positive and negative affectivity and perceived stress influenced the results explained above, we re-analyzed our models including perceived stress and positive and negative affectivity as covariates. After including these covariates, the Neighborhood Risk × Parental Control interaction continued to predict nasal proinflammatory cytokine production (b = -.17, SE = .07, p = .013). Likewise, the moderated mediation findings were unchanged (clinical cold diagnosis: index of moderated mediation = -.082, 95% CI [-.197, -.017]), (infection status: index of moderated mediation = -.074, 95% CI [-.185, -.016]), (objective illness criteria: index of moderated mediation = -.081, 95% CI [-.222, -.010]).

Discussion

In the present study, we examined the extent to which perceived parental control shaped the association between recalled neighborhood risk and cytokine-mediated cold susceptibility. Although neighborhood risk and parental control were not directly associated with cytokine production or clinical illness outcomes, a significant Neighborhood Risk × Parental Control interaction emerged to predict nasal proinflammatory cytokine production and cytokine-mediated cold outcomes. Specifically, at greater (but not lower) levels of parental control, individuals who grew up in a riskier neighborhood had lower cytokine production and a lower likelihood of cytokine-mediated cold susceptibility relative to individuals who grew up in a lower-risk neighborhood. Stated differently, in the context of greater parental control, individuals who grew up in riskier neighborhoods might have experienced a potential immune advantage relative to individuals who grew up in low-risk neighborhoods.

Post hoc examination of these analyses revealed that among individuals who grew up in high-risk neighborhoods (approximately 14% of the sample), those who reported greater parental control throughout childhood were less likely to mount a proinflammatory response to viral exposure relative to those who reported lower parental control. In addition, among individuals who grew up in low-risk neighborhoods, greater parental control throughout childhood was associated with a *greater* likelihood of mounting a proinflammatory response to viral exposure relative to individuals who reported lower parental control (however, this was only significant among approximately 3% of the sample). These findings are consistent with the growing awareness that many parenting behaviors (such as parental control) cannot be viewed in the absence of the broader social context in which children are living (22,26,28-31). As such, it is

worthwhile to consider the potential processes by which parental control might play a role in previously identified links between neighborhood settings and physical health (1-4).

In risky neighborhoods, parental control may represent greater vigilance on the part of parents to prevent dangerous exposures and behaviors among their children (24,25). In this context, parental control may shelter children from stressful and unsafe situations. Further, when parents practice greater levels of control in high-risk environments, youth might be less likely to engage in delinquent behaviors associated with neighborhood risk, such as smoking and drinking (1), that serve as gateways to substance use in adulthood. Thus, greater parental vigilance and control in high-risk neighborhoods may curb hazardous exposures and behaviors in childhood and adolescence, which in turn could shield individuals from vulnerabilities in defending against illnesses in adulthood.

In contrast, in low-risk settings, parental control may not serve to protect youths from danger because they are unlikely to encounter real threats in their environment. As a result, in these low-risk settings, greater control may be perceived as needlessly inhibiting children's independence and autonomy (21-23). Not surprisingly, parental control in low-risk contexts has been linked to immediate and long-term psychological distress (27,34), both of which have implications for physical well-being. Extensive research has demonstrated that poor mental health and psychological distress serve as precursors for poorer self-rated health (54) and immune functioning (55) in adulthood, as well as a greater likelihood of engaging in poor health behaviors (55) and developing chronic physical disease (56).

This study expands knowledge of the ways in which healthy adults' perceptions of childhood contexts and recalled parenting behaviors relate to illness risk in a disease model. More importantly, these findings pose broader conceptual implications for adult physical health.

Although neighborhood risk and compromised immune function have been linked (10,12), the current study's findings suggest that parental control may influence inflammatory processes associated with dangerous childhood environments. As such, parental control may also protect adults who were raised in high-risk neighborhoods from other diseases modulated by excessive inflammation, such as cardiovascular disease and diabetes (39,40).

In support of the hypothesis that parental control protects against inflammation-mediated health problems for individuals from risky backgrounds, previous research suggests that high-quality parenting is associated with better trajectories of inflammatory-mediated health outcomes for young adults from high-risk backgrounds (57,58). Furthermore, recalled nurturant parenting mitigates the association between low-SES environments and proinflammatory processes for young and middle-aged adults (59,60). Future research should explore whether there are unique benefits of parental control that extend beyond previously identified protective parenting characteristics. We note, however, there are likely limits to the extent parenting behaviors can mitigate the progression of poor health (e.g., parental control likely does not affect health outcomes that have a strong genetic basis).

Several study limitations will be important to address in future research. Childhood experiences were measured retrospectively, and biases in reports of recalled parental control and neighborhood risk are possible (e.g., reference 61). Although prior research has shown that individual differences in personality and current mood and life circumstances can lead to biased reconstructions of memories (61-64), including these factors as covariates did not attenuate our findings. Additionally, the measure of parental control asked participants to recall their parents' behaviors over the first 15 years of life; as such, we were unable to address questions about whether parents' controlling behaviors during particular periods in development (e.g., early

childhood vs. adolescence) had an outsized influence on cold outcomes. One intriguing opportunity to address these limitations would be to leverage ongoing longitudinal studies for use in a new viral challenge investigation, which would allow for an examination of how prospective and retrospective assessments of childhood experiences and their precise developmental timing are related to exaggerated inflammatory processes in the context of a disease model.

Another potential limitation of the current study is the wide range of participant ages, which causes variability in the number of years participants had to reflect back on. This variability may have led to differences in memory accuracy for older versus younger participants. We note, however, that age was not associated with reports of parental control or neighborhood qualities (*r* range: .04-.10, *p* range: .15-.57). Therefore, our findings are not likely to be the result of generational differences in recall.

In addition, the analyses in the present study were conducted using an open-access dataset that has been used to address other research questions about psychosocial predictors of cold susceptibility. Long-term confidence in the reported results will require replicating the current pattern of findings in additional studies. Lastly, one final limitation to note is that although our analytic sample was well-powered to detect medium-sized conditional indirect effects, it was only moderately powered to detect small-sized conditional indirect effects (65). As such, the null findings are potentially at risk of being Type II errors.

In summary, findings from the current study suggest the association between recalled neighborhood risk and cytokine-mediated cold susceptibility varies as a function of perceived parental control throughout childhood. As such, controlling parenting behaviors may potentially reduce subsequent health risk sequelae associated with high-risk neighborhoods in childhood and

amplify health risk for individuals from low-risk settings. Future research should evaluate whether parental control protects against other cytokine-mediated illnesses for adults who were raised in high-risk contexts and begin to consider the mechanisms by which controlling parenting behaviors affect long-term physical health for individuals from low-risk contexts.

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Tables

Table 2.1
Sample characteristics

	Total Sample (n = 209)	Met Criteria for Infection (n = 156)	Met Criteria for Objective Cold Diagnosis (n = 63)
Age	30.2 (10.9)	30.3 (11.2)	32.8 (12.1)
Sex			
Male	120 (57.4)	92 (59.0)	31 (49.2)
Female	89 (42.6)	64 (41.0)	32 (50.8)
Race/Ethnicity			
White	139 (66.5)	107 (68.6)	39 (61.9)
Non-White	70 (33.5)	49 (31.4)	24 (38.1)
Years of Education	14.1 (1.9)	14.1 (2.0)	14.1 (2.1)
Season of Trial			
Spring	67 (32.1)	51 (32.7)	11 (17.5)
Summer	85 (40.7)	68 (43.6)	31 (50.8)
Winter	57 (27.3)	37 (23.7)	20 (31.7)

Note. Values are means (SD) or numbers (%).

Table 2.2
Descriptive statistics and Pearson correlations among principal study variables

Variables	1	2	3	4	5	6
1. Neighborhood risk	--					
2. Parental control	.159*	--				
3. Proinflammatory cytokines	-.099	-.053	--			
4. Objective cold diagnosis	-.026	-.036	.442***	--		
5. Infection status	-.044	-.080	.257**	.383**	--	
6. Illness signs	-.011	-.006	.399**	--	--	--
Mean (*SD*)	0.01 (0.79)	2.13 (0.56)	0.02 (2.58)	--	--	--
Range	-1.1 - 2.5	1 - 3.5	-2.1 - 11.5	0 - 1	0 - 1	0 - 1

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

Table 2.3.
Regression analyses predicting nasal proinflammatory cytokine production

Predictor	Model 1 *b* (*SE*)	Model 2 *b* (*SE*)	Model 3 *b* (*SE*)
Pre-Challenge RV39 Antibody Titers	-.546 (.436)	-.525 (.439)	-.501 (.433)
Age	-.020 (.017)	-.022 (.017)	-.020 (.017)
Sex	.277 (.358)	.262 (.360)	.151 (.358)
Race	.803 (.400)*	.725 (.416)	.690 (.410)
Education	.095 (.097)	.077 (.100)	.070 (.099)
BMI	.037 (.029)	.038 (.029)	.030 (.029)
Winter	.760 (.453)	.783 (.456)	.915 (.452)*
Summer	1.452 (.416)***	1.457 (.418)***	1.478 (.412)***
Neighborhood Risk		-.163 (.243)	1.934 (.848)*
Parental Control		-.010 (.054)	-.002 (.053)
Neighborhood Risk × Parental Control			-.168 (.065)*

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

Table 2.4.
Moderated mediation (Hayes model 7) regression results predicting cytokine-mediated cold outcomes

		Conditional Indirect Effects		
Outcome	Index of Moderated Mediation (95% CI)	-1 *SD* Parental Control (95% CI)	Mean Parental Control (95% CI)	+1 *SD* Parental Control (95% CI)
Clinical Illness (n = 209)	**-.073 (-.170, -.016)**	.155 (-.104, .534)	-.089 (-.313, .114)	**-.334 (-.760, -.065)**
Infection Status (n = 209)	**-.071 (-.172, -.015)**	.150 (-.105, .535)	-.086 (-.318, .112)	**-.323 (-.774, -.063)**
Illness Signs (among infected individuals; n = 156)	**-.074 (-.195, -.005)**	.185 (-.117, .677)	-.070 (-.337, .196)	-.325 (-.865, .019)

Note. Bolded coefficients represent statistically significant indices and simple effects.

Figures

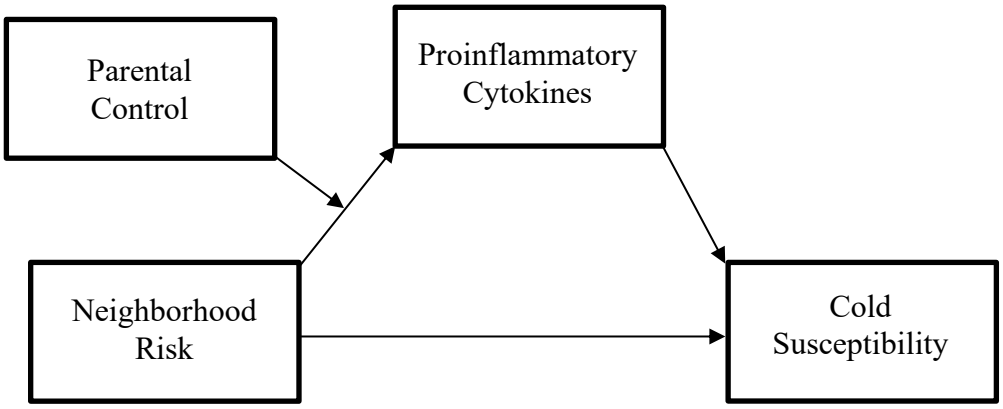

Figure 2.1. Conceptual model.

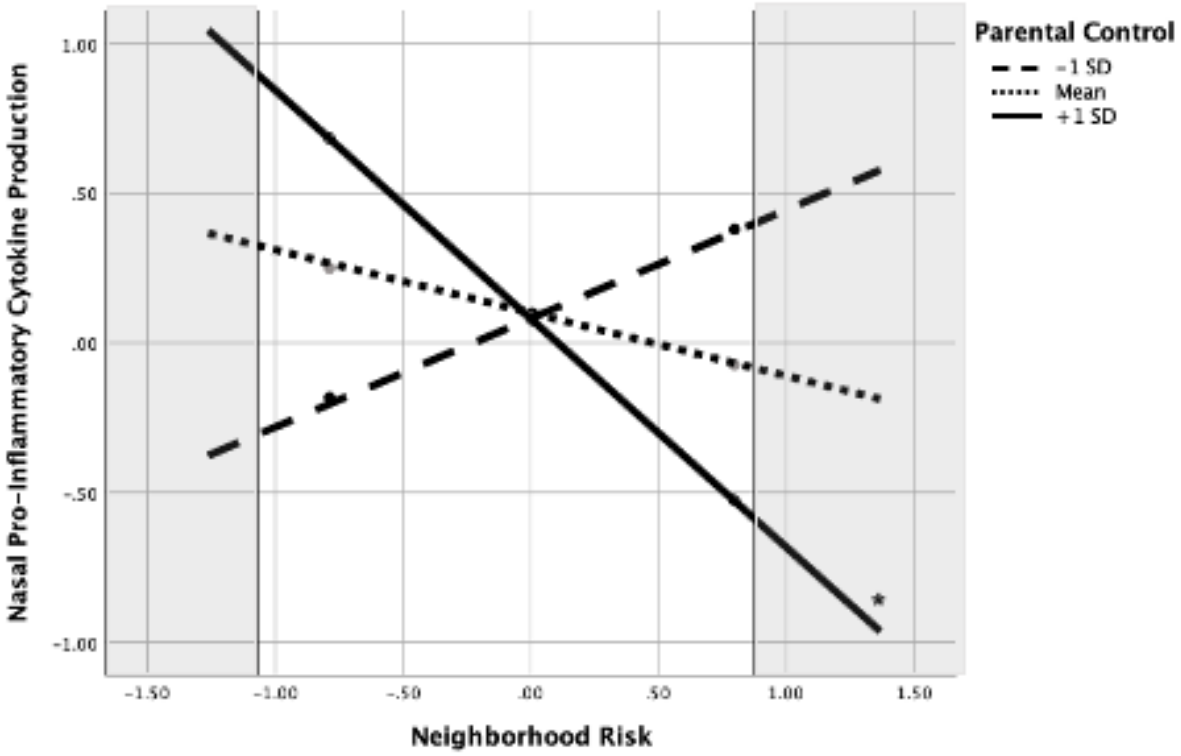

Figure 2. Neighborhood Risk × Parental Control interaction predicting nasal proinflammatory cytokine production. Simple slopes at the mean (b = -.204, p = .40, 95% CI [-.677, .972690]), + 1 *SD* (b = -.763, p = .023, 95% CI [-1.42, -.105]), and -1 *SD* (b = .354, p = .26, 95% CI [-.261, .970]) values of parental control are presented in the figure. The shaded regions represent the Johnson-Neyman regions of significance when the moderator is flipped (i.e., along the continuum of neighborhood risk). * p < .05.

CHAPTER 3

INTERGENERATIONAL TRANSMISSION OF PARENTAL KNOWLEDGE ON CHILD PSYCHOSOCIAL ADJUSTMENT AMONG RURAL AFRICAN AMERICAN FAMILIES [4]

[4] Corallo, K. L., Lyle, S. M., Yu, T., Brody, G. H., Ehrlich, K. B. Submitted to *Journal of Child and Family Studies*, 04/07/2022

Abstract

Parental knowledge, or the extent to which parents are knowledgeable of their children's whereabouts and activities, plays a key role in attenuating delinquent behaviors and activities in adolescence. Parental knowledge may be particularly effective in low-resourced environments, where dangerous exposures are commonplace. Moreover, parenting behaviors (such as parental knowledge) in one generation may influence subsequent generations' psychosocial adjustment. In this pilot study with longitudinal data spanning three generations, we explored whether the first generation's (G1; n = 48) parental knowledge throughout the second generation's (G2, n = 48) adolescence was associated with the third generation's (G3; n = 60) psychosocial adjustment outcomes. Further, we explored whether caregiver-reported family socioeconomic hardship throughout the G2s' adolescence influenced the extent to which G1 parental knowledge affected G3s' psychosocial adjustment. G1 parental knowledge predicted lower G3 depressive symptoms, anxiety, risky behaviors, and poor self-control. We did not find evidence that G2 involved-vigilant parenting or substance use mediated associations among G1 parental knowledge and G3 adjustment outcomes. However, moderation analyses revealed a negative association between G1 parental knowledge and anxiety among G3 children whose parents experienced greater socioeconomic hardship in adolescence, but no association among G3 children whose parents experienced less socioeconomic hardship. Results support the notion that parental knowledge may be "intergenerationally transmitted" to influence successive generations' adjustment and highlight the importance of considering both the socioeconomic and intergenerational context when exploring links between parenting and children's development.

Keywords: parental knowledge, adolescence, intergenerational transmission, three generations, psychosocial adjustment

Introduction

Parental monitoring, or the tendency to track and supervise children's whereabouts and activities, is associated with fewer delinquent behaviors in adolescence (e.g., underage substance use, social aggression, lying and cheating, etc.; Dishion & McMahon, 1998; Fletcher, Steinberg, & Williams-Wheeler, 2004). Most researchers agree that parental knowledge of adolescents' lives plays a key role in the relation between monitoring and problematic behaviors (Bendezú et al., 2018; Fletcher et al., 2004; Laird et al., 2003; Racz & McMahon, 2011; Soenens et al., 2006). For example, findings from several studies show that parental monitoring is positively associated with knowledge of children's peer affiliations and activity engagements; in turn, this knowledge predicts lower levels of delinquency (Bendezú et al., 2018; Fletcher et al., 2004; Laird et al., 2003). As such, parental monitoring and knowledge attenuate the risk for engaging in delinquent behaviors that have unintended and often negative long-term consequences (e.g., encounters with law enforcement, school expulsions, unplanned pregnancies).

Parental Monitoring and Environmental Context

Although early developmental research championed parental monitoring as a predictor of children's adjustment, subsequent work has challenged the global benefits of monitoring (Laird, Marrero, & Sentse, 2010; Rekker et al., 2017; Stattin & Kerr, 2000). Specifically, as researchers began parsing out unique dimensions of monitoring (e.g., disclosure, solicitation, and control; Stattin & Kerr, 2000) and contexts under which parental monitoring is most effective for preventing problem behaviors (e.g., environmental conditions; Bacchini, Miranda, & Affuso, 2011; Laird et al., 2010; Rodriguez-Meirinhos et al., 2020; Soenens, Vansteenkiste, & Petegem, 2015), it has become clear that the association between monitoring and children's outcomes is

nuanced. These more recent investigations emphasize the importance of studying the influence of context in the relation between parental monitoring and adolescent adjustment.

One contextual factor that modifies the association between parental monitoring and child adjustment outcomes is socioeconomic risk. Not surprisingly, parental monitoring is particularly effective in high-risk settings, such as in neighborhoods where access and exposure to precarious activities and substance use are high (e.g., Bacchini et al., 2011; Bendezú et al., 2018; Dishion & McMahon, 1998). Further, some evidence suggests that parents' monitoring behaviors are sometimes unrelated to child adjustment (Kerr & Stattin, 2000; Stattin & Kerr, 2000), or even associated with *poorer* child adjustment in contexts where dangerous exposures are low (e.g., Rekker et al., 2017). Recent work highlights the social and environmental conditions under which monitoring of children might be more or less effective (Soenens et al., 2015), and suggests that social contexts (e.g., neighborhood risk and socioeconomic status) should be considered when determining developmental outcomes associated with parental monitoring and knowledge.

Parental Monitoring and Intergenerational Context

In addition to highlighting the importance of environmental context, a growing body of literature reveals compelling evidence that parenting characteristics, including parental monitoring, may be intergenerationally transmitted (Bailey et al., 2009; Capaldi et al., 2003; Neppl, Diggs, & Cleveland, 2020). This research requires the use of longitudinal, multigenerational assessments of families captured across decades to explore how the transmission of parenting behaviors from one generation relates to developmental processes and outcomes in successive generations (e.g., Augustyn et al., 2020; Bailey et al., 2009; Capaldi et al., 2003; Neppl, Diggs, & Cleveland, 2020). Several studies that have leveraged

multigenerational data show that parental monitoring and harsh parenting tend to be correlated from one generation to the next (e.g., Bailey et al., 2009; Capaldi et al., 2003; Neppl et al., 2020), and suggest that intergenerational transmission of parenting practices gives rise, at least in part, to intergenerational consistencies in child adjustment outcomes (Haggerty & Carlini, 2020).

Research with participants spanning three generations—in which grandparents are the first generation (G1), parents are the second generation (G2), and children/grandchildren are the third generation (G3) of family members being studied—have shown that developmental outcomes associated with parenting behaviors (e.g., internalizing and externalizing problems) may be susceptible to intergenerational transmission either as a direct or indirect result of the previous generation's receipt of parenting practices. For example, in a study of parenting and risky behaviors across three generations, G1 poor parenting practices (e.g., infrequent monitoring behaviors and harsh discipline) predicted G3 challenging behaviors indirectly through G2 delinquency throughout adolescence and parenting practices in adulthood (Capaldi et al., 2003). Another study with three generations of individuals demonstrated intergenerational transmission of G1 parenting behaviors on G3 externalizing behaviors through G2s' externalizing behaviors in adolescence and substance use in adulthood (Bailey et al. 2009). Taken together, parenting behaviors experienced throughout adolescence may predict subsequent generations' behavioral outcomes (e.g., Bailey et al., 2009, Capaldi et al., 2003). However, additional work is needed to make well-informed conclusions about the role of environmental context in these longitudinal, intergenerational associations between parenting behaviors and children's behavioral outcomes.

The Current Study

Prior research suggests that parental monitoring is highly effective for preventing delinquent behaviors among adolescents in high-risk environments, and further, that parenting

characteristics (such as monitoring) may be intergenerationally transmitted to influence subsequent generation's adjustment outcomes (e.g., Bailey et al., 2009, Capaldi et al., 2003). Therefore, we conducted a pilot study with participants spanning three generations drawn from a subsample of The Strong African American Families Healthy Adults Project (SHAPE; Brody et al., 2004; Brody, Kogan, & Grange, 2012; Brody, 2016) to ask the question: Do young adults who received high-quality parenting (e.g., their parents were knowledgeable about their daily lives and activities) as adolescents go on to raise children who thrive behaviorally and emotionally? In other words, do children (G3s) reap the behavioral and psychosocial benefits of high-quality parenting experienced by their parents (G2s) when they were adolescents?

In this pilot study, we explored how G1s' (n = 48) parental knowledge throughout G2s' (n = 48) adolescence might forecast G3s' (n = 60) psychosocial development (e.g., risk-taking behaviors, self-control, and emotional well-being [anxiety and depressive symptoms]). We predicted that greater G1 parental knowledge across the G2s' adolescence would be associated with lower levels of G3 risk-taking, anxiety, depressive symptoms, and poor self-control. We also conducted exploratory mediation analyses to explore potential mechanisms for the association between G1 parental knowledge and G3 psychosocial outcomes. We predicted that G1 parental knowledge would be associated with better G2 parenting quality and lower substance use in early adulthood, which would in turn predict lower G3 risk-taking, anxiety, depressive symptoms, and poor self-control (Figure 3.1).

Additionally, to explore whether the intergenerational transmission of G1 parental knowledge on G3 psychological adjustment varied as a function of socioeconomic context, we conducted exploratory moderation analyses to evaluate whether family socioeconomic hardship throughout the G2s' adolescence modified the association between G1 parenting and G3

outcomes (Figure 3.2). Based on previous findings that parental knowledge might be more effective in high-risk than low-risk settings, we predicted that G1 parental knowledge in the G2's adolescence would be particularly protective for G3 outcomes in families that experienced higher levels of socioeconomic hardship.

Method

Participants

The SHAPE study was originally designed to test the effectiveness of an intervention to prevent adolescent externalizing behaviors, such as substance use, by promoting high-quality family processes and effective parenting (e.g., high parental warmth and appropriate monitoring behaviors) among a sample of African American families from rural Southern counties with high poverty rates. The original SHAPE sample (i.e., the G1 and G2 participants) was recruited from eight rural Georgia county-units in the early 2000s, when target youth (G2s) were approximately 11 years old (M = 11.2, SD = 0.34). (For detailed SHAPE study participant recruitment and methodology, see Brody, 2016; Brody, Kogan, & Grange, 2012; Brody et al., 2004.) At the first wave of data collection, a total of 667 caregiver-child dyads (i.e., G1s and G2s) participated, and primary caregivers reported a median household income of $1,612 per month; 42.3% of families were living below the federal poverty threshold. The majority of G1s were the biological mothers of the G2s, and a small proportion of G1 caregivers were the G2s' biological grandmothers. In the current sample of G1s (n = 48), 43 were biological mothers of the G2s, four were biological grandmothers, and one described themselves as having a different relation to the G2s. Following Wave 1 of SHAPE data collection, the G1s and G2s subsequently have been invited to participate in 12 additional rounds of data collection across a 20-year period.

A random subsample of G2s who participated in the most recent SHAPE assessment (Wave 12) and had at least one child ages 8-16 years old were contacted by research personnel and invited to participate in the current pilot study (n = 64). Families were eligible if children spent at least 50% of their time with the G2 parent, and they were excluded if the child had a cognitive or developmental disability. Of the G2s who were contacted and screened for eligibility, 38 mothers and 10 fathers (M_{age} = 26.5, SD = 0.6) enrolled with their child (or children) in the current study. Sixty G3 youth ages 8 to 16 participated in this study (46.7% male, M_{age} = 10.7, SD = 1.8); thirty-eight G2 parents had one child in the study, eight had two children in the study, and two parents had three children in the study.

Data Collection Procedures

For all waves of data collection, trained research personnel conducted home visits with families using standardized protocols. G1s and G2s (for prior waves of SHAPE data collection) and G2s and G3s (for the pilot study) completed questionnaires via private sessions with African American field researchers. For this pilot study, parents received $80, and children received a $20 gift card for participating. All waves of SHAPE and the pilot study were approved by a university Institutional Review Board, and all parents provided informed consent and children provided informed assent prior to participation.

Measures

G1 Parental Knowledge Throughout G2s' Adolescence

G1 parental knowledge was assessed via a 5-item Child Monitoring questionnaire (Conger, 1989; Spoth, Redmond, & Shin, 1998) administered during Waves 1, 2, 3, 5 and 6, when the G2s were 11, 12, 13, 16, and 17 years old (respectively). The items asked parents to report their knowledge of their child's activities and whereabouts. Example items are "*In the*

course of a day, how often do you know where [Target] is?" and "*How often do you know when [Target] gets in trouble at school or someplace else away from home?"* Items were scored on a scale from *(1) Never* to *(5) Always*, so that higher scores on all items reflected greater parental knowledge and monitoring of youths. The five items were summed at each wave (αs ranging from .63 to .74), and a mean score across all five waves of data collection was created (M = 22.65, SD = 1.76).

G3 Psychosocial Adjustment Outcomes

Depressive Symptoms**.** G3 youth reported on their depressive symptoms via the Center for Epidemiologic Studies – Depression (CES-D) Short Form (Andresen, 1994; Björgvinsson, 2013; Radloff, 1977). The CES-D Short Form is a 10-item questionnaire that asks participants to report on the extent to which they experienced depressive symptoms over the past seven days. Example items include: "*I was bothered by things that don't usually bother me"* and "*I felt depressed."* Seven items[5] were scored on a scale from (*0) rarely or none of the time (0-1 day)* to (*3) most of all of the time (6-7 days)* so that higher scores on all items reflected greater depressive symptomatology (α = .71). Item responses were averaged to create a mean depressive symptoms variable (M = 0.58, SD = 0.56).

Anxiety**.** The Generalized Anxiety and Social Phobia subscales from the Spence Children's Anxiety Scale (Spence, 1997; Spence, 1998; Spence, Barrett, & Turner, 2003) assessed the frequency youths experienced symptoms of anxiety. Six items assessed symptoms of generalized anxiety (e.g., "*I worry about things"* and "*When I have a problem, my heart beats really fast"*) and six items assessed social phobia (e.g., "*I feel afraid to use public toilets or bathrooms"* and "*I worry what other people will think of me"*). All items were scored on a scale

[5] Three items were dropped from the original 10-item questionnaire due to low reliability.

from (*0) never* to (*3) always,* such that higher scores indicated greater frequency of anxiety symptoms (α = .83). Items responses were averaged to create a mean generalized anxiety and social phobia score (*M* = 0.88, *SD* = 0.59).

Risky Behaviors**.** The extent to which G3s engage in risky behaviors was assessed via 10 items from the 2019 Middle School Youth Risk Behavior Survey (YRBS) questionnaire (Centers for Disease Control and Prevention, 2019). Four items assessed safety behaviors (e.g., "*When you ride a bicycle, how often do you wear a helmet?"* and *"How often do you wear a seatbelt when riding in the car?"*), two items assessed violence-related behaviors (e.g., "*Have you ever carried a weapon, such as a gun, knife, or club?"*), and four items assessed smoking, drug, and alcohol use (e.g., "*Have you ever tried cigarette smoking, even one or two puffs?"* and *"Have you ever used marijuana?"*). Item responses varied for each question, so items were recoded on a binary scale such that *(0)* reflected *No Risky Behavior Engagement/Experience* and *(1)* reflected *Risky Behavior Engagement/Experience*. Responses to the 10 items were summed to create a count variable, and higher scores indicated greater engagement in risky behaviors (*M* = 1.72, *SD* = 1.25).

Poor Self Control**.** Poor self-control was evaluated via the four-item Poor Self Control subscale of the Will's Self-Control Scale (Eysenck & Eysenck, 1997; Kendall & Williams, 1982; Wills, 1986). Youth reported on the extent to which statements about having poor self-control were true for them. Example items include "*You have to have everything right away"* and "*You often get in trouble because you do things without thinking*." Items were scored on a scale from (*1) Not At All True* to (*5) Very True.* All items were scored so that higher scores indicated poorer child self-control, and responses were averaged to create a mean poor self-control variable (*α* = .68, *M* = 2.82, *SD* = 1.09).

Exploratory Mediators

G2 Involved-Vigilant Parenting. G3s' perceptions of their parents' (G2s) involved-vigilant parenting were measured via the 18-item Involved-Vigilant Parenting Scale (Brody et al., 2001; Brody et al., 2003). Questions ask children about the extent to which their parents are involved in, and monitor. their everyday lives, as well as the extent to which the discipline they receive is consistent. Example items include *"In the course of a day, how often does your parent know where you are?"* and "*How often does your parent discipline you for something at one time, and then at other ties, not discipline you for the same thing?"* Items were scored on a scale from *(1) Never* to *(5) Always*. All items were scored so that higher scores represented greater parental involvement and vigilance (α = .82, M = 4.28, SD = .51).

G2 Substance Use. During the Wave 11 of data collection (spanning 2014-2016, when G2s were approximately 24.7 years old), G2s were asked about the frequency by which they engaged in cigarette smoking, marijuana use, alcohol use, and binge drinking over the past month (Johnston et al., 2007). Response options were *(0) None, (1) One to Three Days, (2) Four to Seven Days, (3) Eight to Twelve Days, (4) Thirteen to Nineteen Days,* or *(5) Twenty or More Days*. Responses were summed to create a composite substance use score, with higher scores indicating more frequent substance use over the past month (M = 2.37, SD = 2.96).

Family Socioeconomic Hardship Throughout G2s' Adolescence

Family socioeconomic hardship was assessed via two questionnaires administered to G1s during Waves 5, 6, and 7, when the G2s were 16, 17, and 18 years old (respectively). First, a 4-item Unmet Material Needs questionnaire (Conger and Elder, 1994) asked caregivers to reflect on the degree to which they agreed with statements about their family's financial situation. Example items are "*My family has enough money to afford the kind of home we need"* and "*We*

have enough money to afford the kind of medical care we need." Items were scored on a scale from *(1) Strongly Agree* to *(4) Strongly Disagree* and summed at each wave, so that higher scores indicated greater unmet material needs (αs ranging from .88 to .89). Summed scores were averaged across all waves to create a composite unmet material needs variable (M = 9.66, SD = 2.30).

In addition, an 11-item Financial Adjustments questionnaire (Conger and Elder, 1994) was administered to assess whether families experienced various socioeconomic hardships over the past 12 months. Example items include "*Has your family postponed major household purchase(s) because of financial need?"* and "*Has your family reduced household utility use to save money?"* Item responses were *(0) No* or *(1) Yes*, and items were summed at each wave to create a count variable with higher scores reflecting greater financial hardship. The summed scores were averaged across all waves to create a composite financial hardship variable (M = 2.40, SD = 2.17).

The mean unmet material needs and financial hardship variables were highly correlated (r = .62, $p < .001$) and were standardized and summed to create a Family Socioeconomic Hardship variable across the three waves of data collection (M = 0.0, SD = 1.8).

Covariates

G3 age, G3 sex (dummy-coded so that 0 = Male and 1 = Female), and G2 sex (dummy-coded so that 0 = Female and 1 = Male) were included as covariates in all GEE main effects and moderation analyses.

Data Analytic Plan

IBM SPSS statistical software was used to conduct regression analyses. Using generalized estimating equations (GEEs), with family specified as a clustering variable to

account for nonindependence of observations among siblings nested within families, we first evaluated the direct effects of G1 parental knowledge on G3 psychosocial adjustment outcomes. To do so, we conducted GEE models with the covariates and G1 parental knowledge entered as predictors of 1) G3 depressive symptoms, 2) G3 anxiety, 3) G3 risky behaviors, and 4) G3 poor self-control. We specified linear models for continuous outcomes of depressive symptoms, anxiety, and poor self-control, and a Poisson model for the risky behaviors outcome, which was a count variable.

In order to explore potential mechanisms for the link between G1 parental monitoring and G3 outcomes, we conducted exploratory mediation models in Mplus (Muthén & Muthén, 2021). We examined the association between G1 parental monitoring and potential mediators, G2 involved-vigilant parenting and substance use (path A), and the association between the mediators and G3 outcomes (path B). The mediating or indirect effects were quantified as the product of these two regression coefficients (A × B). All the mediation models used the Type = COMPLEX command of Mplus to account for non-independence of observations among G3 participants from the same family.

Finally, we evaluated whether there were significant interaction effects of G1 parental knowledge and family socioeconomic hardship (as reported by G1s) to predict G3 psychosocial outcomes. To do so, we entered the covariates and predictors (i.e., G1 Parental Knowledge and Family Socioeconomic Hardship) as well as the G1 Parental Knowledge × Family Socioeconomic Hardship interaction term using the GEE analysis framework. All interaction analyses were executed based on the conventions that Aiken and West (1991) prescribed, whereby the variables are first standardized, and interactions are calculated as the product of the

standardized variables. To probe the significant interactions, simple slopes were calculated at +1 and -1 *SD* values of the moderator.

Using G-Power software, we determined that our analytic sample was adequately powered (80%) to detect effects $\geq f^2 = .14$.

Results

Intercorrelations among study variables are presented in Table 3.1. G1 Parental Knowledge was negatively associated with G3 Depressive Symptoms, Risky Behaviors, and Poor Self-Control, but was not associated with G3 Anxiety. Family Socioeconomic Hardship was unrelated to all study variables, and among the exploratory mediators, G2 Substance Use was associated with G2 sex (such that males reported greater past-month substance use than females).

Does G1 Parental Knowledge Predict G3 Psychosocial Adjustment Outcomes?

Results of the four GEE models predicting G3 Depressive Symptoms, Anxiety, Risky Behaviors, and Poor Self-Control are presented in Table 3.2. G1 Parental Knowledge was a significant predictor of G3 Depressive Symptoms, Anxiety, Risky Behaviors, and Poor Self-Control after controlling for G3 Age, G3 Sex, and G2 Sex. Greater G1 Parental Knowledge predicted fewer depressive symptoms as well as lower anxiety, engagement in risky behaviors, and poor self-control among G3 participants.

Do G2 Involved-Vigilant Parenting or Substance Use Mediate the Association between G1 Parental Knowledge and G3 Outcomes?

Given evidence for associations among G1 parental knowledge and G3 psychosocial adjustment outcomes, we conducted follow-up analyses to examine potential mechanisms for the link between G1 parental knowledge of G2s and G3s' Anxiety, Depressive Symptoms, Risky Behaviors, and Poor Self-Control. Previous literature suggests that parental monitoring may be

associated with the next generation's own parenting quality as well as substance use in adulthood, which has implications for the subsequent generation's internalizing and externalizing behaviors (e.g., Bailey et al., 2009, Capaldi et al., 2003). Therefore, we conducted exploratory mediation analyses, with G3 Perceptions of G2 Involved-Vigilant Parenting and G2 Substance Use in young adulthood (approximately age 24.7) serving as mediators of the link between G1 parental knowledge and G3 adjustment outcomes. However, the indirect effects were not significant in any of the exploratory mediation models, with all confidence intervals straddling zero (Table 3.3).

Does the G1 Parental Knowledge × Family Socioeconomic Hardship Interaction Predict G3 Psychosocial Adjustment Outcomes?

Exploratory moderation analyses revealed that the G1 Parental Knowledge × Family Socioeconomic Hardship interaction was a predictor of G3 Anxiety (Table 3.4). Further, there was suggestive evidence that the interaction was related to G3 Depressive Symptoms (Table 3.4). However, the G1 Parental Knowledge × Family Socioeconomic Hardship interaction was unrelated to G3 Risky Behaviors and Poor Self-Control (Table 3.4).

Visual depictions of the interactions to predict Anxiety and Depressive Symptoms are presented in Figure 3.3. G1 Parental Knowledge was negatively associated with G3 Anxiety and Depressive Symptoms among the children of G2s who experienced higher (+1 *SD*) levels of Family Socioeconomic Hardship throughout adolescence (Anxiety: $b = -.343$, $p = .014$; Depressive Symptoms: $b = -.345$, $p = .013$). However, G1 Parental Knowledge was unrelated to G3 Anxiety and Depressive Symptoms among the children of G2s who experienced lower (-1 *SD*) levels of Family Socioeconomic Hardship throughout adolescence (Anxiety: $b = -.038$, $p = .565$; Depressive Symptoms: $b = .025$, $p = .742$).

Discussion

In this pilot study with three generations of individuals, we explored whether caregivers' knowledge of their child's whereabouts and activities throughout adolescence had implications for the next generation's psychosocial adjustment. Results suggest that parenting characteristics are indeed "intergenerationally transmitted" to influence subsequent generations' adjustment and well-being, and more importantly, that transmissions might be dependent on socioeconomic context. Grandchildren (G3s) whose grandparents (G1s) had greater knowledge of their parents' (G2s) whereabouts and activities throughout adolescence reported lower levels of depressive symptoms, anxiety, risk-taking behaviors, and poor self-control. These findings demonstrate that effective parenting (e.g., greater parental knowledge of adolescents' lives) may forecast the next generation's adjustment, and thus support findings from previous studies that have explored intergenerational influences of parenting on subsequent generations' internalizing and externalizing behaviors (e.g., Bailey et al., 2009, Capaldi et al., 2003).

We suspect that findings reflect continuity in family processes, wherein individuals' experiences with their caregivers contribute to their own parenting practices and their likelihood of engaging in substance use as young adults, which have implications for their children's psychosocial adjustment. In this pilot study, however, we did not find evidence that G3s' perceptions of G2 involved-vigilant parenting or G2s' reported substance use in young adulthood mediated the association between G1 parental knowledge and G3 psychosocial adjustment. These results are in line with what we would expect with a sample size that is underpowered to detect small indirect effects. Nonetheless, strong associations between G1 parental knowledge and G3 adjustment outcomes suggest that G2 parenting or behavioral factors may serve as possible intermediaries for G1 to G3 transmissions. Our team is continuing to explore potential

mechanisms by which G1 parenting influences G3 outcomes through G2 experiences and characteristics in a follow-up investigation with a larger portion of the sample.

Despite nonsignificant indirect effects to explain the link between G1 parental knowledge and G3 psychosocial adjustment, exploratory moderation analyses revealed that among G3 children whose parents experienced high levels of family socioeconomic hardship throughout adolescence, those whose grandparents reported greater parental knowledge of their parents in adolescence had fewer emotional distress symptoms (i.e., lower levels of anxiety and suggestive evidence for fewer depressive symptoms) relative to children whose grandparents reported lower parental knowledge. However, the link between grandparents' parental knowledge and grandchildren's risk-taking behaviors and poor self-control did not vary as a function of parents' family socioeconomic hardship in adolescence. Although we await replication with a larger sample to draw firm conclusions, findings are consistent with the notion that parental knowledge is highly protective and critical for children in lower-resourced environments where the risk for experiencing emotional distress is high. Conversely, parental knowledge might not be as essential for children in more well-resourced environments where the risk for experiencing emotional distress is low (e.g., Rekker et al., 2017).

Of note, our sample of G3s included children aged 8 to 16 years old, so there was a wide range of G3 ages as well as G2 ages at the time G3s were born. G2s' age at the time of G3 birth might play a role in the extent to which children reaped the benefits of effective G1 parenting. For example, G2s who entered parenthood in late adolescence and thus had to navigate becoming a parent during their own transition to adulthood may have had less time to focus on using effective parenting practices, relative to G2s who waited longer before becoming parents (e.g., those who became parents in their mid-twenties). Moreover, it may be the case that G1

parenting matters more for G3 adjustment during particular developmental periods (e.g., during early adolescence, when depressive symptoms tend to increase). Half of the G3 children in our sample (48%) were in early adolescence (i.e., were 10 or 11 years old). Therefore, effects of G1 parental knowledge on G3 outcomes might look different in a sample of children who are predominantly older and more likely to have experienced social stressors or risky exposures that occur later in adolescence (e.g., peer and romantic conflict, exposure to peer pressure, academic stress, etc.), which play a role in psychosocial adjustment. However, G3 age did not serve as a significant moderator for associations among G1 parental knowledge and G3 psychosocial adjustment outcomes (*b*s = -.041 - .021, *p*s > .29). Future work focusing on G3 age and G2 age at the time of G3 birth represents exciting directions to evaluate the extent to which developmental timing shapes G1's influence on G3 adjustment.

This pilot study was largely exploratory, and we await replication studies that are adequately powered to detect small direct and indirect effects. Nonetheless, results suggest that G1s' parental knowledge throughout G2s' adolescence predicts G3s' depressive symptoms, anxiety, poor self-control, and risk-taking behaviors, and that intergenerational influences on G3s' psychosocial adjustment may vary depending on socioeconomic context. Collectively, these findings highlight the importance of considering how experiences in prior generations can set a cascade of effects in motion that contributes to the mental health of subsequent generations.

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Tables

Table 3.1
Correlations and descriptive statistics among study variables

Variable	1	2	3	4	5	6	7	8	9	10	11
1. G3 age	—										
2. G3 sex, female	-.23	—									
3. G2 sex, male	-.13	.01	—								
4. G1 parental knowledge	-.02	.09	.00	—							
5. Family socioeconomic hardship	.05	-.11	-.09	.10	—						
6. G3 depressive symptoms	-.14	.01	-.06	-.28*	.07	—					
7. G3 anxiety	-.04	.02	.00	-.19	.22	.66***	—				
8. G3 risky behaviors	.03	-.12	.02	-.28*	-.10	.10	.06	—			
9. G3 poor self-control	-.05	-.02	-.15	-.41**	.08	.45***	.49***	.16	—		
10. G2 Involved-Vigilant Parenting	.13	-.08	-.12	.00	-.03	.13	.12	-.03	-.19	—	
11. G2 Substance Use Over the Past Month	-.16	.25	.43**	-.02	.01	.04	.11	.05	.07	-.15	—
Range	8 – 16	0 – 1	0 – 1	18.5 – 25.0	-2.99 – 3.56	0 – 2.14	0 – 2.75	0 – 5	1 – 5	1.89 – 4.87	0 – 11
Mean (SD)	10.7 (1.8)	—	—	22.7 (1.8)	0.0 (1.8)	.58 (.56)	.88 (.59)	1.7 (1.3)	2.8 (1.1)	3.74 (.73)	2.36 (3.0)

Note. N = 60 for all variables except G2 Substance Use Over the Past Month (n = 55).
**p < .05. **p < .01. ***p < .001.*

Table 3.2

G1 Parental Knowledge with G3 Depressive Symptoms, Anxiety, Risky Behaviors, and Poor Self-Control

	G3 Depressive Symptoms		G3 Anxiety		G3 Risky Behaviors		G3 Poor Self-Control	
Predictors	*b*	[95% CI]	*b*	[95% CI]	*b*	[95% CI]	*b*	[95% CI]
1. G3 age	-.052	[-.118, .014]	-.016	[-.089, .058]	.004	[-.123, .131]	-.059	[-.186, .067]
2. G3 sex, female	.025	[-.230, .281]	.064	[-.206, .334]	-.141	[-.527, .246]	-.009	[-.492, .474]
3. G2 sex, male	-.212	[-.464, .040]	-.096	[-.365, .173]	.060	[-.370, .490]	-.581*	[-1.044, -.117]
4. G1 parental knowledge	-.163**	[-.260, -.067]	-.117*	[-.226, -.008]	-.185*	[-.337, -.032]	-.466***	[-.651, -.281]

*Note. N = 60. b = unstandardized regression coefficient; CI = confidence interval. *p < .05. **p < .01. ***p < .001.*

Table 3.3

Indirect Effects from Mediation Models with G3 Perceptions of G2 Involved-Vigilant Parenting and G2 Substance Use as Mediators for Links Between G1 Parental Knowledge and G3 Depressive Symptoms, Anxiety, Risky Behaviors, and Poor Self-Control

Mediators	G3 Depressive Symptoms		G3 Anxiety		G3 Risky Behaviors		G3 Poor Self-Control	
	Indirect Effect	[95% CI]	Indirect Effect	[95% CI]	Indirect Effect	[95% CI]	Indirect Effect	[95% CI]
1. G3 Perception of G2 Involved-Vigilant Parenting (*n* = 60)	.000	[-.009, .009]	.000	[-.009, .009]	.000	[-.017, .017]	.000	[-.006, .006]
2. G2 Substance Use Over the Past Month (*n* = 55)	-.001	[-.011, .009]	-.001	[-.016, .014]	.000	[-.008, .007]	-.002	[-.029, .025]

Note. Fifty-five of the G2s in the current study participated in the previous wave of data collection, when the G2 substance use data were collected.

Table 3.4

Interaction of G1 Parental Knowledge and Family Socioeconomic Hardship with G3 Depressive Symptoms, Anxiety, Risky Behaviors, and Poor Self-Control

	G3 Depressive Symptoms		G3 Anxiety		G3 Risky Behaviors		G3 Poor Self-Control	
Predictors	*b*	[95% CI]	*b*	[95% CI]	*b*	[95% CI]	*b*	[95% CI]
1. G3 age	-.064*	[-.123, -.006]	-.031	[-.102, .040]	.015	[-.102, .132]	-.072	[-.206, .063]
2. G3 sex, female	.073	[-.178, .325]	.143	[-.109, .395]	-.200	[-.613, .212]	.056	[-.447, .558]
3. G2 sex, male	-.150	[-.427, .126]	-.005	[-.262, .251]	-.015	[-.480, .450]	-.507*	[-.953, -.061]
4. G1 parental knowledge	-.192**	[-.301, -.082]	-.159**	[-.272, -.046]	-.146*	[-.279, -.014]	-.500***	[-.697, -.303]
5. Family socioeconomic hardship	.076	[-.072, .223]	.174*	[.019, .328]	-.077	[-.283, .129]	.080	[-.062, .222]
6. G1 parental knowledge × family socioeconomic hardship	-.154^{+}	[-.330, .023]	-.184*	[-.367, -.001]	.156	[-.069, .381]	-.082	[-.236, .073]

Note. N = 60. b = unstandardized regression coefficient; CI = confidence interval. $^{+}p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

Figures

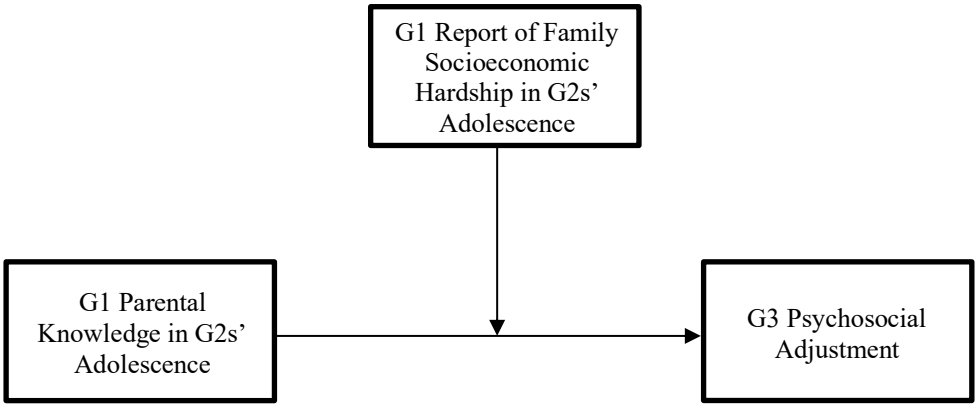

Figure 3.1. Conceptual moderation model.

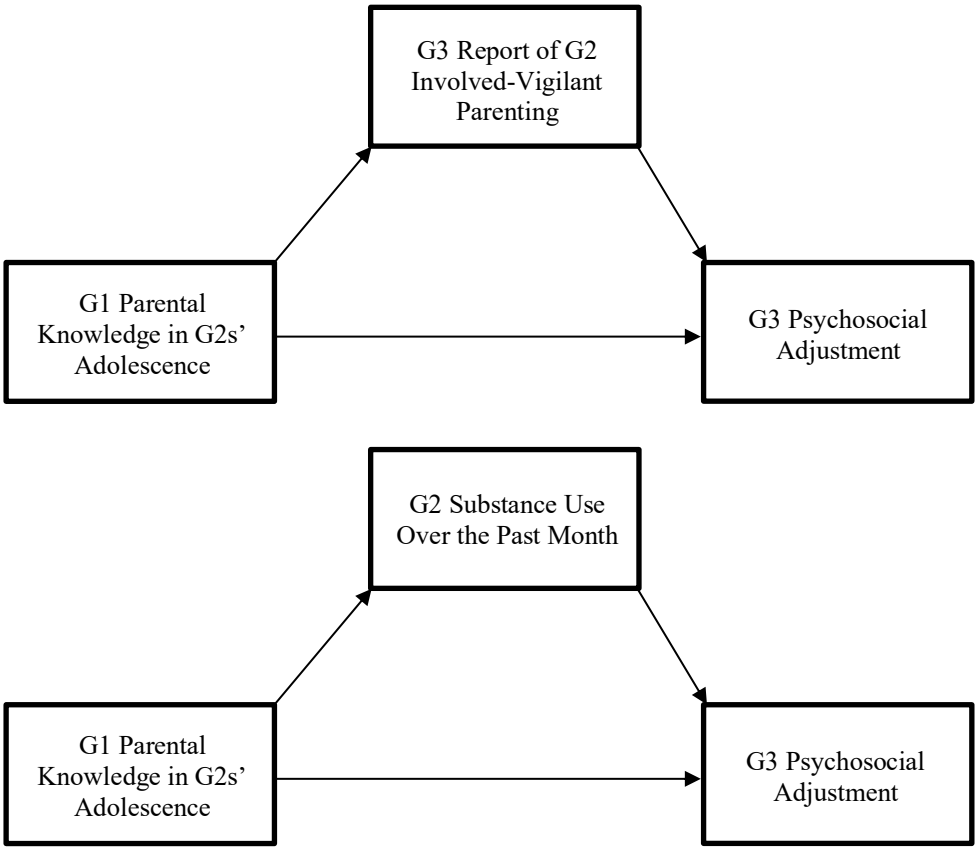

Figure 3.2. Conceptual Mediation Models with G3 Report of G2 Involved-Vigilant Parenting (top) and G2 Substance Use Over the Past Month (bottom) as mediators for the link between G1 Parental Knowledge in G2s' Adolescence and G3 Psychosocial Adjustment.

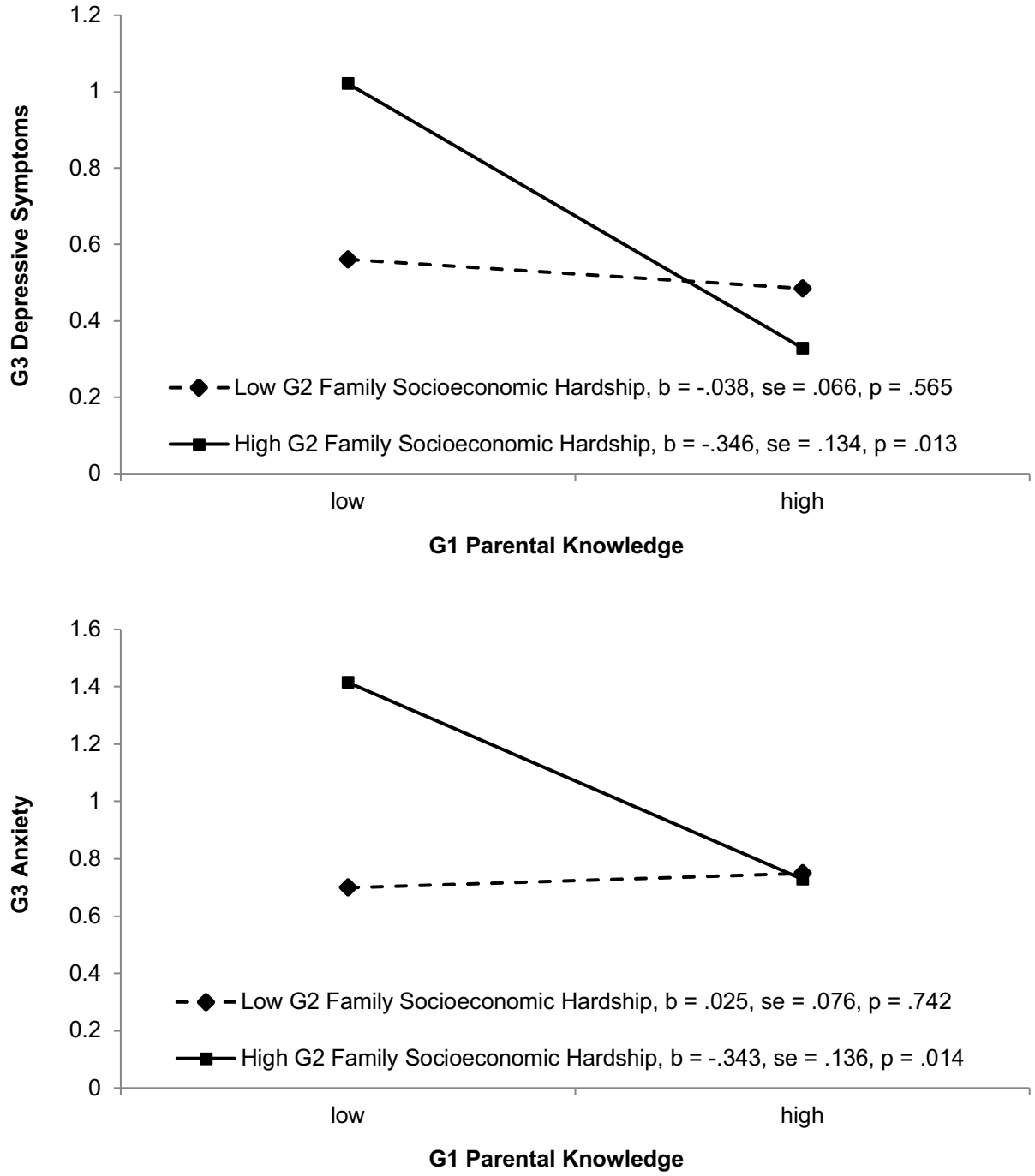

Figure 3.3. G3 depressive symptoms (top) and anxiety (bottom) as a function of G1 parental knowledge by family socioeconomic hardship. Low and high values of G1 parental knowledge and family socioeconomic hardship represent +/- 1 SD from the mean.

CHAPTER 4

THE PROTECTIVE ROLE OF PARENTAL VIGILANCE IN THE LINK BETWEEN RISKY CHILDHOOD ENVIRONMENTS AND HEALTH [6]

[6] Corallo, K. L., Carr, C. P., Lavner, J. A., Koss, K. J., Ehrlich, K. B. Submitted to *Social Science & Medicine,* 4/12/22

Abstract

Growing up in a risky environment is associated with poor lifespan physical and mental health. However, promotive factors that have protective or compensatory effects (i.e., buffering against negative outcomes or promoting positive ones in the context of risk) allow individuals to remain healthy despite adverse upbringings. Parental vigilance, or tendencies to supervise and surveille children and adolescents, has been shown to buffer and protect against negative health outcomes among individuals who grow up in risky environments. Conversely, parental vigilance has been shown to be maladaptive for, or unrelated to, health among individuals who are raised in low-risk environments. The current study leveraged longitudinal data from the National Longitudinal Survey of Youth 97 (NLSY97; https://www.nlsinfo.org/content/cohorts/nlsy97) to explore the link between environmental risk in adolescence and indices of physical and mental health in young adulthood, and whether parental vigilance (monitoring and limit-setting) buffered these associations. Results indicated that childhood environmental risk significantly predicted a greater likelihood of experiencing physical health limitations at age 29 but was not significantly associated with mental health symptoms at approximately age 34. Further, there was evidence that parental limit-setting (but not monitoring) significantly buffered the relation between childhood risk and physical health limitations, such that the association between risk and physical limitations became less pronounced at greater levels of parental limit-setting. Results highlight the importance of parental limit-setting in reducing long-term health consequences associated with childhood risk.

Keywords: childhood risk, parental monitoring, parental limit-setting, mental health, physical health limitations

Introduction

Characteristics associated with one's neighborhood and home environments are associated with health through the lifespan. Residing in low-resourced and/or unsafe neighborhoods is linked to poorer physical and mental health in childhood and adulthood (e.g., Allen et al., 2014; Ellen et al., 2001; Essex et al., 2011; Evans, 2003; Jutte et al., 2015). In addition, adverse home environments—e.g., living conditions associated with poverty or unpaid bills, such as electricity shortages and unkempt or inadequate furniture and appliances—in childhood have long-term implications for individuals' physical and emotional well-being (e.g., Bonnefoy et al., 2003; Liddell & Morris, 2010). High-risk neighborhood and home environments are associated with greater prevalence and severity of chronic health conditions such as asthma (in both childhood and adulthood; e.g., Gold & Wright, 2005), obesity and diabetes (e.g., Ludwig et al., 2011), cardiovascular disease (e.g., Malambo et al., 2016), and anxiety and depression (e.g., Hill & Maimon, 2012).

There are several pathways that may explain associations between risky early environments and poor health trajectories. Exposure to pollutants (e.g., toxins that cause or exacerbate health conditions such as asthma) and engagement in harmful levels of substance use (e.g., frequent alcohol consumption and using addictive drugs), which is more prevalent in risky environments (e.g., Furr-Holden et al, 2015; Leza et al., 2021; Kameg & Mitchell, 2021), are potential links between adverse childhood environments and adult wellbeing. In addition, neighborhood poverty is often associated with a greater likelihood of experiencing childhood stressors, such as abuse, neglect, or family dysfunction (e.g., Evans & Cassells, 2014; Repetti et al., 2002); in turn, these stressors beget physiological disruptions that can lead to mental health

disorders and chronic diseases of aging (Anda et al., 2005; Edwards et al., 2003; Essex et al., 2011; Miller et al., 2011).

In risky environments, vigilant parenting characteristics—parenting behaviors that seek to monitor, protect, and sometimes even control children and adolescents—have been shown to buffer against negative developmental outcomes (e.g., serve as protective factors) as well as promote healthy development in adverse contexts (e.g., have compensatory effects; Bradley et al., 1994; Masten, 2018; Zimmerman et al., 2013). Several studies have shown that in risky environments, greater levels of parental vigilance are associated with better adjustment outcomes throughout childhood and adolescence. For example, parental monitoring serves as a protective factor for externalizing and antisocial behaviors among adolescents experiencing higher levels of risk (Laird et al., 2009; Pettit et al., 1999). Additionally, in accordance with a compensatory model, high levels of parental monitoring have been found to be associated with a lower risk for externalizing behaviors (Bendezú et al., 2018) and early-onset conduct problems (Kilgore et al., 2000) among children residing in low-resourced and unsafe neighborhoods. Accordingly, effective monitoring and vigilance may protect against some of the negative outcomes associated with adverse environments as well as promote positive outcomes in the context of experiencing risk.

Conversely, in lower-risk, high-resourced settings where harmful social and environmental exposures are low, greater levels of parental monitoring may be ineffective or even maladaptive for child and adolescent development (Garthe et al., 2018; Gonzales et al., 1996; Kliewer et al., 2016; McElhaney & Allen, 2001). In lower-risk settings, high levels of parental monitoring can be interpreted as overcontrolling and inhibiting of children's autonomy (e.g., Laird et al., 2018; McElhaney & Allen, 2001; Soenens et al., 2014). Taken together, these

findings suggest that parental monitoring can promote healthy development among children in risky home and neighborhood environments but might be associated with poor outcomes for individuals raised in higher-resourced environments, consistent with the idea that children's needs and interpretations of caregivers' actions are unique to their environment (Soenens et al., 2014).

Behavioral and physical health outcomes associated with parental vigilance are also context dependent. For example, parental control serves as a protective factor and prevents negative externalizing trajectories among adolescents with deviant peers (Galambos et al., 2003; Mason et al., 1996). Further, greater undermining of children's autonomy was associated with positive indices of social functioning among adolescents from high-risk contexts (i.e., lower levels of delinquent activity) and negative indices of social functioning among adolescents from low-risk contexts (i.e., lower levels of friendship competence; McElhaney & Allen, 2001). There is some evidence that these patterns may extend to physical health as well. For example, Corallo et al. (2021) found a negative association between neighborhood risk and cold susceptibility among individuals who retrospectively reported high levels of parental control. Further research is needed to disentangle whether vigilant parenting characteristics measured in childhood have long-lasting ties to individuals' wellbeing into adulthood. To our knowledge, no studies have explored prospective associations among childhood environments, parental vigilance, and adult physical and mental health outcomes using longitudinal datasets, the "gold standard" practice in developmental science.

In the current study, we addressed this gap by leveraging a publicly available longitudinal dataset, the National Longitudinal Survey of Youth (NLSY; Bureau of Labor Statistics, U.S. Department of Labor, 2021) to explore whether children's perceptions of parental vigilance

(monitoring and limit-setting behaviors) throughout adolescence influence associations between childhood environmental risk and mental and physical health in adulthood. The NLSY is a multi-wave longitudinal study spanning from 1997-2020. Information about youths' neighborhood and home environment was collected at the first wave of data collection, when participants were in their early- to mid-adolescence, and parental vigilance measures were collected during the first three rounds of data collection (i.e., throughout a three-year span of youths' adolescence). Assessments of mental and physical health have been administered sporadically throughout various waves of data collection. In the current study, we used a measure of mental health symptoms during the 18th round of data collection (when participants were, on average, approximately 34 years old) and a measure of physical health limitations collected during a supplemental questionnaire administered when participants were 29 years old. These measures represent the most recent, pre-pandemic assessments of mental and physical health in the NLSY97 dataset.

Given strong evidence that childhood environmental factors predict health across the lifespan (e.g., Allen et al., 2014; Ellen et al., 2001; Essex et al., 2011; Evans, 2003; Jutte et al., 2015), we hypothesized that greater childhood environmental risk would be associated with greater mental health symptoms and physical health limitations in adulthood. However, we hypothesized that parental vigilance (monitoring and parental limit-setting) would moderate these associations, such that higher levels of parental vigilance throughout adolescence would offset the risk for poor health among individuals who grew up in high-risk environments (i.e., parental vigilance would serve as a protective factor in high-risk environments). We also hypothesized that higher levels of parental vigilance throughout adolescence would be unrelated

to, or potentially maladaptive for, physical and mental health (i.e., increase the risk for poor health) among individuals who grew up in low-risk environments.

Method

Participants

Participants are a subsample of individuals who participated in the National Longitudinal Survey of Youth (NLSY; Bureau of Labor Statistics, U.S. Department of Labor, 2021), an ongoing investigation of youths followed into adulthood. At the initial round of data collection in 1997 (Round 1), adolescents ages 12-16 were recruited to participate in the NLSY; however, interview questions that assessed physical environmental risk, parental monitoring, and parental limit-setting were only administered to youths ages 12-14, and therefore participants that were 15 or older at the time of Round 1 data collection were excluded from the current study (*n* 12-14-year-olds = 4,829; *n* 15-16-year-olds = 4,155). Adolescents were initially recruited to be representative of people living in the United States ("cross-sectional sample"; *n* = 3,655 in the current study). An additional subsample of adolescents was subsequently recruited to oversample Hispanic or Latino and Black adolescents ("supplemental sample"; *n* = 1,174 in the current study). Collectively this resulted in 4,829 participants in the present analysis. See Table 4.1 for demographic characteristics among the full sample of youths ages 12-14 at the initial round of data collection (Round 1, 1997).

Procedure

Participants have been invited to participate in a total of 19 rounds of data collection, spanning from 1997-2020. Data for the current study are drawn from the first three consecutive rounds of NLSY97 data collection (Round 1: 1997, Round 2: 1998, and Round 3: 1999), Round 18 (which took place in 2017), and a supplemental round when participants turned 29 years old

(between 2012-14, depending on the participant's birth year). For all rounds of data collection, participants completed in-person or phone interviews with researchers. During Rounds 1-3, youth responded to questions about their home life, family relationships, academic performance, health, psychosocial wellbeing, and delinquency. Round 18 interviews included questions about participants' home lives, education, career, income, children, and health. Retention rates for Rounds 2, 3, and 18 were 93.3%, 91.4%, and 79.0% (respectively). See Table 4.1 for demographics among the subsample of participants who have Round 18 mental health data.

From Round 13 (for which data collection took place from 2009-2010) onwards, participants who had turned 29 years old since the previous round of data collection were invited to answer a supplemental series of questions about their current health (described below) and their family history of chronic health conditions. Of the 4,829 participants who were included in current analyses, age 29 health limitations data were available for 4,378 participants. See Table 4.1 for demographics among the subsample of participants with age 29 health limitations data.

Incentives for participating in each round of data collection have varied. Data collection procedures were approved by the U.S. Office of Management and Budget as well as the Ohio State University and NORC at the University of Chicago Institutional Review Boards. For more information about NLSY97 sampling and data collection procedures, please see https://www.nlsinfo.org/content/cohorts/nlsy97.

Measures

Childhood Environmental Risk

A five-item Physical Environment Risk Index, created by Child Trends, Inc. and the Center for Human Resource Research at The Ohio State University (Moore et al., 1999), served as the measure of childhood environmental risk. Information collected from youth interviews and

interviewer observations during the Round 1 (1997) visit was used to create the Index (Bradley et al., 1994; Dryfoos, 1990; Haggerty et al., 1996). Items were: 1) *In the past month, has your home usually had electricity and heat when you needed it?* (youth report), 2) *How well kept are most of the buildings on the street where the adult/youth resident lives?* (interviewer report), 3) *How well kept is the interior in the home in which the youth respondent lives?* (interviewer report), 4) *When you went to the respondent's neighborhood/home, did you feel concerned about your safety?* (interviewer report), and 5) *In a typical week, how many days from 0 to 7 do you hear gunshots in your neighborhood?* (youth report). Items 1, 4, and 5 were coded on a 0 – 1 scale: (0) *no risk* and (1) *risk*. Items 2 and 3 were coded on a 0 – 2 scale, with (0) *no risk,* (1) *risk or moderate risk,* and (2) *high risk*. Item response scores were summed, and higher scores indicated greater childhood environmental risk. See Table 4.2 for the mean, standard deviation, and observed range. For more information about this index, see the NLSY Appendix 9 of the codebook supplement (Moore et al., 1999) and for previous studies that have used this measure, see Day et al. (2009) and Jones-Sanpei et al. (2009). Per the NLSY Appendix (Moore et al., 1999), because the measure was an index rather than a scale, internal consistency statistics could not be ascertained.

Parental Vigilance

Adolescents' perceptions of their mothers' monitoring behaviors and parental limit setting (i.e., parental autonomy control) were assessed at Rounds 1-3 (1997, 1998, and 1999). Latent maternal monitoring and parental limit-setting variables were created using the mean scores from each round that these measures were collected (more information about the creation of these latent variables to represent Maternal Monitoring and Parental Limit-Setting across the three years of analysis is described in the Data Analytic Plan section). Of note, both youths' and

their residential parents' perceptions of maternal monitoring and parental limit-setting were collected in NLSY97. However, we restricted the parental vigilance measures in the current study to youths' reports because previous research suggests children's perceptions of parents' behaviors are often different from parents' perceptions (Achenbach, 2006; Guion et al., 2009; Pasch et al., 2010), particularly among adolescents (e.g., Tein et al., 1994), and examining this discrepancy was beyond the scope of the current study.

Youth Report of Parental Monitoring by Mothers. Four items asked youth to report on the extent to which their residential mothers knew about their relationships and whereabouts (Hetherington et al., 1982; Maccoby & Mnookin, 1992; Moore et al., 1999). Items were: 1) *How much does she know about your close friends, that is, who they are?,* 2) *How much does she know about your close friends' parents, that is, who they are?,* 3) *How much does she know about who you are with when you are not at home?,* and 4) *How much does she know about who your teachers are and what you are doing in school?* Items were scored on a 0 – 4 scale, with higher scores indicating greater parental knowledge and monitoring. See Table 4.2 for the mean, standard deviation, and observed range. Cronbach's alpha = .71.

Youth Report of Parent-Child Limit Setting. Three items asked youth to report on the extent to which their parents set limits on activities, versus youth having their own autonomy in limit-setting (Eccles et al., 1991; Erford, 1995; Moore et al., 1999). Items were: 1) *Who set the limits on how late you stay out at night?,* 2) *Who set the limits on who you can hang out with?,* and 3) *Who set the limits on what kinds of TV shows or movies you can watch?* Items were scored on a 3-point scale: (0) *parent or parents let me decide*, (1) *my parents and I jointly decide*, and (2) *parent or parents set limits*. Responses to the three items were summed so that higher scores indicated greater parental limit-setting and less youth autonomy in limit-setting, and lower

scores indicated greater youth limit-setting and autonomy (and less parental limit-setting). See Table 4.2 for the mean, standard deviation, and observed range. Per the NLSY Appendix 9 (Moore et al., 1999), because the measure was an index rather than a scale, internal consistency statistics could not be ascertained.

Mental Health (Round 18, Age ~34)

Participants reported on their mental health via five items that assessed their mood and feelings over the past month (Strand et al., 2003; Rumpf et al., 2001). Items were: 1) *How much of the time during the last month have you been a very nervous person?*, 2) *How much of the time during the last month have you felt calm and peaceful?*, 3) *How much of the time during the last month have you felt downhearted and blue?*, 4) *How much of the time during the last month have you been a happy person?*, and 5) *How much of the time during the last month have you felt so down in the dumps that nothing could cheer you up?* Items were scored on a 4-point scale: (1) *All of the time*, (2) *Most of the time*, (3) *Some of the time*, and (4) *None of the time*. Items 1, 3, and 5 were reverse scored and responses were averaged so that higher scores reflected poorer mental health. See Table 4.2 for the mean, standard deviation, and observed range. Cronbach's alpha = .79.

Health Limitations (Age 29)

Self-reported physical health at age 29 was assessed via five items asking respondents to report on the extent to which they experienced physical health limitations in their daily lives and activities (Hays et al., 1993). Items were: 1) *Does your health limit you in moderate activities, such as moving a table, pushing a vacuum cleaner, bowling or playing golf?,* 2) *Does your health limit you in climbing several flights of stairs?,* 3) *During the past 4 weeks, have you accomplished less than you would like with your work or other regular daily activities as a result*

of your physical health?, 4) *During the past 4 weeks, were you limited in the kind of work or other activities as a result of your physical health?,* and 5) *During the past 4 weeks, how much did pain interfere with your normal work (both work outside of the home and housework)?* All items were scored on a 1 – 3 scale: (1) *Yes a lot*, (2) *Yes a Little*, and (3) *No Not at All*. Given a high frequency of young adults reporting no health limitations (78.2%), items were recoded into binary responses, such that a score of 3 became 0 (indicating no limitations) and a score of 2 or 1 became a 1 (indicating at least some amount of limitation). Then, the binary scores were summed to create a count variable with possible scores ranging from (0) *no health limitations* to (5) *health limits on all items*. See Table 4.2 for the mean, standard deviation, and observed range.

Covariates

To control for demographic-level differences in childhood environments, parenting, and mental and physical health in adulthood, age (in years) at the first round of data collection (i.e., in 1997), youth-reported biological sex (coded as [0] *female* and [1] *male*), race (dummy-coded with White as the reference category), and sample type (0 = *cross-sectional subsample*; 1 = *supplemental subsample*) were included in all analyses.

Data Analytic Plan

Item and scale descriptive statistics and bivariate correlations were assessed using IBM SPSS Statistics Version 26. Then, the data were analyzed using Stata (StataCorp, 2021) to conduct regression and moderation path analyses. First, we explored whether childhood environmental risk was a predictor of 1) Mental Health at Age ~34 and 2) Health Limitations at Age 29 by conducting regression path analyses. Then, we conducted moderation analyses to explore the Childhood Environmental Risk × Maternal Monitoring and Childhood

Environmental Risk × Parental Limit-Setting interactions to predict 1) Mental Health at Age ~34 and 2) Health Limitations at Age 29.

Maternal Monitoring and Parental Limit-Setting were both latent variables with three indicators: maternal monitoring or parental limit-setting in 1997, 1998, and 1999, respectively. We used fixed factor scaling procedures and examined factor loadings to ensure that the three indicators loaded appropriately onto one construct (i.e., Maternal Monitoring or Parental Limit-Setting) across the three rounds these measures were assessed. We probed significant interaction terms by examining simple effects at the mean and +/- 1 *SD* from the mean of the latent moderator.

Health limitations was a count variable, meaning that Poisson and negative binomial regression frameworks were required for path analyses with this outcome. In these frameworks, the regression coefficients represent the predicted change in the log-count of the variable; as such, coefficients were exponentiated to obtain meaningful interpretations.

Results

Preliminary Analyses

Table 4.1 displays sample characteristics among all participants in 1997 (Round 1), and among those with available mental health and health limitations data. Table 4.2 displays descriptive statistics and bivariate correlations among the study variables used in path analyses. The parental vigilance variables (i.e., Maternal Monitoring and Parental Limit-Setting) were all positively correlated with one another in 1997, 1998, and 1999 (Rounds 1-3). There were significant negative associations between Childhood Risk and Maternal Monitoring in 1997, 1998, and 1999, indicating that youth who resided in higher-risk homes reported consistently lower levels of maternal monitoring across the years of analysis. There was a positive association

between Childhood Risk and Parental Limit-Setting in 1999, but no significant association between these variables in 1997 or 1998. None of the parental vigilance measures were significantly associated with Health Limitations at Age 29; however, Maternal Monitoring from 1997-1999 and Parental Limit-Setting in 1997 were negatively associated with Mental Health at Age ~34. Moreover, Childhood Risk was positively associated with Health Limitations at Age 29, but not significantly associated with Mental Health at Age ~34.

Main Effects of Childhood Environmental Risk on Adult Health

Mental Health at Age ~34

Environmental risk in childhood was not a significant predictor of mental health symptoms in adulthood; *b* (SE) = .01 (.01), *p* = .09 (Table 4.3).

Health Limitations at Age 29

Prior to conducting the regression analyses, we considered several potential models appropriate for count data including the Poisson and negative binomial models as well as the zero-inflated models of each. Upon inspecting the descriptive statistics and distribution of values for the Health Limitations count variable, we determined that a zero-inflated negative binomial (ZINB) model was the most appropriate for the data for several reasons. First, the mean was much smaller than the standard deviation (*M* = .44, *SD* = 1.03); following guidelines described by Coxe et al. (2009), a negative binomial model is preferred over a Poisson model given that the Poisson model assumes a normal distribution (i.e., *M* = *SD*). Second, there was an overdispersion (i.e., abundance) of zeros in the distribution of values for the Health Limitations count variable (70.9% of individual reported experiencing 0 health limitations), providing support that a zero-inflated model is appropriate for the data. Third, the -2 log-likelihood values of the zero-inflated Poisson (ZIP) and ZINB models were 6,338.88 and 6,269.08, respectively, and the model with

the smaller value is a better fit for the data (Coxe et al., 2009). In addition, the ZINB model's Vuong statistic was statistically significant ($V = 2.64$, $p = .004$), indicating that the zero-inflated model was supported over the non-zero-inflated model (Greene, 1994).

In the ZINB model, there are two sets of model results. First, the logistic (or INFLATE) model coefficients represent the log-odds of being a structural (or "excessive") zero (e.g., absence of health limitations) in the dataset[7] for every one-unit change in the predictor. Second, the negative binomial (or COUNT) model coefficients represent the change in the log-count of health limitations (i.e., the "count" ranging from 0-5) for every one-unit change in the predictor while accounting for the structural zeros in the dataset.

The results from the regression models exploring the link between Childhood Environmental Risk and Health Limitations at Age 29 (using ZINB regression) are presented in Table 4.3. Childhood Risk significantly predicted the log-odds of being a structural zero in the dataset (i.e., reporting no health limitations); *b* (SE) = -.14 (.05), p =.01. For every one-unit increase in childhood risk, the odds of reporting no health limitations decreased by a factor of .87 (see Equation A). Additionally, there was a positive but non-significant association between childhood risk and the log-count of health limitations (i.e., the number of health limitations individuals reported; *b* [SE] = .07 [.04], $p = .07$). In other words, childhood risk significantly reduced the likelihood of reporting no health limitations in early adulthood (e.g., predicted being a structural zero in the dataset) but did not significantly predict the number of health limitations individuals reported.

$$\text{(A)} \quad e^{(-.14)} = .87$$

[7] The ZINB model takes factors into account that influence individuals reporting "no health limitations" despite having health limitations (e.g., misinterpreting the questions, not wanting to report physical health limitations, etc.) which represent the zeros within the count portion of the model.

Childhood Risk × Parental Vigilance Interactions on Adult Health

Using the marker variable scaling method to create the Maternal Monitoring latent variable (with Maternal Monitoring in 1997 as the fixed marker variable), the factor loadings were 1 (fixed marker indicator), 1.53, and 1.36 (all *p*s < .001) for Maternal Monitoring in 1997, 1998, and 1999, respectively. Using the same method to create the Parental Limit-Setting latent variable (with Parental Limit-Setting in 1997 as the fixed marker variable), the factor loadings were 1 (fixed marker indicator), 1.32, and 1.08 (all *p*s < .001) for Parental Limit-Setting in 1997, 1998, and 1999, respectively. Given that the variables loaded appropriately onto their respective factors, we proceeded to use the latent variables in the moderation analyses described below.

For the moderation models predicting Health Limitations at Age 29, ZINB regression analyses were conducted using the same rationale as described above.

Predicting Mental Health at Age ~34

The Childhood Environmental Risk × Maternal Monitoring and Childhood Environmental Risk × Parental Limit-Setting interactions did not predict Mental Health at Age ~34 (*b*[SE] = -.001 [.00], *p* = .82 and *b* [SE] = -.002 [.01], *p* = .82, respectively). See Table 4.4 for full model results. However, Maternal Monitoring had a conditional direct effect on Mental Health, such that greater monitoring was associated with lower mental health symptoms (*b* [SE] = -.02 [.01], *p* = .01).

Predicting Health Limitations at Age 29

The Childhood Environmental Risk × Maternal Monitoring interaction did not predict the log-odds of being a structural zero in the dataset (e.g., absence of health limitations), or the log-count, of Health Limitations at Age 29 (*b* [SE] = -.04 [.03], *p* = .27 and *b* [SE] = -.02 [.02], *p* = .28, respectively). However, there was a significant Childhood Environmental Risk × Parental

Limit-Setting interaction to predict both the log-odds of being a structural zero in the dataset (i.e., reporting no health limitations; *b* [SE] = -.33 [.13], *p* = .01) and the log-count of Health Limitations at Age 29 (the number of health limitations; *b* [SE] = -.18 [.06], *p* = .001). See Table 4.5 for full model results.

We probed the interaction predicting the log-count of Health Limitations (simple slopes presented in Figure 4.2)[8]. There were positive associations between Childhood Environmental Risk and the log-count of Health Limitations at all levels of Parental Limit-Setting (i.e., +/- 1 *SD* from the mean and at the mean). However, the slopes became less steep at greater levels of Parental Limit-Setting. Specifically, among participants who reported low levels of Parental Limit-Setting (1 *SD* below the mean), the slope was *b* (SE) = .07 (.02), $p < .001$, meaning that for each one-unit increase in Childhood Risk, there was a 7% increase in the number of health limitations reported among these individuals (see Equation B). Among participants who reported high levels of Parental Limit-Setting (1 *SD* above the mean), the slope was *b* (SE) = .06 (.02), $p < .001$, meaning that for each one-unit increase in Childhood Risk, there was a 6% increase in the number of health limitations reported among these individuals (see Equation C).

$$\text{(B)} \quad e^{(.07)} = 1.07$$

$$\text{(C)} \quad e^{(.06)} = 1.06$$

Discussion

Using data from the NLSY97, we explored the role of parental vigilance (maternal monitoring and parental limit-setting) in the relation between childhood environmental risk and physical and emotional health in adulthood. There was evidence that greater childhood

[8] We did not probe the INFLATE interaction term (i.e., exploring the link between childhood risk and the likelihood of being a structural zero in the dataset at different levels of parental limit-setting) given that it is not a focus of the current study to explore predictors of the absence of health limitations in early adulthood.

environmental risk was associated with reporting physical health limitations at age 29 but was not significantly related to mental health symptoms at approximately age 34. Therefore, the long-lasting direct impacts of environmental risk were limited to physical health, and not emotional health, in young adulthood. These findings are surprising considering previous research that suggests childhood home and environmental risk factors often forecast poor emotional wellbeing across the lifespan (e.g., Allen et al., 2014; Ellen et al., 2001; Essex et al., 2011; Evans, 2003; Hill & Maimon, 2012). However, of note, the current study only captured a snapshot of participants' mental health in adulthood—although beyond the scope of the current study, it may be the case that risky environmental exposures are associated with declines in mental health over time (e.g., across several years of adulthood). Moreover, the measure of childhood environmental risk was captured during a snapshot of adolescence (e.g., at age 12, 13, or 14); significant main effects of childhood risk on mental health may have emerged if participants' physical environments were measured throughout multiple years of adolescence or earlier in childhood.

Our hypothesis that parental vigilance would offset associations among childhood environmental risk, physical, and mental health in adulthood was partially supported. Maternal monitoring and parental limit-setting did not influence the relation between childhood risk and mental health symptoms around age 34. However, there was a main effect of maternal monitoring on mental health symptoms, indicating that among all participants, those whose parents were more knowledgeable about their lives reported fewer mental health symptoms in adulthood. This finding suggests that maternal monitoring was beneficial for emotional health regardless of childhood environmental risk status, in contrast to previous studies that have found monitoring behaviors to be ineffective or even maladaptive for individuals from lower-risk

contexts (e.g., Garthe et al., 2018; Gonzales et al., 1996; Kliewer et al., 2016; McElhaney & Allen, 2001). Given that greater maternal monitoring forecasted better mental health among all participants in the current study, the potential benefits of these parenting behaviors might be broader than previously thought.

Although maternal monitoring did not influence the relation between childhood risk and physical health limitations, there was evidence that parental limit-setting served as a protective factor that buffered the relation between childhood risk and health limitations. Specifically, among participants who grew up in a higher-risk environment, those who also reported greater parental limit-setting throughout adolescence had fewer health limitations relative to those who reported lower levels of parental limit-setting. Although this buffering effect was small, it adds to previous research suggesting that parental vigilance in risky contexts might alleviate processes that often lead to poor health outcomes in adulthood (e.g., Bendezú et al., 2018; Kilgore et al., 2000; Laird et al., 2009; Pettit et al., 1999). Participants whose parents set greater limits on their social lives (e.g., who they were friends with and how late they stayed out at night) may have been less likely to engage in poor health behaviors and delinquent activities (e.g., cigarette smoking; excessive alcohol consumptions) that have negative long-term health consequences. It is important to note that economic stressors in these environments might hinder parents' ability to engage in these types of protective parenting practices (e.g., Racz & McMahon, 2011); therefore, parents' tendencies to control adolescents' daily activities (i.e., the kind of shows and movies they watched) might be indicative of vigilance towards other environmental factors that could be harmful for health.

We had predicted that among individuals who grew up in low-risk environments, greater levels of parental vigilance would be related to poorer health, or unrelated to health outcomes

altogether. Findings related to this prediction are nuanced given the positive effect of maternal monitoring on emotional health for all participants, regardless of childhood risk. However, in general, mental health symptoms and physical health limitations did not vary as a function of parental limit-setting for participants who grew up in less risky environments. Of note, the positive correlation between childhood risk and health limitations in the current study suggests that, on average, individuals who grew up in low-risk environments reported few or no health limitations at age 29; this floor effect may have limited the extent to which parental vigilance could be harmful or protective for these individuals.

The current study has several strengths. First, we leveraged a dataset from an ongoing longitudinal study with a large sample that was racially and socioeconomically representative of the United States. Therefore, our study was well-powered to detect small effects, and findings are generalizable to a diverse population. Second, assessments of childhood environments and perceptions of parental vigilance were collected throughout the participants' adolescence, and therefore these measures are not subject to recall biases introduced in retrospective reporting of childhood experiences. Third, although the measure of physical health limitations is from when participants were age 29 and thus before chronic health conditions typically set in and begin limiting an individual's physical capabilities, we used statistical techniques that overcame the limitation of a relatively healthy sample (i.e., by conducting zero-inflated regression models that allowed us to statistically account for an overdispersion of zeros). Nonetheless, it could be the case that findings will look different when participants are older and more likely to report health limitations due to normative physiological aging and the onset of chronic illnesses.

These notable strengths are met with a few limitations to consider. First, there were 20-33% missing cases in analyses, depending on the number of participants who were missing data

on the outcome measures (e.g., mental health symptoms and physical health limitations). However, the smallest sample size in any analysis was 3,254, and therefore all regression models remained well-powered. Second, the measures of physical and emotional health were self-reported and are thus subjective representations of participants' health. Nonetheless, self-reported health is often highly correlated with and predictive of objective measures of physical health (e.g., DeSalvo et al., 2006; Wu et al., 2013).

In addition, the measure of parental limit-setting in the current study was comprised of only three items. It will be important for future studies to include more comprehensive measures of this construct to disentangle the aspects of parental limit-setting that are protective for youth in risky contexts. For example, does limit-setting convey warmth and protection for children in riskier environments, but overprotection in lower-risk environments? We hypothesize that children's interpretations of their parents' monitoring and limit-setting behaviors play a role in the extent to which these vigilant characteristics are protective or maladaptive for health and wellbeing throughout the lifespan.

Conclusion

The current study adds meaningful information to the existing literature about the potential for parenting characteristics in childhood to offset associations between risky environments and negative health outcomes. Extending this line of work has the potential to determine aspects of parental vigilance that may be effective for reducing physical health limitations in young adulthood among individuals who grew up in high-risk settings. As such, we urge programs that aim to build resilience among youth in risky contexts to consider the role parental vigilance might play in preventing poor health later in life.

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Tables

Table 4.1
Round 1 (1997) sample characteristics among all participants, those with mental health data, and those with health limitations data.

	Total Sample (*n* = 4,829)	**Participants with Round 18 Mental Health Data (*n* = 3,663)**	**Participants with Age 29 Health Limitations Data (*n* = 4,378)**
Age	13.1 (*.79*)	33.9 (*.84*)	29 (*N/A*)
Sex			
Male	2,482 (51.4%)	1,778 (48.5%)	2,218 (50.7%)
Female	2,347 (48.6%)	1,885 (51.5%)	2,160 (49.3%)
Race/Ethnicity			
White, Non-Hispanic	2,535 (52.5%)	1,878 (51.3%)	2,249 (51.4%)
Black, Non-Hispanic	1,236 (25.6%)	985 (26.9%)	1,164 (26.6%)
Hispanic	1,010 (20.9%)	760 (20.7%)	918 (21.0%)
Mixed Race	48 (1.0%)	40 (1.1%)	47 (1.1%)
Sample Type			
Cross-Sectional	3,655 (75.7%)	2,746 (75.0%)	3,283 (75.0%)
Supplemental	1,174 (24.3%)	917 (25.0%)	1,095 (25.0%)

Note. Values are means (SD) or numbers (%).

Table 4.2
Correlations and descriptive statistics among study variables including in path models.

Variable	1	2	3	4	5	6	7	8	9
1. Childhood Environmental Risk (Round 1, 1997)	--								
2. Parental Monitoring (Round 1, 1997)	-.12**	--							
3. Parental Monitoring (Round 2, 1998)	-.07**	.46**	--						
4. Parental Monitoring (Round 3, 1999)	-.04*	.39**	.59**	--					
5. Limit-Setting (Round 1, 1997)	.02	.17**	.11**	.12**	--				
6. Limit-Setting (Round 2, 1998)	.03	.10**	.13**	.13**	.41**	--			
7. Limit-Setting (Round 3, 1999)	.07**	.07**	.12**	.16**	.36**	.50**	--		
8. Mental Health (Round 18, 2017)	.00	-.05**	-.05**	-.05**	-.04*	.00	.00	--	
9. Health Limitations (Age 29)	.08**	-.03	.00	-.01	-.02	.01	.02	.22**	--
n missing	568	184	688	784	1,340	1,639	1,702	1,166	451
Range	0-7	0-16	0-16	0-16	0-6	0-6	0-6	1-4	0-5
Mean (SD)	1.36 (1.43)	10.3 (3.26)	9.94 (3.22)	9.70 (3.38)	3.34 (1.52)	2.58 (1.43)	2.28 (1.39)	1.65 (.47)	.44 (1.03)

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

Table 4.3
Path model results for Zero-Inflated Negative Binomial regression predicting Health Limitations at Age 29 and multiple regression predicting Mental Health at Age ~34

	Health Limitations at Age 29					
	INFLATE Model		**COUNT Model**		**Mental Health in 2017**	
	***b* (SE)**	***p*-value**	***b* (SE)**	***p*-value**	***b* (SE)**	***p*-value**
Constant	.869 (1.15)	.448	1.47 (.81)	.070	**1.75 (.13)**	**< .001**
Age	-.066 (.09)	.440	-.101 (.06)	.096	.000 (.01)	.973
Sex	.231 (.14)	.098	**-.398 (.10)**	**< .001**	**-.113 (.02)**	**< .001**
Sample Type	.339 (.21)	.103	.017 (.15)	.908	-.012 (.02)	.615
Black, Non-Hispanic	.323 (.20)	.107	-.149 (.14)	.299	**-.126 (.02)**	**< .001**
Hispanic	.328 (.21)	.119	-.079 (.15)	.606	**-.069 (.02)**	**.005**
Mixed Race	.258 (.69)	.707	-.064 (.52)	.902	-.111 (.08)	.191
Childhood Environmental Risk	**-.142 (.05)**	**.009**	.065 (.04)	.068	.010 (.01)	.080

Note. For the Health Limitations model, 957 cases were omitted from the model due to missing data. For the Mental Health Model, 1,575 cases were omitted from the model due to missing data. INFLATE Model Results present the relation between the predictor and the log-odds of being an excessive "0" in the dataset. COUNT Model Results present the relation between the predictor and the change in the log count. For the Race/Ethnicity variables, White is the reference category. Bolded coefficients represent statistically significant effects.

Table 4.4
Moderation models predicting Mental Health at Age ~34 with: (a) Maternal Monitoring as the moderator and (b) Parent Limit-Setting as the moderator

	Maternal Monitoring		**Parental Limit-Setting**	
	***b* (SE)**	***p*-value**	***b* (SE)**	***p*-value**
Intercept	1.80 (.14)	.157	**1.75 (.14)**	**< .001**
Age	-.004 (.01)	.730	.000 (.01)	.995
Sex	**-.122 (.02)**	**< .001**	**-.114 (.02)**	**< .001**
Sample Type	-.014 (.02)	.562	-.012 (.02)	.620
Black, Non-Hispanic Dummy Code	**-.129 (.02)**	**< .001**	**-.126 (.02)**	**< .001**
Hispanic Dummy Code	**-.073 (.02)**	**.003**	**-.069 (.02)**	**.005**
Mixed Race Dummy Code	-.115 (.08)	.173	-.110 (.08)	.193
Childhood Environmental Risk	.008 (.01)	.157	.010 (.01)	.080
Maternal Monitoring	**-.021 (.01)**	**.005**	--	--
Parental Limit-Setting	--	--	.007 (.02)	.71
Childhood Environmental Risk × Maternal Monitoring	-.001 (.00)	.824	--	--
Childhood Environmental Risk × Parental Limit-Setting	--	--	-.002 (.01)	.820

Note. 1,575 cases were omitted from the model due to missing data. For the Race/Ethnicity variables, White is the reference category. Bolded coefficients represent statistically significant effects.

Table 4.5
Zero-Inflated Negative Binomial moderation models predicting Health Limitations at Age 29 with: (a) Maternal Monitoring as the moderator and (b) Parent Limit-Setting as the moderator

	Maternal Monitoring				**Parent Limit-Setting**			
	INFLATE Model Results		**COUNT Model Results**		**INFLATE Model Results**		**COUNT Model Results**	
	***b* (SE)**	***p*-value**	***b* (SE)**	***p*-value**	***b* (SE)**	***p*-value**	***b* (SE)**	***p*-value**
Constant	.798 (1.14)	.485	1.50 (.81)	.062	.301 (1.30)	.817	**1.11 (.85)**	**.192**
Age	-.064 (.09)	.459	-.105 (.06)	.084	-.031 (.09)	.735	-.079 (.06)	.206
Sex	.261 (.14)	.063	**-.392 (.10)**	**< .001**	.259 (.15)	.079	**-.384 (.10)**	**< .001**
Sample Type	.362 (.21)	.087	.031 (.15)	.834	.423 (.23)	.071	.062 (.15)	.672
Black, Non-Hispanic Dummy Code	.346 (.20)	.089	-.137 (15)	.346	.330 (.21)	.121	-.150 (.15)	.307
Hispanic Dummy Code	.301 (.21)	.158	-.105 (.16)	.496	.335 (.22)	.128	-.077 (.16)	.619
Mixed Race Dummy Code	.287 (.68)	.673	-.056 (.52)	.914	.162 (.73)	.824	-.143 (.53)	.786
Childhood Environmental Risk	**-.139 (.06)**	**.011**	.061 (.04)	.090	**-.183 (.07)**	**.008**	.054 (.04)	.131
Maternal Monitoring	**.142 (.07)**	**.044**	.056 (.05)	.242	--	--	--	--
Parental Limit-Setting	--	--	--	--	**.408 (.18)**	**.025**	**.244 (.12)**	**.041**
Childhood Environmental Risk × Maternal Monitoring	-.037 (.03)	.273	-.023 (.02)	.282	--	--	--	--
Childhood Environmental Risk × Parental Limit-Setting	**--**	**--**	--	--	**-.327 (.13)**	**.012**	**-.188 (.06)**	**.001**

Note. 957 cases were omitted from the model due to missing data. INFLATE Model Results present the relation between the predictor and the log-odds of being an excessive "0" in the dataset. COUNT Model Results present the relation between the predictor and the change in the log count. For the Race/Ethnicity variables, White is the reference category. Bolded coefficients represent statistically significant effects

Figures

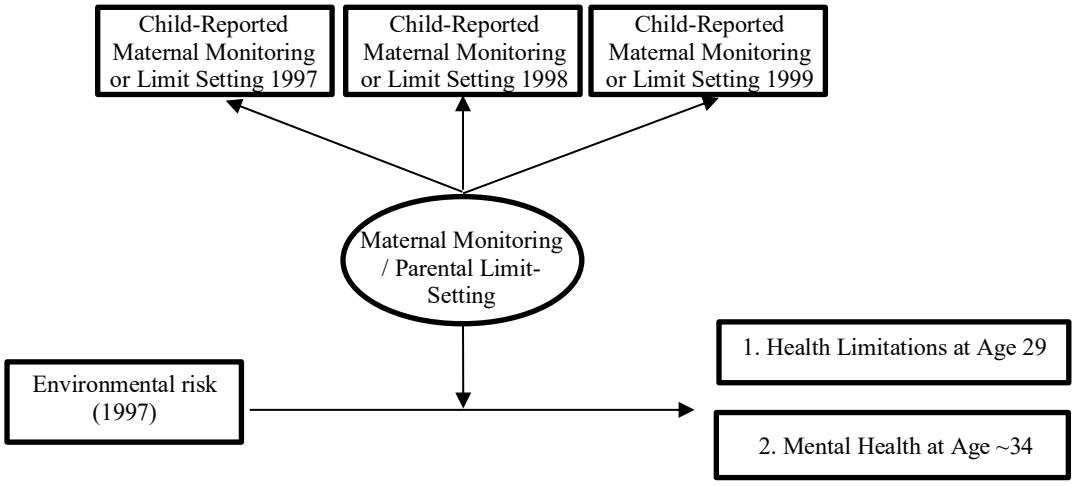

Figure 4.1. Conceptual model for moderation analyses.

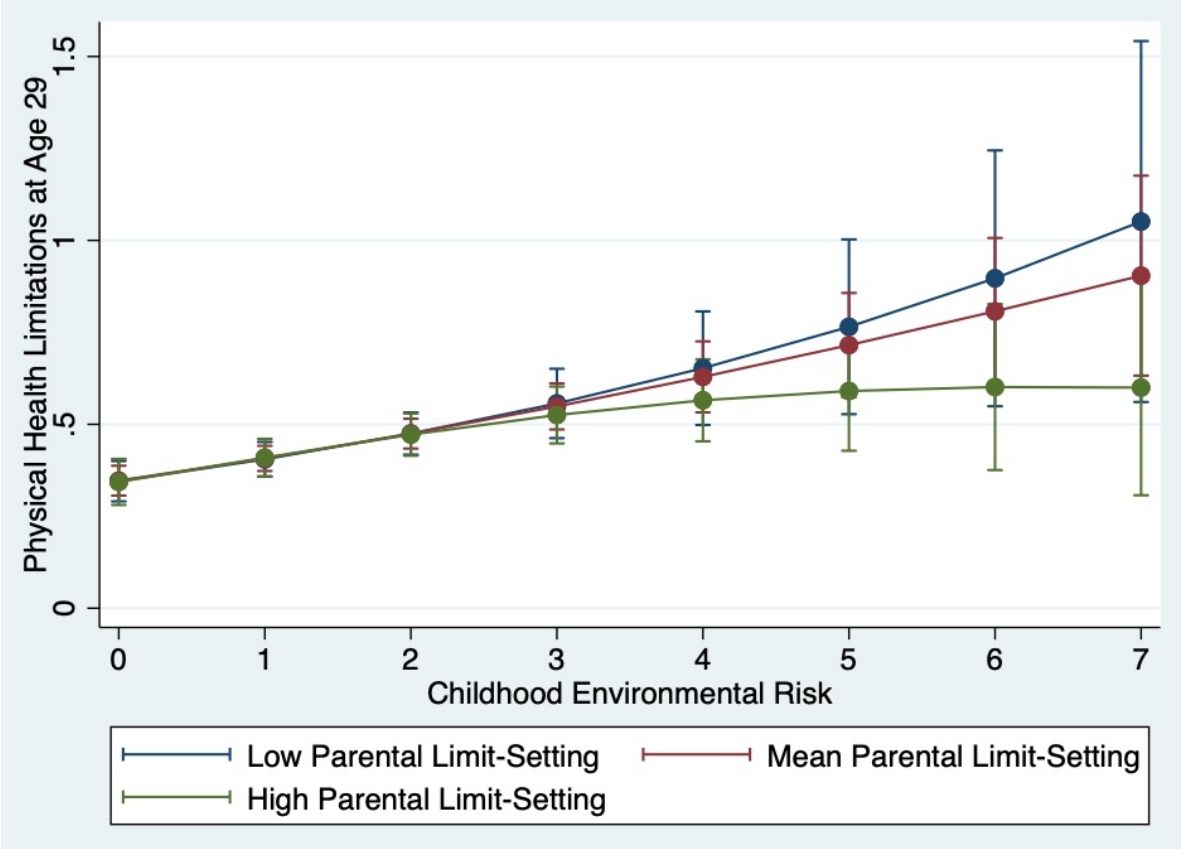

Figure 4.2. Simple slopes from the Childhood Environmental Risk × Parental Limit-Setting interaction to predict the log-count of Health Limitations at Age 29.

CHAPTER 5

GENERAL DISCUSSION

The three studies presented in this dissertation advance our understanding of the ways in which vigilant parenting reduces negative health outcomes in the context of childhood risk and promotes positive development throughout generations of individuals in risky settings. Hypotheses were generally supported throughout the three studies. First, we had hypothesized that parental vigilance would moderate the association between childhood risk and health outcomes in Studies 1 and 3, such that parental vigilance would be related to better health among individuals from higher-risk contexts and be maladaptive for (or unrelated to) health for individuals from lower-risk contexts. Additionally, in Study 2, we hypothesized that grandparents' parental vigilance would be associated with their grandchildren's psychosocial adjustment outcomes, particularly among children whose families experienced greater socioeconomic risk.

Summary of Findings

In the first study, parental control moderated the association between recalled neighborhood risk throughout childhood and cytokine-mediated cold susceptibility in adulthood. In this study, there surprisingly was not evidence of a direct, main effect of neighborhood risk on the likelihood of being diagnosed with a cold. However, nonetheless, there was a negative association between neighborhood risk and cold susceptibility at high levels of recalled parental control (and no relation between neighborhood risk and cold susceptibility at lower levels of recalled parental control). These findings suggest that the relation between parental vigilance and

cold susceptibility later in life was context-dependent, such that controlling parenting behaviors were beneficial for individuals from high-risk homes, but detrimental for those from lower-risk homes.

In the second study, there was evidence that parents' knowledge of their children in adolescence benefitted the subsequent generation's psychosocial adjustment outcomes. These findings are in the context of families in high-risk environments (i.e., African American families who live in the rural South). Interestingly, the promotive effect of parental knowledge was only beneficial for depressive symptoms and anxiety among children whose parents experienced greater socioeconomic risk throughout their adolescence. However, the relation between parental knowledge and children's risk-taking behaviors and poor self-control did not vary as a function of socioeconomic risk. In other words, the promotive effect of parental knowledge on these outcomes (risk-taking and self-control) was the same for all children, regardless of their families' socioeconomic risk.

Finally, in the third study, higher levels of childhood environmental risk were associated with a greater number of self-reported physical health limitations—but not mental health symptoms—in adulthood. Parental limit-setting, but not maternal monitoring, buffered the association between risk and physical health limitations. In contrast, there was no evidence that the link between risk and mental health symptoms varied at different levels of parental vigilance (maternal monitoring or parental limit-setting). However, there was evidence that maternal monitoring was directly associated with mental health, such that greater levels of monitoring in adolescence predicted fewer mental health symptoms in young adulthood, regardless of childhood risk.

In all three studies, parental vigilance served as a protective or promotive factor for children in risky contexts, or adults who grew up in risky contexts. In this sense, parental vigilance, or parents' efforts to control (as in Study 1), monitor (Study 2), and set limits (Study 3) on children's lives prevent negative health consequences often associated with childhood adversity. Although speculative at this point, it might be the case that children's and adolescents' environments inform their interpretations of these parenting behaviors (e.g., Omer et al., 2013; Soenens et al., 2015) – parents who control, monitor, and surveille children in high-risk environments, despite experiencing their own social or financial stressors (e.g., racial discrimination; navigating available resources; working multiple jobs), are likely attentive to and responsive about their children's needs and wellbeing. As a result, these children are potentially shielded from harsh consequences of an adverse environment.

On the other hand, regarding findings from the three studies related to individuals raised in lower-risk contexts, Study 1 is in line with previous studies that have found parental vigilance to be maladaptive for individuals from lower-risk contexts (e.g., Alloy et al., 2006; Laird et al., 2018; McElhaney & Allen, 2001), whereas findings from Studies 2 and 3 support previous studies that have found parental vigilance to be unrelated to children's outcomes (e.g., Stattin & Kerr, 2000; Kerr & Stattin, 2000). To elaborate, in Study 1, findings suggested that greater parental control was related to poorer health (i.e., a greater likelihood of getting sick with the cold) among individuals from low-risk neighborhoods. As such, this study highlights how, in the absence of environmental risk, controlling parenting practices may set physiological mechanisms in motion that have negative long-term physical health consequences.

In Studies 2 and 3, parental vigilance was, in part, unrelated to adjustment and health outcomes for individuals from lower-risk settings. Specifically, in Study 2, grandparents'

parental knowledge was unrelated to depressive symptoms and anxiety among grandchildren whose parents experienced low socioeconomic hardship in adolescence. Additionally, in Study 3, parental limit-setting did not modify the association between childhood environmental risk and physical health limitations in adulthood for participants who grew up in lower-risk environments, suggesting that parental limit-setting was neither good nor bad for these individuals. It is worthwhile to note the distinction between the measure of parental vigilance used in Study 2 (i.e., parental knowledge) versus those used in Studies 1 and 3 (i.e., parental control and limit-setting) – although all these measures are tapping into parents' efforts to be involved in their children's lives, behaviors to control or limit children's autonomy are not encompassed in parental knowledge. Therefore, there may not have been maladaptive effects of parental knowledge on children's emotional health in Study 2 because this aspect of parental vigilance may not be interpreted as negatively as control and limit-setting behaviors in lower-risk settings. Notably, the main effect of parental monitoring on mental health among all individuals (even those from lower-risk environments) in Study 3 supports this notion.

In addition, physical health in Study 3 was assessed when participants were in young adulthood and still generally healthy. It may be the case that maladaptive effects of parental limit-setting on health emerge later in life when chronic conditions are more prevalent. Moreover, although findings from Study 3 contrast with findings from Study 1 (such that among individuals from lower-risk settings, parental control was related to poorer health in Study 1, but parental limit-setting was unrelated to physical health in Study 3), it is important to note that the metrics of physical health were quite different in these two studies. In Study 1, physical health was measured objectively in a quasi-experimental design, whereas in Study 3, poorer health relied on participants self-reporting chronic physical limitations. Therefore, findings might have

looked more similar across the two studies (i.e., there may have been maladaptive effects of parental limit-setting for participants from lower-risk backgrounds) if the measures of physical health were more aligned.

Strengths and Limitations

Each of the three studies had its own unique strengths and limitations. The first study leveraged a quasi-experimental viral challenge study design, and therefore the physical health outcomes (i.e., proinflammatory cytokine production and cold susceptibility) were measured in a controlled setting. However, parental control and neighborhood risk in childhood were retrospectively reported and could be affected by memory recall bias (although, of note, controlling for current levels of stress and trait-level characteristics did not change study findings).

Recall bias was not an issue in Studies 2 and 3, however, because they leveraged longitudinal study designs. In Study 2, the sample size of third-generation youth was relatively small, but despite the sample size limitation, the associations among grandparents' parental knowledge and children's outcomes were consistent across the four domains of adjustment measured in the study (depressive symptoms, anxiety, risky behaviors, and poor self-control). As such, even in the absence of significant second-generation mediation effects, evidence for intergenerational transmission was robust.

The sample size in Study 3 was large and nationally representative, and thus findings could be generalized to a diverse population. Moreover, using advance statistical techniques allowed for identifying associations among childhood risk, parental vigilance, and physical health limitations in young adulthood, even though participants were still relatively healthy. However, it is possible that findings may look different when participants are in middle or late

adulthood, when they are more likely to have developed chronic health condition. Nonetheless, the large sample size (*n*s in analyses over 3,000) and advanced statistical techniques allowed for detection of small effects.

Across all three studies, driving factors that may explain *why* parental vigilance was beneficial, maladaptive, or unrelated to physical and mental health are yet to be empirically assessed. We speculate that children's and adolescents' environments inform their interpretations of parents' vigilant behaviors and influence whether parental vigilance serves to promote or undermine physical and psychological wellbeing. To elaborate, when there are real threats in a child's environment (e.g., exposure to domestic violence; access to illicit drugs and alcohol), parents' efforts to control children and shield them from these threats come from a place of responsiveness and concern. To a certain extent, these efforts to shield and protect children from environmental stressors likely alleviate maladaptive physiological alterations (e.g., to their stress-response and immune systems), which may prevent negative long-term health consequences (McEwen, 1998a; McEwen, 1998b; Miller et al., 2011). Conversely, in low-risk settings where environmental threats are low, parents' attempts to control their children are likely interpreted as unreasonable or overbearing, and therefore may forecast high levels of stress and detriments to psychological wellbeing. In turn, elevated stress and poor emotional wellbeing may directly affect health by disrupting physiological set-points or lead to engaging in unhealthy coping behaviors (disordered eating habits; substance use)—both of which cause chronic health conditions when sustained.

Future Directions

To substantiate theories that may explain the role parental vigilance plays in the association between childhood environments and health, future studies will need to assess

parental vigilance, children's interpretations of parental vigilance, and indices of short- and long-term physical and emotional health. In addition, the studies presented in this dissertation used data from projects in which data collection has concluded (Study 1) or began decades ago (Studies 2 and 3), and therefore measures of parental vigilance were limited. As such, future research will ideally be intentional about developing and administering questionnaires or qualitative interviews that effectively combine different aspects of parental vigilance (e.g., monitoring, knowledge, control, limit-setting, etc.) into one construct that can be used with populations in diverse settings (i.e., appropriately assesses vigilance regardless of context).

Additional considerations for future studies are to address how environmental characteristics and psychosocial factors (e.g., personality traits; depressive symptoms; social relationships) drive parents to be vigilant towards their children—and further, how these factors relate to health outcomes. For example, what are the implications for children's health when vigilant parenting behaviors are a natural response to raising children in high-risk environments (e.g., Pinderhughes et al., 2007)? Alternatively, are there intrinsic or social characteristics (e.g., having a supportive co-parent) that predict vigilant parenting despite challenging situations, and therefore explain the compensatory nature of parental vigilance? Conversely, are parents who practice greater vigilance those who are also high in trail-level anxiety or neuroticism (which have direct implications for children's wellbeing, e.g., Ellenbogen & Hodgins, 2004) or experiencing a lack of control in other areas of their life (e.g., in their marriage or careers)? These factors are of interest for examining intergenerational transmission of parental vigilance; particularly, to identify how intrinsic characteristics or changes in families' environments explain the link between first-generation parental vigilance and the third generation's outcomes.

There are certainly still unknowns in this line of work, and future research is warranted to extend conclusions about mechanisms that explain why long-term health consequences associated with parental vigilance are context dependent. Nonetheless, the three studies presented in this dissertation highlight that encouraging parents to be vigilant may be a viable pathway for promoting physical and mental health among children and adolescents in high-risk contexts. Programs can leverage findings from these studies (and others that champion parental vigilance as a protective and compensatory factor in risky contexts) to promote high-quality parental responsiveness, and in return, positive youth outcomes.

Additionally, empirical evidence showing that high levels of parental vigilance is sometimes maladaptive in lower-risk contexts should be communicated to broad audiences for the purpose of alerting parents that certain aspects of vigilant parenting behaviors may be harmful for their children's wellbeing (specifically, parental control). For example, this research can be translated in op-eds or blog posts that inform parents about how tendencies to control, monitor, and supervise children have long-term implications for mental and physical health—for better or for worse.

One final note to highlight is that this research is acutely relevant in the context of the social climate created by the COVID-19 pandemic, when children may be spending more time at home than at school. With parents and children in close quarters more often now than prior to 2020, it is imperative for parents to be cautious of tendencies to control children when environmental threats are low. However, in settings where environmental threats are high, parental vigilance may be important to reduce the risk for poor health trajectories, which may currently be amplified due to pandemic-related stressors.

Conclusion

The three studies presented in this dissertation emphasize the need for deeper analysis into the mechanisms by which parental vigilance informs long-lasting ties between childhood environments and health. The next steps in this line of work include assessing children's interpretations of parental vigilance (in high- versus low-risk contexts); incorporating systematic assessments that accurately capture different aspects of parental vigilance regardless of environmental context; and finally, exploring the extent to which environments, personality traits, and social situations inform vigilant parenting behaviors, and whether these processes in turn influence the relation between childhood environments and health. Although there is certainly not a single definition of "good" parenting, takeaways from this dissertation and next steps increase broader understanding of what constitutes high-quality, responsive parenting in different settings—a topic with public health relevance, particularly in the era of the COVID-19 pandemic.

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