EARLY ENVIRONMENTS, PARENTAL OVERPROTECTION, AND SUSCEPTIBILITY TO THE COMMON COLD

by

KELSEY L. CORALLO

(Under the Direction of Katherine B. Ehrlich)

ABSTRACT

Childhood stressors influence mental and physical health outcomes across the lifespan. Researchers have identified various mechanisms that explain these associations, such as increased engagement in poor health behaviors and biological embedding. However, many people who experience childhood adversity remain healthy in adulthood due to numerous protective factors. The current study examined whether parental overprotection throughout childhood influenced the associations among recalled neighborhood environments, cold susceptibility, and trait-level depression in adulthood. Results indicated that for individuals who reported greater perceived parental overprotection, neighborhood risk was suggestive for being negatively associated with the probability of developing a clinical cold and nasal proinflammatory cytokine production. Further, for those who reported lower levels of parental overprotection, neighborhood risk was positively associated with mucus production and depression scores. Study findings suggest that perceived parental overprotection throughout childhood may benefit or be a detriment to adult physical and mental health depending on recalled neighborhood environments.

INDEX WORDS: Social determinants of health, early life adversity, cold susceptibility

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KELSEY L. CORALLO

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KELSEY L. CORALLO

Major Professor:	Katherine B. Ehrlich
Committee:	Michelle vanDellen
	Anne Shaffer

Electronic Version Approved:

Ron Walcott
Interim Dean of the Graduate School
The University of Georgia
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CHAPTER 1

INTRODUCTION

Childhood experiences have lasting influences on physical and psychological health, even after controlling for subsequent life stressors, social experiences, and health behaviors (Cicchetti & Toth, 2005; Hostinar et al., 2015; Miller, Chen, & Parker, 2011). In particular, childhood adversity (e.g., poverty, maltreatment, chaotic home environments, poor quality family relationships) is associated with poor health trajectories across the lifespan (e.g., Ehrlich, Miller, & Chen, 2016; Repetti, Taylor, & Seeman, 2002). However, despite decades of research that links early adversity to detrimental outcomes, there is evidence that many individuals reared in stressful environments remain healthy and resilient to physical and psychological illness (Luthar, Cicchetti, & Becker, 2000; Masten, 2007). Therefore, researchers have worked to disentangle the social, environmental, and biological factors that might buffer or exacerbate the relation between childhood stress and health outcomes later in life. The current study seeks to examine the associations among recalled early life environments (measured as perceived level of risk in neighborhood environments throughout childhood), parental overprotection, susceptibility to the common cold, and trait-level depression.

Social Determinants of Health and Biological Embedding of Early Life Adversity

Findings associated with the study of social determinants of health (i.e., research that explores the associations among social experiences and physical health) suggest that adverse experiences in childhood are particularly powerful predictors of physical health later in life. Felitti and colleagues' landmark Adverse Child Experiences (ACE) study found that people who experienced four or more adverse childhood experiences (e.g., physical or sexual abuse, parental

separation or divorce, domestic violence, etc.) were at a greater risk for several poor health outcomes later in life (e.g., cardiovascular and lung disease, cancer, etc.), and were also more likely to engage in poor health behaviors (Felitti et al., 1998). The decades of research that followed have begun to uncover the mechanisms by which stressful experiences in early life influence future physical health trajectories.

One empirically-supported theory that explains the link between childhood stress and poor health outcomes is the biological embedding model of early life adversity (Miller et al., 2011). This model posits that chronic stressors in childhood sensitize the body's stress-response and immune systems to adapt to stressful experiences in such a way that, over time, compromises one's ability to fight off infection and disease. To elaborate, the body undergoes maladaptive changes such as hormone dysregulation, epigenetic modifications, and nonresolving cellular inflammation, all of which program the body to remain in a state of chronic inflammation. These changes result in people having a higher likelihood of developing chronic diseases of aging, such as cardiovascular disease, stroke, diabetes, autoimmune disease, and some cancers (Miller et al., 2011).

In addition to cellular programming in childhood and adolescence, early life stress is linked to an increased prevalence of developing unhealthy lifestyles and engaging in risky health behaviors (Felitti et al., 1998; Miller et al., 2011; Power et al., 2005). For example, poor quality family relationships in early life have been linked to an increased risk for obesity in young adulthood (e.g., Lissau & Sorensen, 1994), and youth who are reared in high-risk environments are more likely to smoke cigarettes (e.g., Doherty & Allen, 1994; Fisher & Feldman, 1998) and abuse drugs and alcohol later in life (e.g., Barnes et al., 2000; Mounts & Steinberg, 1995), as well as engage in risky sexual behaviors in adolescence (e.g., Scaramella, 1998). The

aforementioned health conditions and behaviors serve as direct pathways to chronic disease in adulthood. For example, obesity and alcohol abuse are linked directly to the development of many chronic diseases, such as diabetes and cardiovascular disease (World Health Organization, 2019). Additionally, certain health behaviors might serve as mediators of the link between childhood adversity and poor health in adulthood, such that being raised in high-risk environments in childhood may give rise to a greater likelihood of engaging in risky health behaviors in adulthood (e.g., drug and alcohol abuse, cigarette smoking), which are related to poor physical health trajectories across the lifespan (e.g., Phelan, Link, & Tehranifar, 2010; Stimpson et al., 2007).

Risky Neighborhood Environments

Stressful neighborhood environments in childhood may be related to developing acute and chronic illness later in life (e.g., Chen & Paterson, 2006; Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Ellen, Mijanovich, & Dillman, 2001; Janicki-Deverts et al., 2014; Jutte, Miller, & Erickson, 2015; Leventhal & Brooks-Gunn, 2000). Research has shown that residing in risky neighborhood environments – characterized by poor-quality physical and social characteristics such as uncleanliness, pollution, high traffic volume, noisiness, limited access to public services and amenities, and compromised safety due to high rates of crime and violence – put people at risk for poor physical and mental health outcomes (e.g., Buschmann, Prochaska, Cutchin, & Peek, 2018; Cohen et al., 2010; Diez Roux & Mair, 2010; Schulz et al., 2012). For example, perceived neighborhood quality has been found to be negatively associated with allostatic load (a measure of the body's physiological "wear and tear;" Buschmann et al., 2018) and neighborhood poverty has been found to be positively associated with allostatic load (Brody, Lei, Chen, & Miller, 2014; Schulz et al., 2012). In addition, one study found an association between

neighborhood context (i.e., high vs. low SES neighborhoods) and perceived levels of stress and depression (Elliott, 2000), and a review found that in 11 out of 14 studies, neighborhood characteristics were associated with depressive symptoms (Blair, Ross, Gariepy, & Schmitz, 2013). Further, findings suggest that childhood neighborhood environments have lasting effects on health even after controlling for socioeconomic status in adulthood (Cohen et al., 2010).

Neighborhood environments reflect an individual's socioeconomic status as well as the availability of social resources and exposure to environmental stressors (Cohen et al., 2010; Ellen et al., 2001). Those who live in under-resourced and disadvantaged neighborhoods are more likely to experience poor social capital (i.e., quality of relationship networks), which is related to an increased risk for various physical and mental health outcomes, such as poor self-reported health (Kawachi, Kennedy, & Glass, 1999), higher mortality rates (Lochner, Kawachi, Brennan, & Buka, 2003), and an increased prevalence of mental illness (McKenzie, Whitley, & Weich, 2002). Further, residing in risky neighborhoods is associated with increased familial conflict due to chronic instability and financial burdens. This emotional strain, in turn, is linked to the development of emotional and behavioral problems in childhood, as well as mental health disorders and chronic diseases later in life (Leventhal & Brooks-Gunn, 2000; Plybon & Kliewer, 2002; Repetti et al., 2002). In addition, exposure to crime and violence is a stressor that uniquely predicts both mental and physical health outcomes. For example, exposure to neighborhood violence in childhood has been linked to asthma morbidity (Wright et al., 2004), and levels of neighborhood crime and violence are associated with a greater likelihood of engaging in poor health behaviors (Ellen et al., 2001).

Parenting as a Protective Factor in High-Risk Neighborhood Contexts

Given that raising children in high-risk neighborhood contexts is robustly associated with poor physical and emotional well-being, it is important to understand various protective factors that can buffer children from experiencing negative consequences of early life adversity. Parental monitoring and supervision emerge as notable protective factors associated with child development in adverse neighborhood environments (Sullivan, Kung, & Farrell, 2004). Parents raising children in high-risk environments sometimes develop vigilant parenting styles, marked by close monitoring of children's whereabouts and relationships in an effort to prevent youth from making choices that might be detrimental to their health and well-being (Omer, Satran, & Driter, 2016). Indeed, parental discipline and control are thought to emerge at least in part as a result of neighborhood characteristics (Pinderhughes et al., 2001). Although well-intentioned, this type of parenting is sometimes interpreted as harsh, authoritarian, and a threat to the child's autonomy (Baumrind, 1966; Gray & Steinberg, 1999).

The parenting literature suggests that autonomy-supportive parenting promotes positive child development, whereas excessive protection (i.e., overprotection) and monitoring are harmful to child development and well-being (e.g., LeMoyne & Buchanan, 2011; Reed et al., 2016; Soenens, Vansteenkiste, & Van Petegem, 2015). To support these claims, parental autonomy support (i.e., parenting behaviors that promote children's autonomous goals and choices) has been found to be positively related child executive function skills (Bernier, Carlson, & Whipple, 2010), prosocial behavior engagement (Gagne, 2003), and adolescent emotion regulation (Brenning et al., 2015). Conversely, parental overprotection (i.e., the tendency for parents to be hypervigilant towards children's whereabouts and choices) has been found to be associated with heightened academic stress in a sample of medical students (Kang, Lee, & Youn,

2017), greater anxiety and emotional vulnerability in child populations (Thomasgard, 1998; Thomasgard, Metz, Edelbrock, & Shonkoff, 1997; Thomasgard, Shonkoff, Metz, & Edelbrock, 1995), and poorer psychological well-being in college students (LeMoyne & Buchanan, 2011).

Although some researchers adopt a perspective that autonomy-promoting parenting behaviors are uniformly beneficial for youth development, whereas controlling parenting behaviors are maladaptive for children, others embrace a relativistic view of parenting practices, such that the quality of parenting should be evaluated in the context of environmental and cultural factors (Soenens et al., 2015). That is, greater parental monitoring and involvement promote positive child behaviors and outcomes in particular socioeconomic and cultural contexts and environments (Grusec, 2008; Soenens et al., 2015). Child temperament (e.g., level of agreeableness), and the social, cultural, and economic environment where children reside might influence the extent to which a child perceives parental monitoring and control to be excessive (Omer et al., 2016; Soenens et al., 2015). In turn, children's physical and mental health may vary with regard to their perceptions of parental monitoring. In certain socioeconomic and cultural contexts, children typically do not interpret excessive parental monitoring and control as threats to autonomy, but rather as signs of warmth and protection (Soenens et al., 2015). Thus, it is possible that protective, or even *overprotective* parenting might be beneficial for children who grow up in high-risk neighborhood environments.

Several studies have explored child outcomes associated with parenting styles in diverse socioeconomic and cultural contexts, and findings have suggested that protective parenting can be advantageous for at-risk youth (e.g., Brody & Flor, 1998; Garmezy, 1985; Murry & Brody, 1999; Varner et al, 2017). For example, Brody and Flor (1998) found that in a sample of single-parent households (82% of whom lived in poverty), maternal "no nonsense" parenting,

characterized by high levels of parental control, was positively associated with children's cognitive and social competence and negatively associated with children's internalizing problems. Additionally, in a study with African American families, involved-vigilant parenting (characterized by greater levels of monitoring and behavioral control) was positively associated with children's psychological well-being and academic performance trajectories (Varner et al., 2017). These findings suggest that parenting practices characterized by greater vigilance toward and greater protection and monitoring of children are associated with more favorable child outcomes in high-risk contexts.

The concept of parental overprotection connotes the idea of overly-controlling, autonomy-abating parenting practices, but in risky neighborhoods, greater levels of parental control could attenuate adverse health consequences associated with riskier environments. For example, in high-risk neighborhoods characterized by physically or socially hazardous conditions (e.g., unsafe street conditions, interpersonal violence), high parental control can serve to instill vigilance to threat and danger in offspring, which may prevent children from engaging in harmful behaviors and activities, such as substance abuse (Willis, 1992). Children who diverge from unhealthy and harmful behaviors often associated with risky neighborhoods might be more likely to tend to their physical health and well-being, and thus be at reduced risk for developing acute and chronic illnesses. Additionally, parents of children in high-risk environments are likely to experience economic stressors such as job insecurity and difficulty paying bills, and the demands associated with providing for their family may take priority over caregiving; thus, parents might find it difficult to be proactive about their children's needs. However, parents who engage in overprotective or hypervigilant parenting practices monitor and address their children's whereabouts and needs. Therefore, parental overprotection could

possibly buffer children from long-term maladaptive effects of childhood stress, and as such, might prevent the onset and severity of acute and chronic illness.

The Present Study

Hypervigilant, no-nonsense parenting in high-risk contexts is associated with better youth psychosocial outcomes (e.g., Brody & Flor, 1998; Varner et al., 2017), and it may be that overprotective parenting serves to prevent at-risk youth from developing negative health consequences associated with childhood adversity. The current study examined whether overprotective parenting in the context of risky neighborhood environments was associated with physical and mental health outcomes later in life. Many studies have found an association between childhood stressors and susceptibility to acute illnesses, such as the common cold (e.g., Cohen et al., 2004; Cohen et al., 2013; Janicki-Deverts et al., 2014; Murphy, Cohen, Janicki-Deverts, & Doyle, 2017). Therefore, we examined whether perceived parental overprotection during childhood moderated the associations among recalled neighborhood environments in childhood and susceptibility to the common cold and proinflammatory cytokine production in an adult population. In addition, given that previous research has evidenced a link between parenting behaviors and mental health outcomes (e.g., depressive symptoms; Dallaire et al., 2006) we also explored whether overprotective parenting moderated the association between childhood environments and trait-level depression in adulthood.

We predicted that for adults who reported greater levels of perceived parental overprotection throughout childhood, neighborhood risk would be negatively associated with cold susceptibility, markers of illness, and trait-level depression. This study was conducted using publicly available data from The Common Cold Project (a publicly available dataset described in

more detail below), and data were drawn from the Pittsburgh Cold Study 3. The data are available online at www.commoncoldproject.com.

CHAPTER 2

METHOD

Participants

Participants were 213 adults ages 18 to 55 recruited from metropolitan Pittsburgh, Pennsylvania between 2007 and 2011 via newspaper advertisements. To participate, individuals were required to be in good health as per medical history and physician examination; exclusion criteria included history of psychiatric illness, chronic physical illness, egg allergies, and history of certain types of ear, nose, and throat surgeries (Cohen et al., 2013). Additionally, participants were excluded if they had abnormal urinalysis or blood test results, were pregnant or lactating, taking regular medication, seropositive for HIV, or if they had evidence of pre-challenge immunity to the virus. Participants were also excluded if they had a cold or flu-like illness within 30 days prior to the study. Each participant received $1,060 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 signed informed consent.

Procedure

Participants completed pretrial questionnaires assessing demographic information, social and psychological constructs, and health behaviors, and they were assessed for baseline biological measures such as height, weight, body mass index, and blood pressure. Two to three days prior to the study, participants provided blood samples to assess baseline immunity (pre-challenge antibody titer) to rhinovirus 39 (RV39). Participants were subsequently quarantined in a hotel room for six days. On the first day, they were exposed to the virus via nasal drops containing RV39 and were monitored for the following five days to assess objective and

subjective symptoms and criteria of illness. Quarantining procedures required that participants stay on their assigned floor of the hotel and have limited interaction with other study participants. They were instructed to stay at least 3 feet away from each other and were not permitted to enter each other's rooms in an effort to avoid transfer of illness. They were not allowed to have visitors outside of the study. Participants had a follow-up visit approximately 28 days after initial viral exposure in order to evaluate post-challenge antibody levels and determine clinical cold diagnosis.

Measures

Early neighborhood risk. The Places You've Lived Interview (PLI; Janicki-Deverts et al., 2014) is a 13-item questionnaire about neighborhood safety (three items, Cronbach's α = 0.64-0.67; e.g., "Was your street considered safe"), social environment (four items, Cronbach's α = 0.71-0.76; e.g., "Did adult neighbors watch out for you?"), and physical environment (six items, Cronbach's α = 0.76-0.82; e.g., "Was the condition of the street very poor?"). Participants completed items in reference to their neighborhoods at age 5, 10, and 15. The average neighborhood safety, social environment, and physical environment scores across the three age assessments were standardized into z-scores, and the mean of the standardized scores served as a composite for early neighborhood environment risk, with higher scores indicating riskier childhood neighborhood environments.

Parental overprotection. The Overprotection Subscale of the Parental Bonding Instrument (PBI; Parker, Tupling, & Brown, 1979) assessed the level of perceived parental overprotection throughout the first 15 years of life. The scale included six items, such as "my parents were overprotective of me," and "my parents tended to baby me." Higher scores indicated more perceived parental overprotection throughout childhood (Cronbach's α = 0.68).

Cold susceptibility. Researchers evaluated clinical cold diagnosis based on objective symptoms throughout quarantine and antibody production both throughout quarantine and at a 28-day follow-up visit. Participants met objective criteria for cold diagnosis if they were both infected with the virus (i.e., if there was recovery of the challenge virus in nasal secretions on any of the post-challenge quarantine days, or at least a 4-fold increase in antibody titers from pre-challenge to 28-day post-challenge) and met objective symptom criteria (further details provided below; Cohen et al., 1997).

Objective symptom criteria. Total baseline-adjusted mucus weight (measured by having participants use pre-weighed tissues to collect their mucus secretions; Doyle et al., 1992) of at least 10 g, and average nasal mucociliary clearance time (determined by measuring the amount of time it takes for 20 μL of dye solution placed in the participant's nasal turbinate to produce a taste in his/her mouth; Doyle & van Cauwenberge, 1987) of 7 min or longer were included as criteria for objective cold diagnosis. At the end of each day, the bags with tissues containing nasal secretions were weighed, and the total secretion weight was determined by subtracting the bag and tissue weight from the total weight. Total adjusted post-challenge mucus weight and average adjusted post-challenge nasal clearance time were used to determine clinical cold diagnosis and were also assessed as symptom severity among those who were infected with the challenge virus.

Nasal inflammatory markers. A composite of the nasal proinflammatory cytokines interleukin-6 (IL-6), interleukin-1β (IL-1β), and tumor necrosis factor alpha (TNF-α) was included as an outcome variable among all participants, as proinflammatory cytokine production has been identified as a potential pathway for the link between social experiences and cold susceptibility and is thus regarded as a marker of illness (e.g., Cohen, Doyle, & Skoner, 1999;

Murphy et al., 2017). Participants' daily quarantine nasal wash fluid was assayed for IL-6, IL-1β, and TNF-α via commercially available enzyme-linked immunosorbent assays (ELISAs) 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 five post-exposure days in quarantine, adjusted for the baseline cytokines. The composite measure was created by summing the standardized IL-6, IL-1β, and TNF-α scores (for examples of previous studies using this method with nasal proinflammatory cytokine measures, see Brody, Yu, Miller, & Chen, 2015 and Murphy et al., 2017).

Trait Depression. Depression was measured via the three-item Depression Subscale of the Trait Affect Scale. Participants were asked to rate on a scale from 0 (*not at all accurate*) to 4 (*extremely accurate*) how much the following traits described them "at the present time" and "generally or typically, as compared with other persons you know of the same sex and roughly the same age:" sad, unhappy, and depressed. The items were administered at two timepoints: approximately two months prior to quarantine and on the first day of quarantine. Scores at the two time points were highly correlated ($r = .56$, $p < .01$), and thus were averaged to create a mean trait depression score, with higher scores indicating greater trait-level depression.

Standard control variables. Seven standard covariates were included in all analyses: age, sex (dummy-coded as male = 0 and female = 1), race (dummy-coded as White = 0 and non-White = 1), socioeconomic status (represented by years of education), body mass index, season (recorded using two dummy-coded variables to represent winter, spring, and summer, with spring as the reference category), and baseline antibody titer (on day 0) to RV39 (coded as $< 4 = 0$ and $\geq 4 = 1$). These covariates were measured prior to beginning the study and were included

based on recommendations for using these data (e.g., see Cohen et al., 1997 and Murphy et al., 2017).

Data Analyses

The data were analyzed using IBM SPSS Statistics Version 25 and the PROCESS macro version 2.16 (Hayes, 2013). Simple moderation models (Hayes Model 1) were used to assess the interaction between early neighborhood risk and parental overprotection to predict six different outcomes: clinical cold diagnosis, infection status, nasal inflammatory markers, and trait-level depression among all participants, and mucus production weight and nasal mucociliary clearance function among participants who were infected with the challenge virus. In the Early Neighborhood Environment × Parental Overprotection moderation models, Early Neighborhood Environment was entered as the predictor variable, and Parental Overprotection was entered as the moderator. All of the standard control variables listed above were entered into all of the moderation models as covariates. Interactions were probed using the Johnson-Neyman and pick-a-point techniques.

CHAPTER 3

RESULTS

Demographic Characteristics

Demographic characteristics of the total sample and of those who were infected with the RV39 virus are presented in Table 1. The total sample consisted of 213 participants (57.7% male) ages 18-55 (M_{age} = 30.1, *SD* = 10.9), with 143 participants who identified as White and 71 participants who identified as non-White. The average education of participants was 14.1 years (*SD* = 1.89). Of the total sample, 160 participants met criteria for RV39 infection, and of those who were infected, 63 participants met objective criteria for clinical cold diagnosis. Bivariate correlations among study variables for all participants and for infected participants are presented in Table 2.

Childhood environments, parental overprotection, cold susceptibility, and depression

Results among all participants. Early Neighborhood Environment × Parental Overprotection moderation analyses were conducted with the total sample (*n* = 213) to evaluate (a) the probability of developing a clinical cold, (b) nasal proinflammatory cytokine production, (c) infection status (i.e., whether or not participants shed the challenge virus), and (d) trait-level depression.

There was suggestive evidence for the Early Neighborhood Environment × Parental Overprotection interaction predicting clinical cold diagnosis (*b* = -.11, *p* = .08, *n* = 212). Although nonsignificant, the pattern of results was such that for those who reported greater levels of perceived parental overprotection, neighborhood risk was negatively associated with the

probability of clinical cold diagnosis, and for those who reported lower levels of perceived parental overprotection, neighborhood risk was positively associated with the probability of clinical cold diagnosis (see Figure 1). A significant interaction emerged for Early Neighborhood Environment × Parental Overprotection to predict nasal proinflammatory cytokine production ($b = -.17$, $p = .01$, $n = 210$). The Johnson-Neyman probing technique revealed that at greater levels of parental overprotection (at a value of 14.8 or greater [29.7% of the sample] on the Parental Overprotection subscale), neighborhood risk was negatively associated with nasal proinflammatory cytokine production ($b = -.58$, $p = .044$; see Figure 2). The Early Neighborhood Environment × Parental Overprotection interaction was not significantly related to whether participants evidenced RV39 viral shedding ($b = .01$, $p = .89$, $n = 213$).

There was also a significant Early Neighborhood Environment × Parental Overprotection interaction to predict trait-level depression ($b = -.11$, $p = .02$, $n = 213$). The Johnson-Neyman probing technique revealed that at lower perceived parental overprotection (at a value of 11.3 or lower [38.0% of the sample] on the Parental Overprotection subscale), neighborhood risk was positively associated with trait-level depression scores ($b = .36$, $p = .045$, $n = 213$; see Figure 3).

Results among participants who were infected with the virus. To examine cold symptoms among those who were infected with the RV39 virus ($n = 160$), Early Neighborhood Environment × Parental Overprotection moderation analyses were conducted to evaluate (a) mucus production and (b) nasal mucociliary clearance time. A significant interaction to predict mucus production emerged ($b = -1.26$, $p = .03$, $n = 160$). The Johnson-Neyman probing technique revealed that at lower levels of perceived parental overprotection (at a value of 8.15 or lower [10.6% of the sample] on the Parental Overprotection subscale), neighborhood risk was positively associated with mucus production ($b = 7.09$, $p = .042$; see Figure 4). There was not a

significant interaction between early neighborhood environments and parental overprotection to predict nasal mucociliary clearance time ($b = -.17$, $p = .15$, $n = 159$), although the pattern was similar to the previous models: Among those who reported greater levels of perceived parental overprotection, neighborhood risk was negatively associated with nasal mucociliary clearance time (see Figure 5).

CHAPTER 4

DISCUSSION

The current study evaluated whether perceived parental overprotection throughout childhood influenced the associations among early neighborhood environments, cold susceptibility, and depression. Overall, there was suggestive evidence to support the hypothesis that for people who reported greater levels of perceived parental overprotection throughout childhood, neighborhood risk was negatively associated with the probability of developing a clinical cold and proinflammatory cytokine production. Additionally and interestingly, for participants who reported lower levels of perceived parental overprotection throughout childhood, neighborhood risk was positively associated with depression scores and mucus weight. These findings suggest that parental overprotection influenced markers of illness related to cold susceptibility and trait-level depression as a function of recalled childhood environments in a sample of healthy adults. Further, findings provide preliminary evidence that although parental overprotection might be beneficial for the well-being of individuals who report having grown up in riskier neighborhood environments throughout childhood, it might be detrimental to the physical and mental health of individuals who grew up in less risky neighborhood environments. Overall, the current study findings support the notion that health outcomes associated with parenting behaviors are contextual (e.g., Soenens et al., 2015).

The Early Neighborhood Environment × Parental Overprotection interaction was significantly related to nasal proinflammatory cytokine and mucus production and was suggestive for being associated with clinical cold diagnosis. However, the interaction was not

related to infection status (i.e., evidence of RV39 viral shedding throughout quarantine or a four-fold increase at the 28-day follow up visit) or nasal mucociliary clearance time. One possible explanation for these findings is that early neighborhood environments and parental overprotection might have influenced particular aspects of immune system functioning related to proinflammatory cytokine production, but not viral shedding. Proinflammatory cytokine production is a component of innate immunity, which is the immune system's first line of defense when a pathogen is encountered (Elwenspoek, Keuhn, Muller, & Turner, 2017). Conversely, viral shedding involves activation of both the innate and adaptive immune systems, which requires the recruitment and utilization of specialized and complex cells and cellular functions (Elwenspoek et al., 2017). In this sample of healthy adults, perceived childhood experiences (i.e., parental overprotection and neighborhood environments throughout childhood) influenced innate immune system activity, but perhaps these recalled experiences were unrelated to changes in adaptive immunity.

This study provided further evidence that childhood experiences have long-lasting impacts on adult physical and mental health (Ehrlich, Miller, & Chen, 2016). It is possible that the extent to which participants perceived their parents to be overprotective throughout childhood influenced subsequent psychological adjustment and lifestyle choices depending on their neighborhood context, which may have consequentially affected adult well-being. To elaborate, previous research findings indicate that in low-risk community samples, perceived overprotective (and by proxy, "helicopter") parenting behaviors are negatively associated with psychological well-being and positively associated with engaging in risky health behaviors, such as non-prescription pain pill usage (LeMoyne & Buchanan, 2011; Schiffrin et al., 2014). However, in at-risk populations, parental overprotection may actually have a positive influence

on health behaviors and lifestyles; for example, in a sample of urban African American adolescents, greater perceived parental monitoring was negatively associated with the likelihood of engaging in risk behaviors over a 4-year period (Li, Stanton, & Feigelman, 2000). The accumulation of psychological consequences and lifestyle choices associated with perceived parental overprotection in low vs. high risk contexts might translate to depressive symptoms and defenses against pathogens, such as the common cold virus, in adulthood.

The current study findings provide insight into the social correlates of acute illness upon viral exposure. However, findings should be considered in light of several limitations. First, the utilization of retrospective self-reports of early neighborhood environments and parental overprotection raises concerns about recall bias. Retrospective self-reports can be inaccurate due to time-lapses between experience and recall of events, current life circumstances, and personality characteristics, among other factors (Brewin, Andrews, & Gotlib, 1993; Maughan & Rutter, 1997; Reuben et al., 2016; Schwarz, 1999; Schwarz & Sudman, 1994). However, although memory reconstruction can be an issue when relying on retrospective perceptions of childhood experiences, there is evidence that some elements of individuals' lived experiences in childhood match their recall of events in adulthood (e.g., indicators of household dysfunction; Reuben et al., 2016). Furthermore, prior research findings have indicated that retrospective perceptions of experiences are equally and sometimes more powerful predictors of health outcomes than prospective observations (Newbury et al., 2018; Scott, McLaughlin, Smith, & Ellis, 2012; Widom, Weiler, & Cottler, 1999). In an ideal setting, researchers have access to both prospective and retrospective measures when evaluating links between childhood experiences and adult health outcomes. Therefore, an interesting future direction would be to capitalize on a longitudinal study to evaluate parenting characteristics and measures of neighborhood

environments in a sample of children, with the goal of linking these constructs with susceptibility to the common cold and trait-level depression in adulthood.

Another limitation is that participant's ages ranged from 18-55 years old, meaning that some participants were reporting on childhoods that took place recently (e.g., within the past five years) whereas some participants were reporting on experiences that took place decades earlier. Given the study's sample size, analyses are underpowered to explore a moderated moderation (i.e., with age moderating the Early Neighborhood Environment × Parental Overprotection interaction). Accordingly, we are unable to conclude whether findings might only be relevant for young, middle-aged, or older adults. However, it is worth noting that there were no systematic age differences in reports neighborhood risk or parental overprotection—age was not correlated with either construct (*r*s = -.10 and .04, *p*s > .05). Therefore, it is unlikely that study findings are driven by age cohort effects, such as generational differences in perceptions of neighborhood risk or parenting behaviors. However, a future direction would be to recruit narrow age groups in order to explore the possibility that study findings are influenced by participants' ages.

A final study limitation to consider is that the measure of parental overprotection might not have captured the extent to which participants perceived their parents to be overprotective throughout childhood due individual differences in the degree to which individuals interpreted overprotection (e.g., as being nurturing versus controlling). Therefore, the measure of parental overprotection may have captured other aspects of parenting, such as parental monitoring or involvement. As such, the measure of perceived parental overprotection may or may not have been a proxy for parental monitoring or involvement, both of which foster positive child and adolescent outcomes. For example, parental monitoring is associated with engaging in fewer risk behaviors in adolescence (e.g., DiClemente et al., 2001), which translates to better health in

adulthood. Ideally, future studies will include additional measures that evaluate several aspects of perceived parenting (e.g., parental overprotection, monitoring, and involvement) in order to precisely identify which behaviors influence adult physical and mental health.

A substantial amount of studies such as this one rely on adult populations to link childhood experiences with adult physical health, but far fewer studies address the question as to whether childhood experiences matter for *child* health. However, researchers have begun to capitalize on studies that utilize disease-centered approaches (i.e., studies that "reverse engineer" physical and psychological illnesses) in order to evaluate the social correlates of relevant health outcomes (Miller, Chen, & Cole, 2009). This approach makes it possible to study the social determinants of health in child populations for diseases that are prevalent in youth and for which the physiology is well known, such as asthma and depression (Miller et al., 2009). For example, Chen et al. (2019) found that for asthmatic children who live in risky neighborhood environments, having positive family relationships was associated with fewer asthma symptoms, whereas family relationships were unrelated to asthma symptoms for children living in low-risk neighborhoods. Therefore, an interesting future direction to pursue is the exploration of ways in which parental overprotection might influence health outcomes related to illnesses that are appropriate to study in child populations (e.g., asthma and depression) for children who live in high versus low-risk neighborhoods.

Researchers often refer to "the long arm of childhood" (Hayward & Gorman, 2004) when exploring the mechanisms by which illness and disease manifest later in life, and this study supports the notion that childhood experiences have long-lasting effects on physical and mental health into adulthood. Despite limitations, the current study provided preliminary evidence that perceived parental overprotection throughout childhood is not uniformly beneficial or

detrimental to adult health. Rather, greater levels of perceived parental overprotection buffered the link between recalled neighborhood risk and cold susceptibility, whereas lower perceived parental overprotection exacerbated the associations among recalled neighborhood risk, cold susceptibility, and depression. Study findings raise the questions: What are the mechanisms by which perceived overprotective parenting influences adult health? And further, what factors contribute to individual differences in perceived parenting behaviors? Future studies that investigate the questions raised by current study findings (e.g., via longitudinal study designs and the inclusion of sophisticated measures of perceived parenting) will help researchers better understand and address the childhood correlates of cold susceptibility and mental health outcomes in adulthood.

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Table 1.

Participant demographics among all participants (n = 213) and those who met criteria for infection (n = 160).

Demographic Characteristic		All participants (n = 213)	Met criteria for infection (n = 160)
		N (%)	N (%)
Age (years)			
	18-25	110 (51.6)	83 (51.9)
	26-40	56 (26.3)	41 (25.6)
	41-55	47 (22.1)	36 (22.5)
Sex			
	Male	123 (57.7)	95 (59.4)
Race/ethnicity			
	White	142 (66.7)	110 (68.8)
	Non-white	71 (33.3)	50 (31.3)
Education (years)			
	10-12	51 (23.9)	38 (23.8)
	13-15	108 (50.7)	81 (50.6)
	16-20	54 (25.4)	41 (25.6)
Season of trial			
	Spring	70 (32.9)	54 (33.8)
	Summer	86 (40.4)	69 (43.1)
	Winter	57 (26.8)	37 (23.1)

Table 2.

Correlations Among Study Variables for all participants (n =213) and participants who met criteria for infection (n = 160).

Variable	1	2	3	4	5
All Participants *(n = 213)*					
1. Early neighborhood risk	----				
2. Parental overprotection	.17*	----			
3. Clinical cold diagnosis	-.02	-.03	----		
4. Infection status	-.05	-.08	.38**	----	
5. Nasal proinflammatory cytokine production	-.10	-.05	.44**	.25**	----
6. Trait Depression	.04	.09	-.02	-.04	.06
Infected Only *(n = 160)*					
1. Early neighborhood risk	----				
2. Parental overprotection	.16*	----			
3. Mucus weight	.03	-.10	----		
4. Nasal mucociliary clearance function	-.08	.03	.22**	----	

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

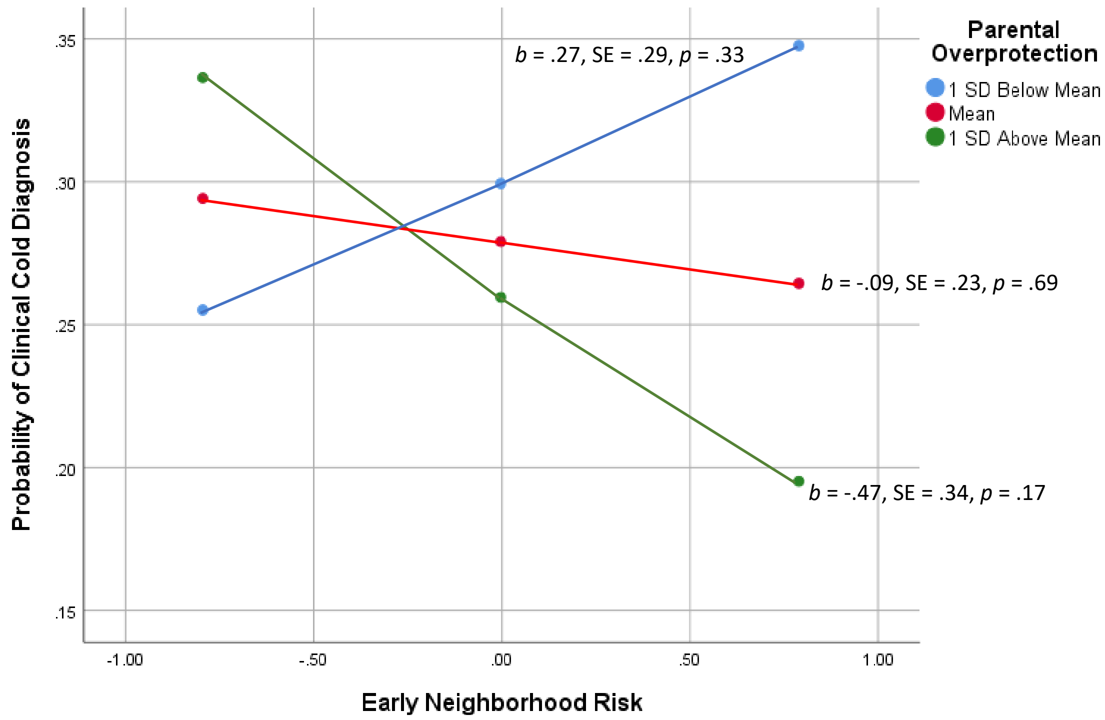

Figure 1. Probability of clinical cold diagnosis predicted by the Early Neighborhood Environment × Parental Overprotection interaction.

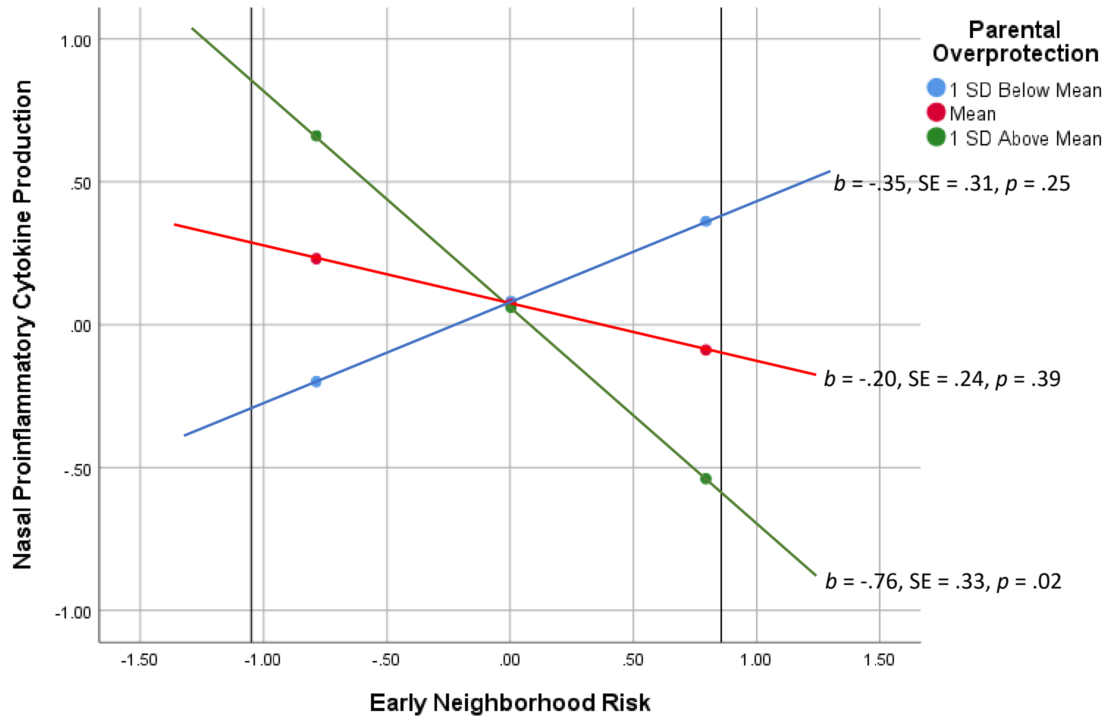

Figure 2. Nasal proinflammatory cytokine production predicted by the Early Neighborhood Environment × Parental Overprotection interaction.

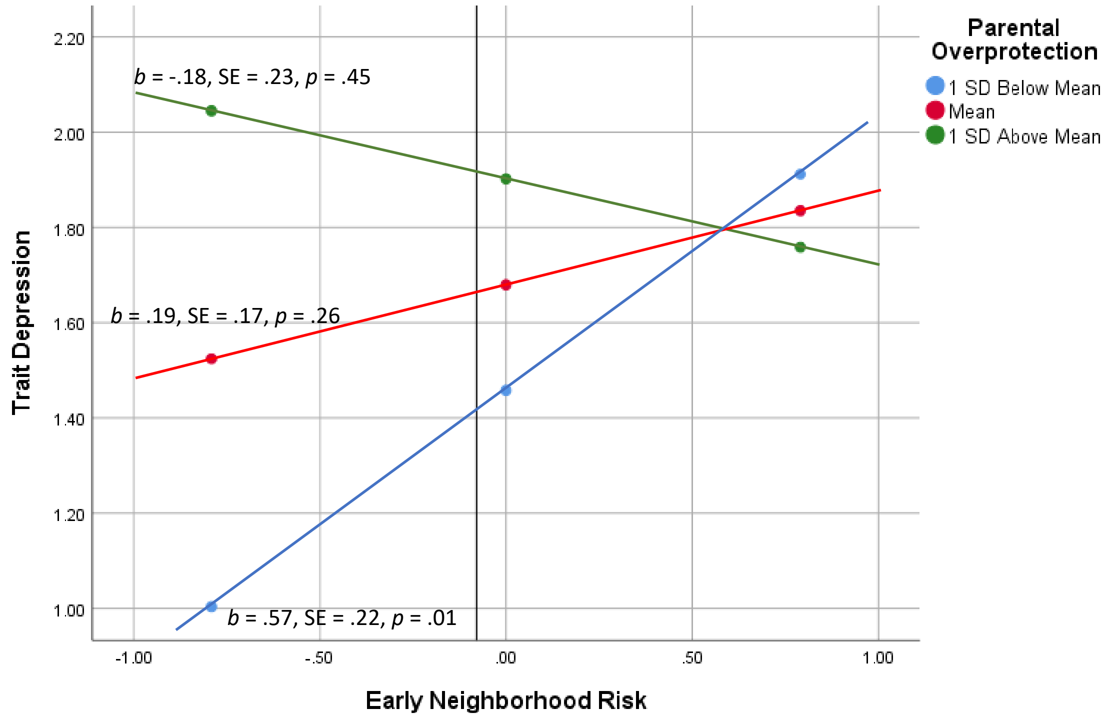

Figure 3. Trait-level depression predicted by the Early Neighborhood Environment × Parental Overprotection interaction.

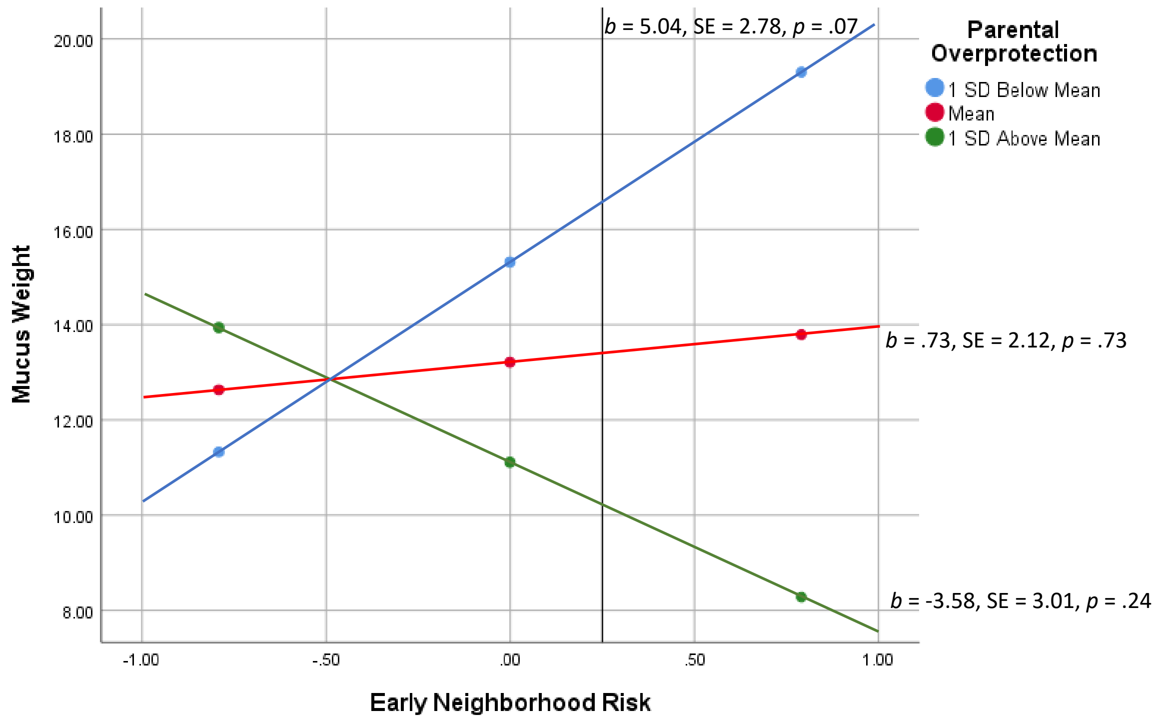

Figure 4. Mucus weight predicted by the Early Neighborhood Environment × Parental Overprotection interaction (among participants who were infected with the challenge virus).

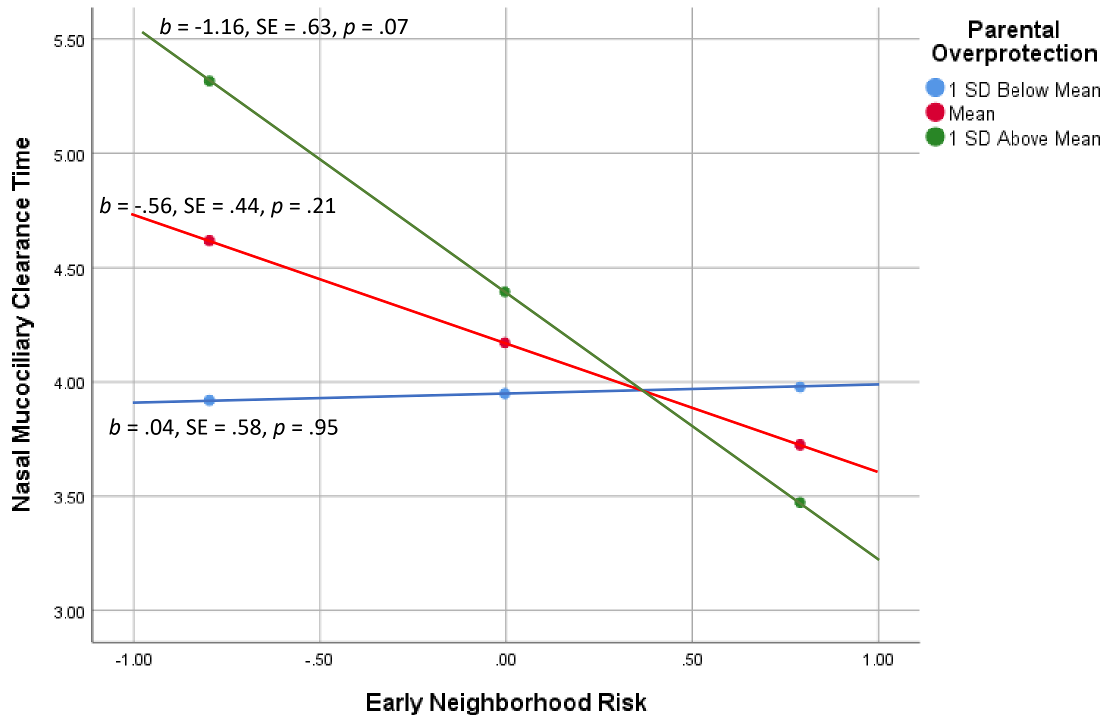

Figure 5. Nasal mucociliary clearance time predicted by Early Neighborhood Environment × Parental Overprotection interaction (among participants who were infected with the challenge virus).