Critical Windows of Poverty Exposure and Parental ACEs Predict Adolescent Behavioral and Physiological Health

by

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DISSERTATION ABSTRACT

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Title: Critical Windows of Poverty Exposure and Parental ACEs Predict Adolescent Behavioral and Physiological Health

Childhood poverty significantly increases the likelihood of children being exposed to poorer life conditions with long-term health consequences while also limiting access to optimal medical care, leading to increased morbidity and mortality across the lifespan. While the link between poverty and later life outcomes has been examined, gaps remain in our understanding of how the timing of childhood exposure to poverty and the specific impacts of family-level factors interact to shape adolescent health outcomes. The present study aims to investigate 1) if there is a differential impact of poverty on internalizing/externalizing behaviors and physiological outcomes in adolescence based on the timing of exposure, i.e., in early childhood, middle childhood, or adolescence, 2) whether family-level factors of parental stress and parental adverse childhood experiences (ACEs) moderate the associations between poverty exposure and internalizing/externalizing behaviors and physiological outcomes in adolescence, and 3) whether the aggregate effects of cumulative poverty exposure across developmental periods are associated with outcomes in adolescence, and whether these associations are moderated by family-level factors. Leveraging data from the Environmental influences on Child Health Outcomes (ECHO) program, a longitudinal, prospective, racial-ethnically diverse sample, I used structural equation modeling to test my research questions. I found differential effects of poverty on adolescent behavioral and physiological outcomes dependent on the timing of exposure. Specifically, exposure to poverty in adolescence (not in early childhood or middle childhood)

was significantly positively associated with internalizing behaviors in adolescence, and exposure to poverty in early childhood (not in middle childhood or adolescence) was significantly associated with poorer physiological outcomes in adolescence. Parental ACEs had a significant moderating effect such that the effects of exposure to poverty in middle childhood on adolescent internalizing and externalizing outcomes were significant when parental ACEs were high. Parental stress did not have a significant moderating effect. Moreover, both parental ACEs and parental stress were themselves significant predictors of both internalizing and externalizing behaviors. Overall, the findings indicate that the negative effects of childhood poverty may be dependent on developmental timing of exposure, where different critical windows lead to distinct adolescent outcomes and interactions with intergenerational effects. The findings highlight the need for trauma-informed interventions to potentially disrupt the intergenerational cycle of adversity.

Keywords: childhood poverty exposure, parental ACEs, adolescent health, developmental timing

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Finally, I want to thank my family: Ellen Gordon, Kris Kahn, Rachael Gordon, Juan Gramajo, and my Spanish family for always believing in my biggest dreams and encouraging me to follow what makes me happiest. And to my niece Camila: you are the reason all this work matters.

Dedication

This dissertation is dedicated to the children and families of my hometown, Oakland, California. Growing up in the culturally rich environment of Oakland shaped both the form and substance of my perspective on prevention research. Surrounded by people from heterogeneous backgrounds, I was immersed in a community from heterogeneous backgrounds, and where the lived realities of adversity, resilience, and trauma played out in everyday life. These experiences during my upbringing sparked my commitment to developing and delivering supports that are accessible, culturally responsive, and rooted in the strengths of the communities they serve. To the children and families of Oakland, you are the reason I do this work. May this research, in whatever ways it can, contribute to interventions that promote healing, equity, and thriving futures.

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CHAPTER I

INTRODUCTION

Childhood poverty is a public health concern impacting approximately 1 in 5 children (between birth–18 years old) in the United States, with this number steadily increasing (Shrider, 2024). Childhood poverty is a stressor that significantly increases the likelihood of children being exposed to poorer life conditions with long-term health consequences while also limiting access to optimal medical care (Cohen et al., 2010; Miller et al., 2011). Childhood exposure to poverty is associated with significant health impairments in adolescence (e.g., any physical health limitation that prevents them from working or going to school; Green et al., 2018), leading to increased morbidity and mortality across the lifespan. Moreover, it has also been linked to cardiometabolic risk in children as early as the age of 10, extending into adolescence (Doom et al., 2019). Further, evidence shows that later physiological health impairments, such as allostatic load in adolescence (Gordon et al., 2024) and BMI percentile trajectories throughout adulthood (Wells et al., 2010), are also shaped by early life exposure to poverty and poverty-related stressors, such as lack of resources and neighborhood dysfunction. This evidence suggests that the complex environmental landscape of poverty leads to increased risk that impacts youth not only through the behavioral processes at the individual level, but across biological domains, such as neuroendocrine, immune, and cardiovascular functioning, which dynamically interact to shape health outcomes (Minh et al., 2017; Halfon et al., 2018).

The etiology of behavioral and physiological health impairments (e.g., metabolic, immune, cardiovascular systems) is similarly related to early life exposure to poverty and poverty-related stressors. One potential mechanism for how exposure to poverty impacts behavioral and physiological health involves dysregulation of bidirectional pathways between

the neural and immune systems (Juster et al., 2011). Under favorable conditions, these systems work together to regulate behavior and stress responses, enabling individuals to adapt to environmental demands. However, repeated or prolonged activation of these systems can lead to chronic inflammation. This response can produce a dysregulated stress response which is positively associated with poor emotional regulation and consequently high-risk behaviors, such as physical inactivity and high-fat eating across the life course (Hostinar et al., 2018). Therefore, behavioral and physiological systems are interlinked and a deficit in one system may impede development and lead to deficits in another (Miller et al., 2011). Further, contextual factors linked to poverty, such as parental stress, can operate as moderators (Balistreri & Alvira-Hammond, 2016), potentially altering the impact of poverty exposure on behavioral and physiological outcomes.

Although the impact of childhood exposure to poverty on later health and behavioral outcomes has been investigated (McFarland, 2017), there remains a substantial gap in our understanding of the timing of poverty effects across development on long-term health outcomes. No study to date has systematically examined the effects of timing of poverty exposure on long-term health and behavioral outcomes using a longitudinal sample. The current study aims to address this gap by examining the timing of poverty exposure (in early childhood, middle childhood, and adolescence) on behavioral and physiological outcomes in adolescence using a longitudinal sample.

The Timing of Stressors

So far, no study has systematically tested if some developmental periods (i.e., early childhood, middle childhood, or adolescence) are more sensitive than others to the impacts of poverty exposure. Early childhood, specifically between birth and age five, represents a known

sensitive period for child development (Knudsen, 2004). Research suggests that rapid growth of children's brains during this time makes them particularly susceptible to environmental stimuli, both positive and negative (Belsky & Pluess, 2009). Early life stress, including poverty exposure, can have potentially permanent effects on brain development, such as impairments in learning and behavior, as well as stress-related physical and mental illness in later years (Shonkoff et al., 2012).

Single cohort studies have provided some evidence that the effects of exposure to poverty on behavior and health outcomes may differ depending on whether the exposure occurs during earlier vs. later development (Halfon et al., 2018). For example, exposure to poverty during biologically sensitive periods, such as early childhood (birth to age 5 years), is associated with more problematic behaviors at younger ages, which endure over time, than exposure to poverty at less critical periods (McFarland, 2017). Specifically, exposure to poverty during infancy is linked to more externalizing and internalizing problem behaviors in early childhood, and poverty during early childhood is consistently linked to internalizing symptoms in middle childhood. Moreover, exposure to poverty during early childhood is linked to higher risk of later health conditions such as autoimmune dysfunction, hypertension (Ziol-Guest et al., 2012) and other health issues across the lifespan (Boyce & Hertzman, 2018; Rasmussen et al., 2019). Although early childhood provides foundations for later development, exposure to poverty during middle childhood (age 6–10 years) and adolescence (age 11–18 years) is also critical for developmental outcomes (Green et al., 2018). However, the operationalization of middle childhood in past research has been arbitrary and often includes an overlap with early childhood or adolescence, making it difficult to isolate the unique effects that are specific to the middle childhood period. To overcome this challenge, middle childhood needs to be defined using age ranges grounded in

developmental theory and assessed distinctly from other developmental periods. Adolescence represents another sensitive period marked by rapid physical, social, emotional and cognitive development, during which exposure to poverty can be especially detrimental for adolescent health outcomes (Sawyer et al., 2012). Thus, longitudinal research is needed to isolate and compare the time-specific effects of exposure to poverty across developmental periods to better understand how timing shapes later behavior and health outcomes.

As mentioned above, a major limitation of existing research is a lack of focus on exposure to poverty across specific sensitive developmental periods, which may be critical for understanding how these factors shape health outcomes over time (Halfon et al., 2018). This is because identifying these longitudinal trajectories of exposure to poverty remains challenging, as the majority of evidence is retrospective with recollections of exposure to poverty measured at a single time point in adulthood (Lee et al., 2021). Further, there is debate as to whether the developmental timing of exposure to poverty during sensitive periods is more important for later health than continuous or cumulative exposure to poverty (Farrell et al., 2017; Green et al., 2018). In cases where poverty is stable and continuous, the aggregate exposure to poverty or cumulative effects may better explain health outcomes (Evans & Kim, 2007). However, when poverty exposure fluctuates across the developing years, it may be important to identify which periods of development are most sensitive to its harmful effects. Understanding these differential effects can inform more precise intervention strategies tailored to the timing of exposure.

The Family Environment

Despite the well-documented adverse effects of early life exposure to poverty, not all individuals exposed to poverty early in life develop health and behavioral problems, highlighting significant individual differences in susceptibility to psychosocial and neurobiological factors

(Ellis & Boyce, 2011; Belsky & Pluess, 2009). These differences may stem from the absence or presence of risk and protective factors. The family environment (i.e., the caregiving environment) is not only a structural intermediary between the individual and their physical environment, including housing, neighborhood conditions, and environmental exposures, but also the crucible of influences and stressors that shape developmental outcomes. Variations in the caregiving environment can impact the relationship between early stress exposure and physiological stress reactivity, as seen in studies of institutionalized children (McLaughlin et al., 2015). Prior research has also demonstrated that risk factors in the context of poverty (e.g., residential density, noise levels, housing quality) both negatively affect parent-child relationships (which influence the development of child self-regulation) as well as uniquely disrupt children's self-regulation processes (i.e., the ability to control attention, to plan, and to inhibit or initiate behaviors; Doan et al., 2012). Early deficits in self-regulation in turn predict behavioral problems in late adolescence suggesting that risk factors in the context of poverty have both unique and overlapping effects.

Parental stress is one potential family-level factor that may moderate the effects of childhood exposure to poverty on adolescent health outcomes. Evidence from a cross-sectional study on adolescents suggests that lower levels of parental stress not only have direct effects on adolescent internalizing problems and physical health outcomes but also serve as a protective factor by moderating (i.e., buffering) the negative impact of adverse experiences on child development (Balistreri & Alvira-Hammond, 2016). In comparison, the role of other family-level factors, such as parents' own adverse childhood experiences (ACEs), remains less understood. Notably, recent research suggests that a key predictor of adolescent functioning occurs even before birth, with intergenerational factors transmitted from the parent to child, such that the

greater ACEs a parent experienced in their own childhood is directly associated with greater behavioral problems in their offspring (Schickedanz et al., 2018). Further, a recent adoption design study by Leve et al. (2024) found that children whose biological mothers experienced high life stress had worse externalizing behaviors when adoptive mothers also reported that they had experienced more ACEs during their own childhood. The effects of these family-level factors can shape a child's stress response and behavior, with impacts that may persist beyond childhood.

Adolescent Functioning as an Emergence Period

Adolescence is a sensitive period marked by rapid biological, cognitive, emotional, and social changes that heighten sensitivity to both inherited vulnerabilities and environmental influences (Dahl, 2004). During this time, physiological functioning may begin to reflect the accumulation of early exposure to poverty and can be a potential precursor for later disease and illness, including cardiovascular disease and other non-communicable diseases (Doom et al., 2017). As such, the accumulated latent effects of poverty and early family adversity may emerge during adolescence to impact emotions and behaviors. For instance, poor health behaviors, such as smoking, physical inactivity, and high-fat eating, often emerge during adolescence and are direct determinants of health and well-being (Green et al., 2018). However, these behaviors are more likely products of the child's upbringing and environmental influences across earlier developmental periods, rather than due to immediate circumstances alone.

Existing research has examined how childhood poverty predicts various health outcomes in adolescence, marking this period as a potential target for intervention. For instance, childhood poverty is associated with health limitations (Green et al., 2018), poor general physical health (Luby et al., 2017), and elevated cardiometabolic disease risk (Wickrama et al., 2015). However,

relatively few studies have focused on modifiable factors, such as parental stress (Niu et al., 2018), that could mitigate the negative effects of early poverty exposure on specific markers of physiological functioning and behavior problems during adolescence. The present study seeks to fill these gaps by using a longitudinal sample to examine how the timing of childhood exposure to poverty and cumulative poverty exposure interacts with family-level factors to shape behavior and physiological health outcomes in adolescence. To do this, I examined the moderating effects of parental stress and ACEs on the association between poverty exposure and adolescent behavioral and physiological outcomes.

Present Study

The present study sought to (a) examine the effects of childhood exposure to poverty at different developmental time points as well as the cumulative effects on adolescent internalizing behaviors, externalizing behaviors, and physiological functioning, (b) determine whether family-level factors of parental stress and parental ACEs moderate these associations, and (c) examine whether the aggregate effects of cumulative poverty exposure across developmental periods was associated with outcomes in adolescence, and whether these associations were moderated by family-level factors. To do so, this study leveraged multiple waves of data from a longitudinal, prospective sample collected through the Environmental Influences on Child Health Outcomes (ECHO) program (Knapp et al., 2023). The specific research questions were as follows:

RQ 1: Does the timing of poverty exposure have a differential impact on behavioral and physiological outcomes in adolescence? Specifically, does the developmental timing [i.e., early childhood (birth–5 years), middle childhood (6–10 years), and adolescence (11–18 years)] of exposure to poverty have an impact on adolescent (age 11–18) behavioral (i.e., internalizing and externalizing behaviors) and physiological outcomes (e.g., blood pressure, BMI, waist

circumference). I hypothesized that poverty exposure would have an influence on all behavioral and physiological outcomes at each of the three time points (Figure 1). Further, early childhood poverty exposure was expected to have the strongest effects on adolescent internalizing behaviors, externalizing behaviors, and physiological outcomes, as compared to middle childhood or adolescence poverty exposure.

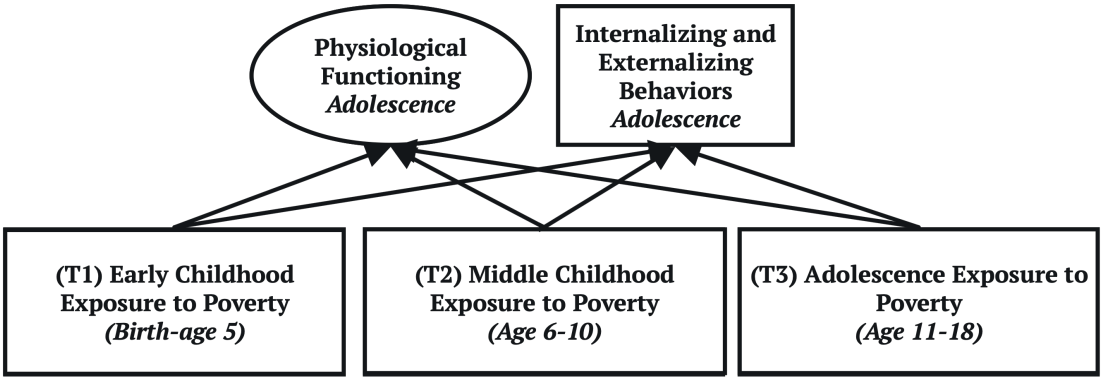

Figure 1. *Conceptual model for RQ1. Observed poverty exposure variables at each developmental period (early childhood, middle childhood, adolescence) and behavioral outcomes (internalizing, externalizing) are shown as rectangles. The latent physiological functioning outcome is depicted as an oval. Arrows indicate hypothesized paths.*

RQ 2: Do family-level risk factors (i.e., parental ACEs and parental stress) moderate the association between exposure to poverty at specific developmental periods [i.e., early childhood (birth–5 years), middle childhood, (6–10 years), and adolescence (11–18 years)] and adolescent behavioral and physiological outcomes (Figure 2)? I hypothesized that both parental ACEs and parental stress would significantly moderate the associations between poverty exposure and adolescent outcomes, such that the negative effects of poverty exposure on adolescent outcomes would be stronger at higher levels of parental stress and parental ACEs.

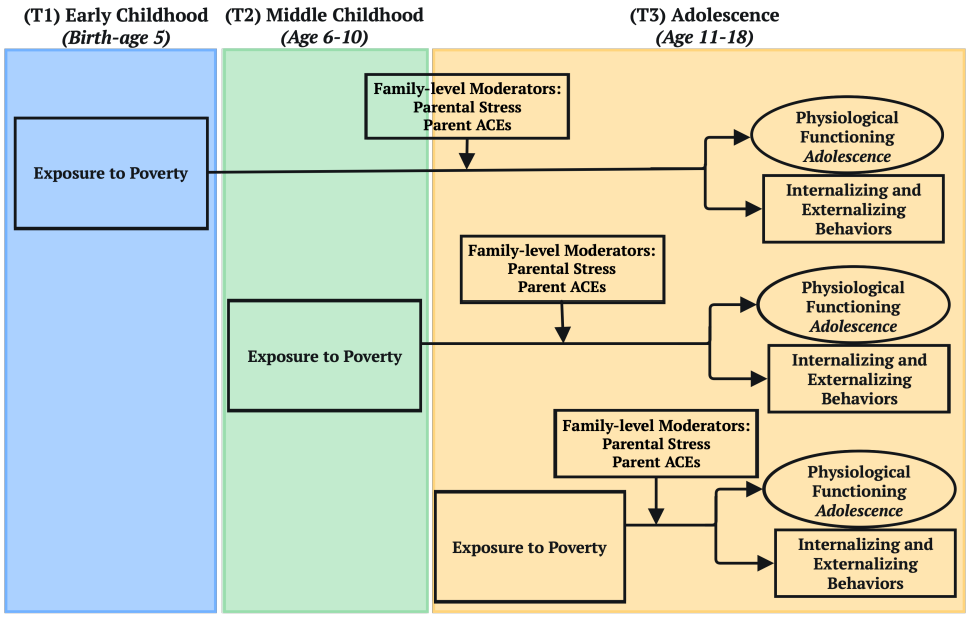

Figure 2. *Conceptual Model for RQ2. Family-level factors (i.e., time-averaged parental stress and parental ACEs) are modeled as moderators of the paths from poverty exposure at each developmental period (early childhood, middle childhood, adolescence) to three adolescent outcomes: internalizing symptoms, externalizing symptoms, and physiological functioning (latent).*

RQ 3: Does cumulative exposure to poverty across childhood and adolescence predict adolescent outcomes, and are these associations moderated by parental ACEs and parental stress? Because prior studies have commonly assessed poverty through cumulative exposure, and to determine whether aggregate exposure better explains adolescent health outcomes, I sought to examine whether the aggregate effects of cumulative poverty exposure across developmental periods (i.e., the sum of exposure to poverty across developmental periods) was associated with outcomes in adolescence and whether these associations were moderated by family-level factors (Mazza et al., 2017a). I predicted that the extent of cumulative exposure to poverty would determine the likelihood of adverse adolescent outcomes, and this relationship would also be moderated by family-level factors.

CHAPTER II

METHODS

Study Data Collection and Sample

Data were drawn from the National Institutes of Health (NIH)-funded Environmental influences on Child Health Outcomes (ECHO) program. ECHO is a prospective, longitudinal, nationally representative sample of approximately 50,000 children and their caregivers across 69 pediatric cohort study sites from 44 US states and Puerto Rico (Knapp et al., 2023). Participant data include repeated and independent measurements at three timepoints across critical periods: early childhood (birth to 5.9 years), middle childhood (age 6 to 10.9 years), and adolescence (age 11 to 18 years). Participants consented to the ECHO-wide Cohort Data Collection Protocol and provided consent to share extant data with the ECHO program. Recruitment of new participants and follow-up of existing cohort participants is ongoing (2023 to 2030). For this analysis, I used data that were collected after January 1, 1994, harmonized and shared on the ECHO-wide cohort database through August 31, 2022. After sub-setting the data for unique participants who had time-specific data for at least one assessment, outcome data were available for $N = 26{,}650$ adolescents (age 11–18 years; $M_{age} = 13.9$, $SD = 2.17$). The sample was 50% female, 44% non-Hispanic White, 17% Black/African American, 18% Hispanic, and 21% multiple or another race/ethnicity. Additionally, 32% of the sample reported an annual household income below $100,000 at childbirth.

Measures

Predictor

Childhood Exposure to Poverty was measured by comparing the total family income against the federal poverty level (FPL) thresholds using parental reports in early childhood,

middle childhood, and adolescence. Parents were asked for their total combined income of their household during the last calendar year. Total combined household income includes all money received by household members who contribute to household expense, such as total wages, salaries, self-employment income after expenses, government assistance of any kind, and interest and dividends before taxes. Total combined household income was calculated by summing all income sources contributed by household members toward expenses, including wages, salaries, self-employment income after expenses, government assistance, and interest or dividends, all assessed before taxes. This total income was then compared to the federal poverty level, adjusted for the number of household members and children under 18 years of age and inflation each assessment year. Based on established thresholds (Blackwell et al., 2024), families were categorized into a continuous FPL severity score: 0 = >350% of FPL (low poverty exposure), 1 = 133% to 350% of FPL (moderate exposure), and 2 = ≤133% of FPL (high exposure), with higher values reflecting greater exposure to poverty. Thirty-one percent of the sample reported a household income at or below 133% of FPL in early childhood, 42% in middle childhood, and 48% in adolescence. For the cumulative models, the sum of timepoints at or below 133% of FPL across the three time points was calculated (M = 2.39, SD = 1.01, range = 0–3), with higher scores indicating greater exposure to poverty.

Family-level Moderators

Two family-level factors were assessed. First, parental stress was based on parents' self-reports using the Perceived Stress Scale (PSS–10; Cohen et al., 1983) version of the NIH Toolbox Perceived Stress Scale Fixed Form Ages 18+ v2.0 (NIH PSS 18+). PSS–10 is a global measure of current perceived stress consisting of ten (six negative and four positive) items about feelings and thoughts related to events and situations that occurred in the last month. Responses

were recorded using a five-point Likert scale (never to very often), with a possible range of 0–40. The total score across the three time points (early childhood, middle childhood, and adolescence) was averaged to create a cumulative parental stress score with higher scores indicating more perceived stress (M = 18.76, SD = 4.54). Inter-item reliability in this sample was acceptable (α = 0.64).

Second, parents retrospectively reported on their own ACEs using the ACEs questionnaire (Felitti et al., 1998). They self-reported whether they had experienced 10 types of ACEs comprising emotional, physical, and sexual abuse, emotional and physical neglect, and familial dysfunction during their own childhood (before age 18). Responses were binary (0 = did not occur; 1 = did occur) and summed to produce a total ACEs score with a possible range of 0–10, with higher scores indicating more ACEs (M = 1.63, SD = 1.93). Inter-item reliability was acceptable in this sample (α = 0.86).

Adolescent Outcomes (child age 11–18)

Behavioral Functioning. The Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001), an established and widely used instrument with high validity and reliability, was used to assess behavioral functioning in adolescence (age 11–18). Caregivers completed the CBCL School-Aged version in adolescence. The CBCL consists of 113 items related to child behavioral issues scored on a 3-point scale ranging from not true (0) to often true or very true (2). The raw CBCL scores were standardized, and relevant items were summed to create the externalizing problem score (i.e., sum of the rule-breaking and aggressive behavior scores) and the internalizing problem score (i.e., sum of the anxious/depressed, withdrawn/depressed, and somatic complaints scores). The corresponding sex- and age-standardized t-scores were used for analysis. Higher scores indicate higher prevalence of problematic behaviors. Externalizing and

internalizing behavior t-scores were correlated at 0.62 ($p < .001$).

Physiological Functioning included six biological indicators measured in adolescence (age 11–18 years): 1) systolic blood pressure, 2) diastolic blood pressure, 3) waist circumference, 4) triceps skinfold measurement, 5) subscapular skinfold measurement, and 6) BMI. Each measure was examined individually using bivariate correlations. Then, all six were reduced into a single latent factor using Confirmatory Factor Analysis (CFA) to represent overall physiological functioning. Measures were collected when children were 11–18 years old by trained research staff according to guidelines specified in the National Youth Fitness Survey Body Measures Procedures Manual (NHANES, 2012). These included: 1) the average of three repeated systolic and 2) diastolic blood pressure measurements for each participant using a mercury sphygmomanometer, 3) subscapular and 4) triceps skinfold thickness measured on the right side of the body with a Holtain caliper (Holtain Ltd) as a sum of skinfolds to the nearest 0.1 mm, 5) waist circumference was assessed using the Young Adult survey self-waist measurement with a measuring tape at the uppermost lateral border of the hip crest (ilium) to the nearest 0.1 cm, and 6) body mass index percentile was assessed by collecting child height and weight and used to calculate BMI percentile (BMI; weight kg/height m^2). Measures prior to the COVID-19 pandemic were collected by home visit, then stopped between 2020–2021, and resumed as virtual visits in 2021. All body measures were taken three times, and the average was calculated across the three measurements. Measurement protocols for all variables were standardized across all ECHO study sites.

Covariates

All models were estimated separately for each outcome, with covariate controls for child sex and child race-ethnicity. This is based on the sex-based differences in BMI (Shah et al.,

2020), and that racial-ethnic minority groups in the United States tend to have higher rates of physiological dysregulation related to the increased rates of systemic oppression experienced (Richardson et al., 2021).

Statistical Analysis

A power analysis was conducted using G*Power Version 3 to estimate the required sample size for detecting main effects and moderation effects in regression models. With a medium effect size (d = 0.15), a sample size of 79 is sufficient to achieve high power (.99**)** at an alpha level of 0.01 for detecting main effects in standard multiple regression. For the moderation analyses, to detect a power of at least 0.80 at an alpha level of 0.05, a sample size larger than 200 is sufficient. To achieve higher power (.99) for the same effect size at $\alpha = .01$, a larger sample size of 300 is sufficient. A sample size of 150 is sufficient to achieve adequate power (0.99 at alpha .01) to detect medium effect sizes (0.15) with traditional moderation models.

Path analyses using structural equation modeling (SEM) were used to examine the associations between childhood exposure to poverty at different time points and adolescent outcomes, and whether the relationship was moderated by parental stress and parental ACEs. The hypotheses were tested in Rstudio (version 4.2.1) using the *lavaan* package for SEM (version 0.6–12; Rossell et al., 2012; Gana & Broc, 2019). Initial data screening showed non-normality, and models were estimated with maximum likelihood (ML) with robust standard errors and full information maximum likelihood (FIML) to account for missing data, allowing for the inclusion of all available data points. For the moderation models, the effects were tested with 95% confidence intervals (CI) for the model estimates. Moderation models were used to assess whether family-level factors moderate the associations between childhood exposure to poverty across developmental periods (i.e., early childhood, middle childhood, and adolescence)

and adolescent internalizing behaviors, externalizing behaviors, and physiological functioning. The models were assessed separately for each outcome with both family-level factors included as predictors and moderators to account for covariance between them. Interactions were probed using the Johnson-Neyman method to identify regions of significance (Figure 7). While the behavioral outcomes were retained as simple continuous variables, the continuous measures of physiological outcomes were assembled into a latent factor as has been done in prior studies (King et al., 2019). These indicators were standardized into a normal variable with $M = 0$ and $SD = 1$, which was performed so that the indicators would all be in the same metric.

Missing Data

Due to the longitudinal nature of the current study, some data were missing across time points. Specifically, of the 26,650 unique participants, only 395 had complete data across all waves, comprising a single birth cohort with poverty exposure assessed at each developmental period and outcomes measured in adolescence. To evaluate whether the missing data influenced the results, a sensitivity analysis was conducted comparing the full analytic sample to the complete case subsample. Descriptive statistics and correlations were calculated using all available data. The sample size varied by variable depending on missingness. To test whether data were missing completely at random (MCAR), Little's MCAR test was used on the sample of participants. The test indicated that data on variables used in all analyses reported were MCAR ($\chi^2(11) = 16.6$, $p = .121$). Hence, missing data were dealt with using FIML estimation (Enders, 2010) with an expectation maximization algorithm to obtain parameter estimates and standard errors, and account for missing data patterns in the analyses.

CHAPTER III

RESULTS

Descriptive statistics for all study variables and Pearson's correlations (*r*) are presented in Table 1. The pairwise bivariate correlations revealed that across the three developmental periods and cumulative poverty, only middle childhood exposure to poverty was significantly negatively correlated with internalizing behaviors ($r = -.07$, $p = .04$), but no significant correlations were found between any developmental periods or cumulative poverty and externalizing behaviors. Early childhood exposure to poverty was associated with BMI percentile ($r = .20$, $p = .001$), systolic blood pressure ($r = .11$, $p = .004$), and diastolic blood pressure ($r = .13$, $p = .001$) in adolescence. Middle childhood exposure to poverty was associated with BMI percentile ($r = .23$, $p = .001$), waist circumference ($r = .15$, $p = .001$), systolic blood pressure ($r = .07$, $p = .017$), diastolic blood pressure ($r = .09$, $p = .002$), subscapular skinfold thickness ($r = .16$, $p = .001$), and triceps skinfold thickness in adolescence ($r = .09$, $p = .042$). Adolescent exposure to poverty was associated with BMI percentile ($r = .10$, $p = .001$) and diastolic blood pressure ($r = .09$, $p = .02$) in adolescence. Parental stress was positively associated with both externalizing ($r = .19$, $p = .001$) and internalizing behaviors in adolescence ($r = .24$, $p = .001$). Parental ACEs were positively associated with both externalizing ($r = .15$, $p = .001$) and internalizing behaviors in adolescence ($r = .16$, $p = .001$). Parental stress was negatively associated with subscapular ($r = -.11$, $p = .014$) and triceps skinfold thicknesses ($r = -.11$, $p = .010$) in adolescence. Parental ACEs and parental stress were positively associated ($r = .18$, $p < .001$).

The standardized variable loadings on the latent adolescent physiological functioning factor were: subscapular skinfold thickness ($\beta = 0.94$), waist circumference ($\beta = 0.76$), triceps skinfold thickness ($\beta = 0.77$), BMI ($\beta = 0.68$), systolic blood pressure ($\beta = 0.33$), and diastolic

blood pressure (β = 0.27). All factor loadings were statistically significant ($p < .001$), with standardized estimates ranging from 0.94 to 0.27. The CFA fit index was poor, root mean square error of approximation (RMSEA) = .17, standardized root mean square residual (SRMR) = .11, and comparative fit index (CFI) = .74. Model fits were fully saturated when predicting internalizing and externalizing outcomes. Including predictors and covariates improved the model fit parameters for the latent physiological functioning factor indicating acceptable fit (RMSEA = .02, CFI = .69, SRMR = .07). The model outputs are presented in Table 2, and the results of the moderation analyses are illustrated in Figures 3–6.

Hypothesis 1

Regression models revealed that childhood exposure to poverty during specific time periods did significantly predict internalizing behaviors and physiological functioning in adolescence but did not significantly predict externalizing behaviors. Specifically, exposure to poverty in adolescence (not in early childhood or middle childhood) significantly predicted greater internalizing behaviors in adolescence (β = 1.561, SE = .65, $p = .024$; 95% CI [0.202, 2.920]), and exposure to poverty in early childhood (not in middle childhood or adolescence) significantly predicted poorer physiological functioning in adolescence (β = 0.332, SE = .10, $p = .001$; 95% CI [0.136, 0.527]). Further, I found that cumulative exposure to poverty did not predict internalizing nor externalizing behaviors, nor did it significantly predict physiological functioning. Therefore, while Hypothesis 1 was partially supported, these results revealed a more complex and nuanced relationship between the timing of poverty and outcomes.

Hypothesis 2

Direct Effects of Family-level Factors

Examining the family-level factors as predictors of outcomes in adolescence, I found that

parental ACEs predicted externalizing (β = 0.597, SE = 0.21, p = .004; 95% CI [0.261, 0.558]) and internalizing (β = 0.583, SE = 0.24, p = .016; 95% CI [0.111, 1.056]) behaviors in adolescence, but did not predict physiological functioning. Specifically, each one-unit increase in parental ACEs was associated with a 0.583-unit change and 0.597-unit change in internalizing and externalizing behaviors, respectively. Time-averaged parental stress significantly predicted externalizing (β = 0.410, SE = 0.08, p < .001; 95% CI [0.261, 0.558]) and internalizing behaviors (β = 0.576, SE = 0.09, p < .001; 95% CI [0.408, 0.744]) in adolescence, but not physiological functioning. Specifically, each one-unit increase in parental stress was associated with a 0.576-unit change and 0.410-unit change in internalizing and externalizing behaviors, respectively.

Moderating Effects of Family-level Factors

Higher middle childhood exposure to poverty significantly predicted greater externalizing behaviors (β = 0.965, SE = 0.48, p = .043; 95% CI [0.032, 1.898]) and internalizing behaviors (β = 1.390, SE = 0.537, p = .010; 95% CI [0.337, 2.443]) when parental ACEs were high. Due to the low mean and positively skewed distribution of parental ACEs, instead of probing interactions using ±1 SD, quantile-based categories of parental ACEs (low, moderate, high) were used. Simple slopes analyses revealed that middle childhood poverty predicted fewer internalizing problems when parental ACEs were low (low ACEs quantile = 0, n = 1198; t = -2.54, p = .011) and moderate (moderate ACEs quantile between 1 and 2, n = 1455; t = -2.44, p = .015). However, among families with high parental ACEs (high ACEs quantile > 2, n = 817), the association between poverty and internalizing problems was positive but not statistically significant (t = 0.82, p = .41). This finding is inconsistent with prior theoretical expectations that poverty would be associated with worse internalizing problems. This interaction is further

evaluated in the Sensitivity Analyses section.

The interactions between exposure to poverty × parental stress were not significantly associated with internalizing and externalizing behaviors nor physiological functioning at any time points. For the physiological outcomes, neither the interaction between adolescent exposure to poverty × parental stress, nor the interaction between childhood exposure to poverty × parental ACEs at any of the timepoints were significant. These findings partially support my predictions for Hypothesis 2, specifically that parental ACEs would moderate the relationship between childhood exposure to poverty and internalizing and externalizing behaviors in adolescence as illustrated in Figure 4. It is also notable that time-specific effects of middle childhood exposure to poverty on these outcomes were only significant when accounting for the moderating effect of parental ACEs.

Finally, when examining moderation on the cumulative effect of exposure to poverty across all time points, I found that when parental ACEs were high, higher cumulative exposure to poverty was significantly associated with greater internalizing behaviors (β = 0.664, SE = 0.314, p = .035; 95% CI [0.047, 1.280]) and externalizing behaviors (β = 1.002, SE = 0.361, p = .005; 95% CI [0.296, 1.709]), but not with physiological functioning in adolescence. The interaction between cumulative exposure to poverty × parental stress yielded no significant results for any of the abovementioned outcomes.

Sensitivity Analyses

To assess the robustness of the findings, I conducted a sensitivity analysis comparing the full sample to the subset of complete cases (N = 395). Descriptive statistics including mean, standard deviation, and variance tests (independent samples t-tests and Levene's tests for equality of variances) were used to examine differences in variables. Several variables showed

statistically significant differences in means between the two samples (Table 3). Specifically, poverty exposure was significantly lower in the complete case subsample across early childhood, middle childhood, and adolescence (all *p*s < 0.001), suggesting less poverty exposure among participants with complete data. Further, the variance of outcome variables in adolescence was significantly higher for the complete case subsample in middle childhood. For the outcomes in adolescence, the average scores for internalizing and externalizing symptoms were significantly lower in the complete case subsample (*p*s < .001 and .001, respectively), while physiological functioning indicators (i.e., BMI, waist circumference, and systolic blood pressure were significantly higher; *p*s ranging from .001 to .024), potentially reflecting socioeconomic differences. Restricting the sample to complete cases is therefore likely introducing bias. When using complete case analyses, the regression estimates for Hypothesis 1 were no longer statistically significant. This likely reflects sample bias, as children from higher socioeconomic backgrounds with fewer risk exposures may have been more likely to have complete data, attenuating the observed associations. Therefore, the direct effects of poverty exposure reported in Hypothesis 1 should be interpreted with caution, and future research should account for potential bias introduced by complete case analyses.

Importantly, the moderation patterns for both cumulative and middle childhood poverty exposure in predicting internalizing and externalizing problems remained consistent across the full and complete case samples. Additionally, there were no significant differences in neither mean nor variance for parental stress or ACEs between the two samples. Notably, the previously observed simple slope indicating that middle childhood poverty was associated with fewer internalizing problems when parental ACEs were low or moderate was no longer statistically significant in the complete case models. However, the direction and magnitude of the effect

remained similar, indicating that the pattern persists but may not reach significance thresholds in the smaller analytic sample.

CHAPTER IV

DISCUSSION

To my knowledge, this is the first study to examine the effects of timing of childhood exposure to poverty on adolescent physiological functioning, internalizing behaviors, and externalizing behaviors. In addition, this work further extends the literature by examining whether family-level factors, specifically, parental ACEs as a proxy for parental adversity, and parental stress within the caregiving environment interact with poverty exposure to predict adolescent outcomes.

Developmental Timing of Exposure to Poverty

The effects of exposure to poverty varied based on timing of exposure, with distinct, time-specific pathways linking poverty exposure to adolescent outcomes, highlighting the importance of timing. In support of my predictions for Hypothesis 1, I found that adolescent exposure to poverty was detrimental to adolescent internalizing behaviors, while early childhood exposure to poverty was detrimental to physiological functioning. Specifically, the immediate impact of adolescent poverty on adolescent internalizing behaviors may reflect proximal risk factors such as material deprivation, increased responsibilities, or financial stress (Miller et al., 2025). These stressors can compound during this developmental period when rates of internalizing problems, such as anxiety and depression, naturally start to increase (Miller et al., 2025). Exposure to poverty during this critical period can have a unique detrimental impact on developmental outcomes. The influence of poverty on internalizing problems could be related to poverty-related stressors (e.g., perceived discrimination, neighborhood context; Wadsworth et al., 2008) as well as poverty's direct effects on brain development (Luby et al., 2013) and self-regulation (Gordon et al., 2024).

Consistent with my prediction for the first hypothesis, I found a direct effect of early childhood exposure to poverty on adolescent physiological functioning. This pattern suggests that poverty's harmful effects manifest differently depending on when exposure occurs. This finding adds to a growing body of literature examining the long-term health costs of exposure to poverty (De France et al., 2022). Given that poor cardiovascular and metabolic markers are associated with increased risk for later disease and morbidity (Doom et al., 2017), poverty-related stress exposure during early childhood may be a critical pathway through which socioeconomic disadvantage "gets under the skin" to impact physiological functioning. The current results suggest that early childhood may represent a sensitive period during which exposure to poverty exerts particularly potent effects on the developing physiological stress response system. Moreover, the long-term impact of early childhood poverty on adolescent physiological functioning may reflect the effects of chronic or prolonged physiological stress, poor access to healthcare, and the early development of health-related behaviors, such as high-fat eating and low physical activity, that negatively influence physiological outcomes over time (Hostinar et al., 2018). These outcomes are likely compounded by both behavioral mechanisms (e.g., poorer nutrition, reduced access to healthcare) and biological pathways such as heightened hypothalamic-pituitary-adrenal (HPA) axis activity and chronic inflammation.

Despite the strong effects observed on physiological outcomes, early childhood poverty did not appear to have an impact on adolescent behavioral problems. One possible explanation is that this study looked at distal effects in adolescence while past studies have shown that the effects of early childhood poverty exposure may only be detectable on proximal outcomes (Mazza et al., 2017b). In addition, this finding contrasts with the results from McFarland et al. (2017), who found that poverty in early childhood significantly predicts internalizing and

externalizing behavioral outcomes that emerge in childhood and extend into adolescence. It is possible that the effects of early childhood poverty exposure on later behavioral outcomes may depend on the presence or absence of risk factors across development (i.e., disrupted emotion-regulation processes; Luby et al., 2013), which may be differentially distributed between the sample populations across our studies. Additionally, differences in how poverty was defined may contribute to the discrepancy in findings. McFarland et al. (2017) used stricter poverty thresholds, potentially capturing only the most extreme cases of deprivation compared to the broader criteria used in the current study. In these cases, exposure to more severe poverty may have yielded disruptions to brain development leading to enduring impacts on behavior that would not appear in cases of less severe poverty. Future research should aim to identify the differential effects of poverty exposure during developmental periods across a gradient of poverty severities to determine the extent to which these differences in type and magnitude of later outcomes are generalizable to diverse sample populations.

In contrast to the significant time-specific results, I did not find significant associations between the cumulative effects of accumulated exposure to poverty when averaged across all developmental time periods and adolescent internalizing behaviors, externalizing behaviors, or physiological functioning. While cumulative models are important for understanding effects of exposure to poverty in some contexts, they assume that all exposures contribute equally and linearly to outcomes which can obscure the distinct effects of individual exposures. These findings contrast with the accumulation of risk model (Mishra et al., 2009) which posits that poverty and low-income effects accumulate over time, leading to more pronounced adolescent behavior problems (Mazza et al., 2017a). One potential explanation for the lack of a cumulative

effect is that by collapsing risk across time, the cumulative model failed to capture unique and developmentally specific mechanisms of risk, as highlighted by the time-specific findings.

Direct Effects of Family-level Factors

I found that family-level factors (i.e., time-averaged parental stress and ACEs) directly predicted both internalizing and externalizing behaviors, while cumulative poverty was not predictive. This suggests that children were more likely to exhibit these behaviors when their parents experienced childhood adversity or when parental stress was high, regardless of poverty status. While both family-level factors were significant, the coefficient estimates suggest that parental ACEs had a stronger impact on adolescent outcomes than parental stress. These findings clearly indicate that the role of these familial factors is important for determining the behavioral outcomes of adolescents. Moreover, the quality of the caregiving environment and the adverse experiences of the parents may override the effects of poverty (as assessed by federal poverty level). This conclusion is further supported by emerging evidence from systematic reviews and meta-analyses. Specifically, children whose parents experienced ACEs are at greater risk of impaired brain development, physical health, emotional development, internalizing behaviors, and externalizing behaviors (Arnold et al., 2023; Racine et al., 2023). As the number of ACEs a parent was exposed to increases, the greater the negative effect on their children (Arnold et al., 2023). Further research should assess the mechanisms by which parental ACEs increase risk for behavior problems in offspring, such as impacts on their parenting and self-regulation which can in turn impair development of their children's self-regulation, especially for children who were also exposed to poverty.

Moderating Effects of Family-level Factors

I found that not only are parental ACEs strong predictors of these outcomes, but also that

they moderate the effect of exposure to poverty both in a cumulative context and during specific developmental periods. Moreover, these results are particularly robust as we know that parental ACEs were not masking effects of poverty since the bivariate correlations between parental ACEs and poverty in each developmental period were small. In partial support of Hypothesis 2, I found that parental ACEs moderated the positive association between childhood poverty exposure and adolescent internalizing and externalizing problems, with this effect emerging exclusively for poverty exposure in middle childhood. In the full sample model, the simple slopes analysis revealed that poverty in middle childhood predicted fewer rather than more internalizing problems, but this effect only occurred when parental ACEs were low and moderate, not when they were high. This could suggest that children in families with low and moderate parental ACEs develop adaptive coping skills or benefit from protective factors, such as stable caregiving or community support, that offset the negative effects of economic hardship which help build resilience to internalizing problems (Hostinar & Miller, 2019). However, this association did not appear when assessed with the complete case subsample. This suggests the moderation results from the full sample model may have been influenced by missing data patterns or sample variability. Therefore, while this finding offers an interesting avenue for exploring protective factors for children in families with low and moderate parental ACEs, it should be interpreted with caution.

Moderation effects emerged for behavioral outcomes (internalizing and externalizing symptoms) but not for physiological functioning. Behavioral symptoms may be more immediately responsive to relational stressors, particularly in middle childhood and adolescence, while physiological systems likely reflect more prolonged poverty exposure. Further, I found significant moderation on the cumulative poverty effects, suggesting that the impact of

accumulated exposure to poverty across development on behaviors in adolescence is amplified when parents have a history of ACEs. One potential explanation could be that parents with more ACEs may have difficulties with self-regulation, may have less supportive and/or maladaptive parent-child relationships (Rowell & Neal-Barnett, 2020), and may have less financial stability or a lack of resources (Harter & Harter, 2021). Middle childhood is a particularly vulnerable period where children are exploring their autonomy and taking on more responsibilities but are still reliant on caregivers for emotional support (Collins & Madsen, 2019). As a result, less supportive caregiving environments or dysregulated caregiving may have stronger negative effects on children's emerging internalizing and externalizing behaviors. Relatedly, it was surprising not to find a moderating effect for parental stress on behavioral outcomes in adolescence. It is possible that summing parental stress across developmental periods diluted the ability to detect specific effects which may have been more proximal. Further, I did not find moderation effects from family-level factors in early childhood and adolescence, which may have a developmental explanation. In early childhood, children are more sensitive to immediate environmental influences, such as caregiving quality and home chaos (Doan & Evans, 2020). At this stage, the intergenerational effects of parental ACEs may not yet have fully manifested to impact behavioral development. Whereas in adolescence, shifts in parent-child relationship dynamics and increased importance of peer relationships may buffer the negative effects of parental adversity.

Previous studies show that middle childhood may be a sensitive period during which internalizing and externalizing behaviors develop and become established (Wang et al., 2023). While parental ACEs and poverty exposure are known risk factors for externalizing behaviors, findings from this study suggest that middle childhood may be a sensitive period during which

these interacting risk factors can add further vulnerability. Exposure to poverty in middle childhood while also having parents who also experienced childhood adversity may impede children's ability to regulate their emotions and behavior and puts them at greater risk of problematic behaviors in the future. Further research should aim to identify the mechanisms underlying the potential impacts of parental dysregulation on problem behavior development during middle childhood.

Prior work suggests that parent-child relationships can similarly exacerbate the effects of exposure to poverty or serve as a protective factor for children depending on environmental contexts, such as poor neighborhood quality (Bush & Peterson, 2013). As such, greater parental ACEs within the context of childhood poverty exposure can exacerbate later behavioral outcomes as shown in this study. Despite the growing evidence, few studies have examined the interaction between parents' own childhood adversity or parental stress and time-specific poverty exposure during development on adolescent outcomes. Trauma-informed prevention efforts that address parental stress and impacts of parental ACEs on parent-child relationships and parenting behaviors among children living in poverty may be most effective at breaking the intergenerational transmission of ACEs (Woods-Jaeger et al., 2023).

Limitations

Despite the robustness of the findings, several limitations should be noted. First, while the study offers insights into the relationship between family-level factors and exposure to poverty on adolescent outcomes in a racial-ethnically diverse, longitudinal sample, the results may not be generalizable across different subgroups (based on socio-demographic factors). Second, the majority of data used in this study were self-reported with the exception of physiological markers, which introduces potential risk for recall bias and misclassification.

Third, although the study controlled for various demographic factors, other potential confounders such as genetic predispositions, environmental exposures, and psychosocial stressors were not explicitly measured and may have impacted the observed relationships. Fourth, the current poverty measure was solely based on income thresholds. This approach may fail to capture the many ways poverty can impact outcomes through deprivation or lack of resources and exposure to risks. Finally, considerable missing data exist, as only a subset of the larger cohort provided complete information for all relevant measures. While FIML was used to handle the missingness, there are limits to this approach. As such, findings should be interpreted with caution. Moreover, some constructs (e.g., parental stress) were well assessed in early childhood, but similar constructs were not assessed in adolescence or were not assessed across time points. Ideally, future longitudinal studies would administer the same measures across developmental time points, allowing researchers to examine whether these patterns differentially relate to other variables of interest, such as environmental (i.e., neighborhood quality) and poverty-related stressors (i.e., lack of resources, financial stress). Additionally, future research should aim to incorporate other relevant covariates and confounding variables that were not available in the current study, such as parental depression and anxiety, neighborhood violence, and access to social support systems. Accounting for these factors would help clarify the unique and interacting effects of poverty and parent adversity on adolescent health outcomes.

Conclusion

The study found evidence for not only *when* childhood exposure to poverty is most critical, but also, I found evidence *for whom* the effects are stronger. My findings show that the consequences of poverty exposure may not be uniform but rather varied depending on the time period of incidence, indicating a complex relationship with distinct developmental processes.

Further, I found additional evidence that risk factors in the caregiving environment (through parental ACEs) may have long-lasting downstream consequences that persist across generations. Specifically, the findings highlight that the impact of childhood poverty may vary depending on parental trauma history and the developmental periods in which exposure to poverty occurs, highlighting the importance of family-focused, trauma-informed targeted interventions during these sensitive periods. These sensitive periods may present critical windows during which tailored supports and social policy can reduce later problem behaviors and improve health across the life course.

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Table 1. *Correlation Matrix and Descriptive Statistics of Study Variables*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Poverty Exposure–Early Childhood															
2. Poverty Exposure–Middle Childhood	0.717***														
3. Poverty Exposure–Adolescence	0.610***	0.622***													
4. Cumulative Poverty Exposure	0.790***	0.768***	0.728***												
5. Externalizing Symptoms (Adolescence)	0.009	-0.042	0.017	0.025											
6. Internalizing Symptoms (Adolescence)	-0.031	-0.070*	-0.024	-0.035	0.624***										
7. Parental Stress	-0.080***	0.082***	-0.060*	-0.112*	0.190***	0.236***									
8. Parental ACEs	0.167***	0.151***	0.169***	0.130**	0.149***	0.156***	0.182***								
9. Waist Circumference (Adolescence)	0.093	0.145***	0.046	0.032	0.025	0.114***	0.024	-0.059							
10. BMI percentile (Adolescence)	0.196***	0.225***	0.104***	0.06	0.023	0.006	-0.043	0.003	0.650***						
11. Systolic BP (Adolescence)	0.112**	0.073*	0.045	0.015	-0.004	0.099**	0.021	0.015	0.359***	0.218***					
12. Diastolic BP (Adolescence)	0.128***	0.093**	0.085*	0.003	0.022	0.102***	0.044	0.048	0.236***	0.159***	0.490***				
13. Subscapular Skinfold Thickness (Adolescence)	0.073	0.164***	0.08	-0.03	0.045	0.058	-0.114*	0.065	0.668***	0.582***	0.173***	0.152***			
14. Triceps Skinfold Thickness (Adolescence)	0.115	0.090*	0.022	0.073	0.051	0.015	-0.115*	-0.003	0.465***	0.467***	-0.001	0.064*	0.753***		
15. Child Sex	-0.002	0.002	0.038	0.041	-0.060**	0.015	-0.002	0.026	-0.087***	0.023	-0.184***	0.013	0.051	0.080**	
16. Child race/ethnicity	0.126***	0.153***	0.087***	0.141**	0.042*	0.037	-0.057***	-0.001	0.005	0.046*	-0.033	-0.004	0.016	0.028	0.004
N	7314	6818	2306	475	2821	2821	13715	3470	1786	3133	2270	2273	1091	1168	
M	0.78	0.75	0.79	2.31	45.79	49.02	18.76	1.59	58.51	67.80	82.97	50.13	11.69	15.09	
SD	0.79	0.78	0.79	1.06	10.31	11.32	4.54	1.91	12.76	28.74	8.78	8.24	7.34	8.60	
Min	0	0	0	0	33	33	0	0	16.75	1.05	53	26.25	2.5	2.5	
Max	2	2	2	3	95	98	36	10	123.03	100	123.75	201	71.75	119.25	

Note. BP = Blood Pressure; ACEs = Adverse Childhood Experiences. *p < .05; **p < .01; ***p < .001.

Table 2. *Regression and Moderation Model Outputs.*

	Predicting Adolescent Internalizing Symptoms			*Predicting Adolescent Externalizing Symptoms*			*Predicting Adolescent Physiological Functioning*		
Regression Models	**β**	**SE**	**B [95%CI]**	**β**	**SE**	**B [95%CI]**	**β**	**SE**	**B [95%CI]**
Poverty Exposure – Early Childhood	-1.12	0.98	-1.73 [−3.66, 0.20]	-1.10	0.87	-0.29 [-0.306, 0.106]	0.33*	0.1	0.25 [0.052, 0.446]
Poverty Exposure – Middle Childhood	-1.12	0.89	-1.71 [−3.45, 0.03]	-0.05	0.79	-0.66 [-0.258, 0.158]	0.17	0.09	0.12 [-0.005, 0.251]
Poverty Exposure – Adolescence	1.11*	0.69	1.56 [0.20, 2.92]	0.09	0.63	1.15 [-0.053, 0.227]	-0.13	0.08	-0.10 [-0.290, 0.090]
Parents' Perceived Stress	0.24***	0.09	0.58 [0.41, 0.74]	0.19***	0.08	0.41 [0.141, 0.240]	-0.01	0.01	-0.01 [-0.023, 0.021]
Parental ACEs	0.09***	0.24	0.58 [0.11, 1.06]	0.11***	0.21	0.59 [0.035, 0.186]	-0.05	0.03	-0.09 [-0.170, -0.002]
Moderation Models									
Poverty Exposure – Early Childhood × Parents' Perceived Stress	-0.10	0.24	-0.28 [−0.75, 0.19]	-0.05	0.21	-0.12 [-0.123, 0.031]	-0.01	0.03	-0.04 [-0.072, 0.054]
Poverty Exposure – Middle Childhood × Parents' Perceived Stress	0.08	0.23	0.24 [−0.22, 0.70]	0.01	0.20	0.03 [-0.072, 0.095]	-0.01	0.03	-0.04 [-0.065, 0.041]
Poverty Exposure – Adolescence × Parents' Perceived Stress	0.04	0.16	0.13 [−0.18, 0.44]	0.04	0.14	0.12 [-0.049, 0.127]	0.017	0.02	0.06 [-0.020, 0.054]
Poverty Exposure – Early Childhood × Parental ACEs	-0.10	0.53	-0.10 [−1.15, 0.94]	-0.06	0.47	-0.38 [-0.167, 0.054]	0.028	0.06	0.04 [-0.092, 0.148]
Poverty Exposure – Middle Childhood × Parental ACEs	0.19**	0.54	1.39 [0.34, 2.45]	0.14*	0.48	0.96 [0.031, 0.253]	-0.09	0.07	-0.12 [-0.213, 0.041]
Poverty Exposure – Adolescence × Parental ACEs	-0.08	0.44	-0.06 [−1.50, 0.21]	-0.01	0.38	-0.07 [-0.098, 0.077]	0.09	0.06	0.12 [-0.039, 0.209]

Note. The outputs are standardized regression coefficients (β), standard errors (SE), and 95% confidence intervals (CIs) with unstandardized regression coefficients (B) for the effects of poverty exposure in early childhood, middle childhood, and adolescence on adolescent internalizing symptoms, externalizing symptoms, and physiological functioning. Interaction terms test whether these associations vary by family-level factors (parental stress and parental ACEs).

Table 3. *Descriptive Statistics from the Sensitivity Analysis Comparing the Full Sample and the Subset of Complete Cases Including Mean, Standard Deviation, Mean Difference, and Variance Difference.*

	Full Sample				Complete Cases Subset				
Variable	*Missing Percent (%)*	*N*	*Mean*	*SD*	*N*	*Mean*	*SD*	*Mean Difference*	*Variance Difference*
Poverty Exposure (Early Childhood)	72.6	7314	0.78	0.79	395	1.28	0.78	-0.49 ***	1.026
Poverty Exposure (Middle Childhood)	74.4	6818	0.75	0.78	395	1.28	0.7	-0.53***	1.224**
Poverty Exposure (Adolescence)	91.3	2306	0.79	0.79	395	1.05	0.75	-0.26***	1.114
Parental ACEs	87	3470	1.59	1.91	395	1.56	1.8	0.03	1.125
Parental Stress	48.5	13715	18.76	4.54	395	18.95	4.66	-0.19	0.949
Internalizing Symptoms	89.4	2821	49.01	11.33	395	45.62	10.64	3.4***	1.134
Externalizing Symptoms	89.4	2821	45.79	10.31	395	43.5	10.38	2.29***	0.986
BMI z-score	88.2	3133	0	1	386	0.25	0.91	-0.25***	1.205*
Waist Circumference z-score	93.3	1786	0	1	71	0.36	1.18	-0.36*	0.718
Subscapular Skinfold z-score	95.9	1091	0	1	65	0.05	0.98	-0.05	1.035
Triceps Skinfold z-score	95.6	1168	0	1	68	0	1.02	0	0.965
Diastolic BP z-score	91.5	2273	0	1	79	0.1	0.74	-0.1	1.812**
Systolic BP z-score	91.5	2270	0	1	79	0.42	0.85	-0.42***	1.382

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

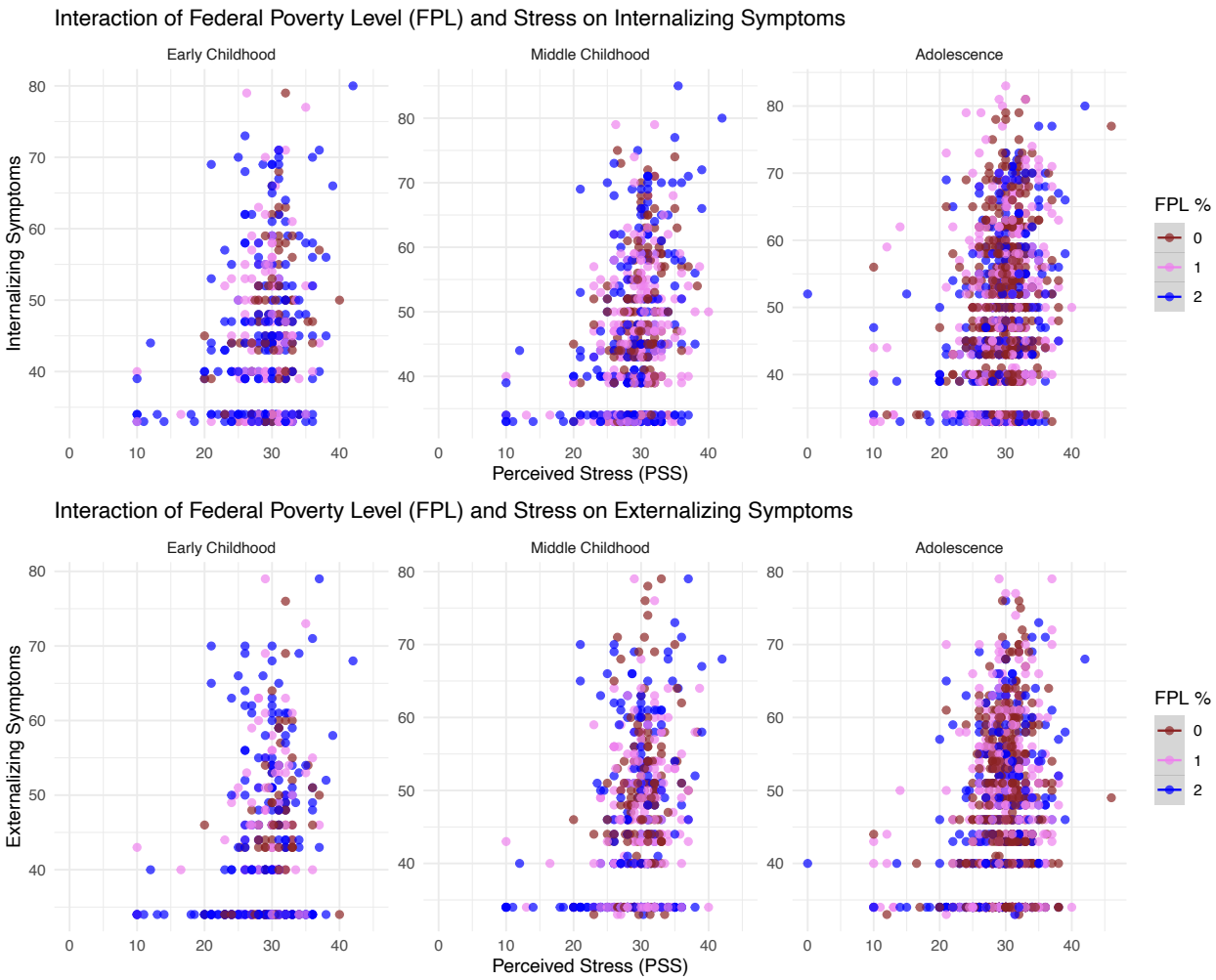

Figure 3. *Scatterplot Showing the interaction of Federal Poverty Level (FPL) and Parental Stress on Internalizing Symptoms & Externalizing Symptoms*

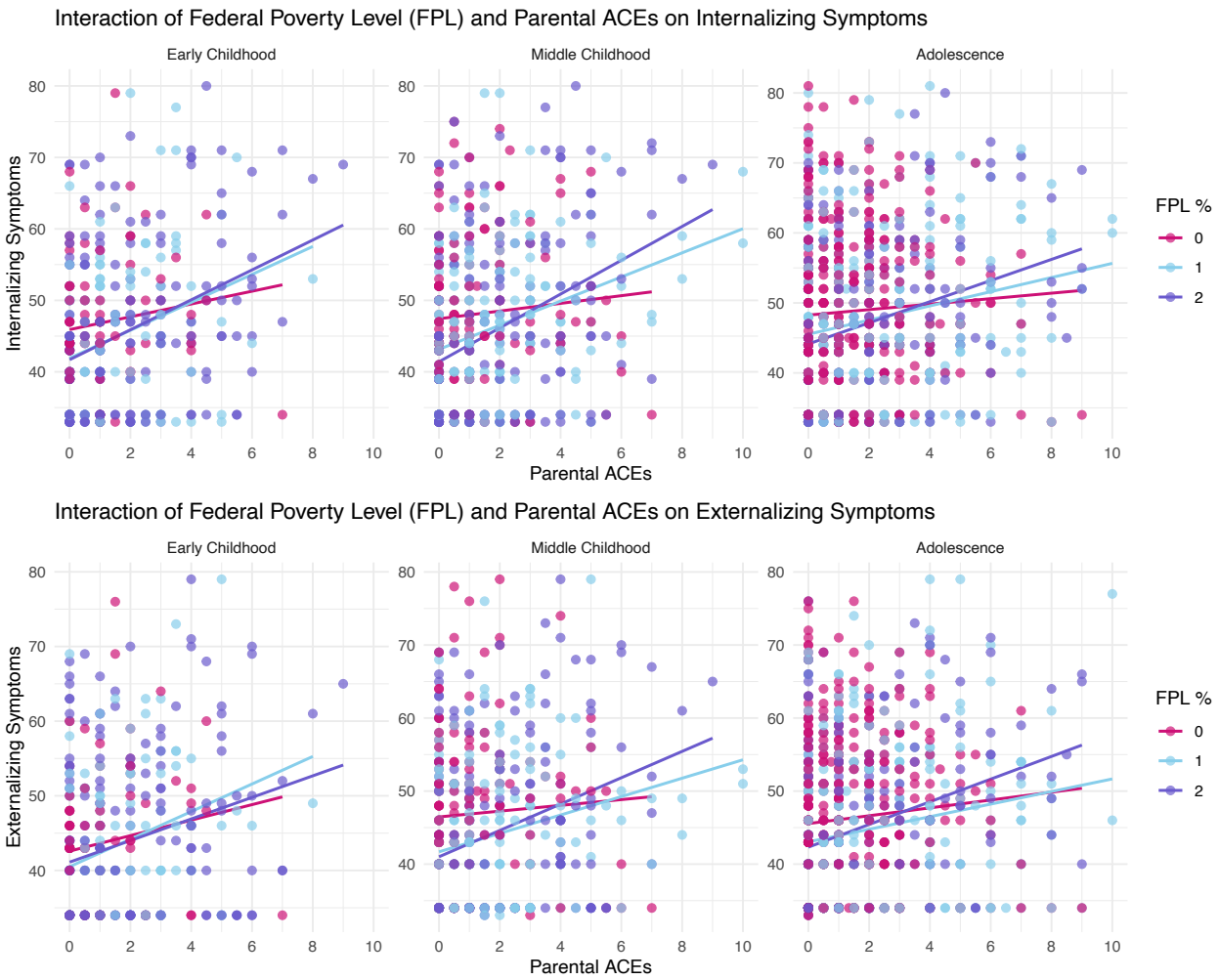

Figure 4. *Scatterplot Showing the Interaction of Federal Poverty Level (FPL) and Parental ACEs on Internalizing Symptoms & Externalizing Symptoms*

Figure 5. *Scatterplot Showing the Interaction of Federal Poverty Level (FPL) and Parental Stress on Physiological Functioning*

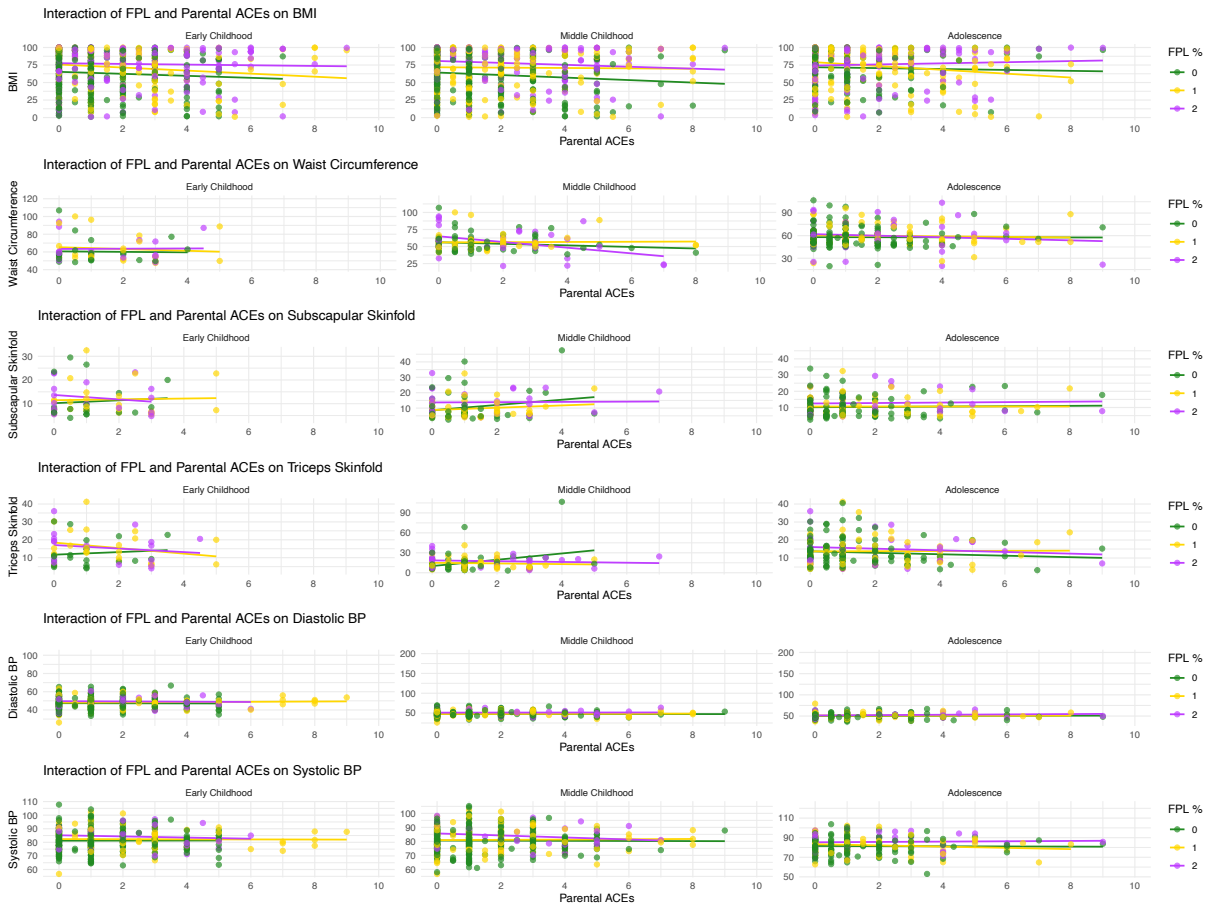

Figure 6. *Scatterplot Showing the Interaction of Federal Poverty Level (FPL) and Parental ACEs on Physiological Functioning*

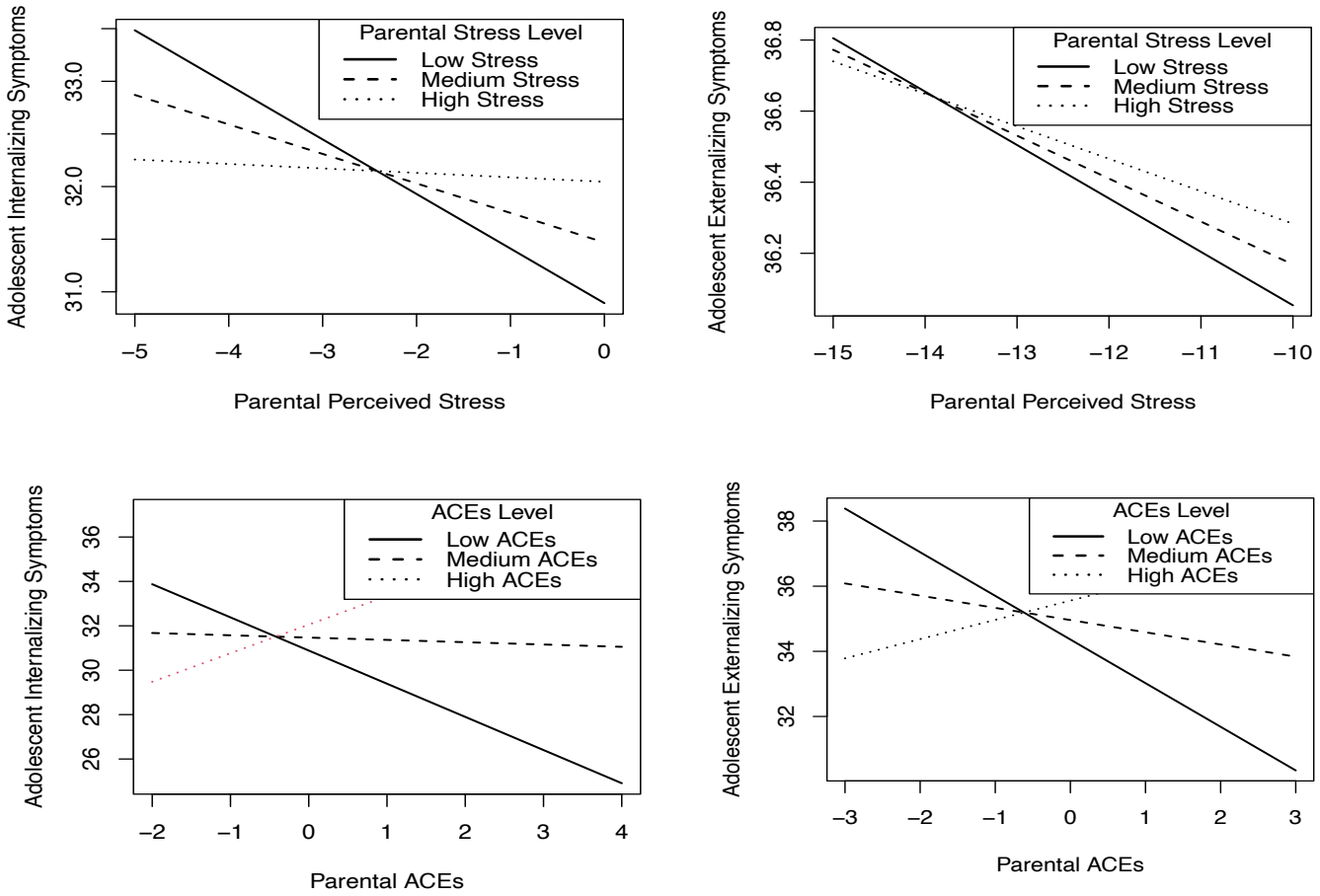

Figure 7. *Johnson-Neyman Probes of Moderation. Each panel shows the interactions between adolescent behavioral outcomes and childhood exposure to poverty, at three levels of the family-level moderator (parental stress or parental ACEs). Lines represent the simple slopes when the moderator is 1 SD below the mean (solid), at the mean (dashed), and 1 SD above the mean (dotted).*