THE IMPACT OF DISADVANTAGE ON RISK FOR DISORDERED EATING IN YOUTH: LONGITUDINAL ASSOCIATIONS AND INTERACTIONS WITH GENETIC INFLUENCES

By

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ABSTRACT

Contrary to historical stereotypes, emerging research suggests eating disorders (EDs; e.g., anorexia nervosa, bulimia nervosa, binge-eating disorder) and disordered eating symptoms (e.g., body dissatisfaction, binge eating) may be elevated among people experiencing at least some forms of socioeconomic disadvantage. However, the relatively small body of research conducted on poverty and EDs thus far has generally focused on proximal and relatively narrow indices of disadvantage, such as food insecurity. Disadvantage is a multifaceted construct, and fully understanding the impact of disadvantage on disordered eating requires integration of both proximal (e.g., lower family income) and more distal (e.g., poverty and decreased resources in the neighborhood) aspects of the socioeconomic context. In addition, there has been little research on interactions between disadvantage and other risk factors for disordered eating (e.g., genetic risk), changes in the impact of disadvantage across development, or the "active ingredients" of more distal forms of disadvantage at the neighborhood level that most strongly contribute to disordered eating. The three studies included in this dissertation addressed these gaps in the literature. Study 1 conducted developmentally informed genotype x environment analyses to understand how proximal (family income) and distal (neighborhood disadvantage) forms of socioeconomic disadvantage may interact with genetic influences to impact risk for disordered eating in boys. These analyses built on prior work in girls by examining interactions between disadvantage and genetic influences across the developmental period most critical for emergence of genetic risk for disordered eating in boys (adrenarche, an early stage of puberty). Boys from disadvantaged neighborhoods or families showed earlier activation of genetic influences on disordered eating in early adrenarche, when genetic influences were minimal for boys living in more advantaged contexts. Boys living in disadvantaged contexts also reported greater phenotypic disordered eating

symptoms in late adrenarche, suggesting earlier activation of genetic influences may increase later behavioral risk. Study 2 was the first longitudinal study investigating how initial levels and change in neighborhood disadvantage shape trajectories of disordered eating across the critical adolescent risk period in girls and boys. Both girls and boys who lived in more disadvantaged neighborhoods in middle childhood showed greater contemporaneous disordered eating, and this elevation relative to their more advantaged peers persisted into emerging adulthood even for youth who later transitioned to more advantaged neighborhoods. Study 3 examined lower neighborhood resource availability (e.g., recreational centers, green spaces, medical facilities) and exposure to community violence as potential "active ingredients" of associations between neighborhood disadvantage and disordered eating both cross-sectionally in middle childhood and mid/late adolescence and longitudinally across development. Greater community violence exposure was associated with greater contemporaneous disordered eating in girls and boys in both childhood and adolescence, and greater community violence exposure in childhood indirectly predicted greater disordered eating in adolescence through its association with childhood disordered eating. Although not associated with disordered eating cross-sectionally, fewer neighborhood resources in childhood predicted a greater increase in disordered eating from childhood to adolescence. Altogether, findings highlight the importance of considering broader contextual forms of disadvantage in etiologic models of disordered eating. Results also suggest early disadvantage during childhood may have a particularly profound and enduring impact on disordered eating risk, underscoring the importance of early screening and intervention for disadvantaged youth. Finally, findings point to the importance of public policy to increase access to care for under-resourced youth and help shape communities that support positive youth development.

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GENERAL INTRODUCTION

Socioeconomic disadvantage in childhood and adolescence is a significant risk factor for numerous forms of psychopathology (Reiss, 2013), including depression and anxiety (Najman et al., 2010; Ridley et al., 2020; Simanek et al., 2021), conduct problems (Goodnight et al., 2012), and co-occurring mental illness and substance abuse (Salom et al., 2014). The detrimental effects of poverty on mental health may be mediated by the considerable stress associated with living in a disadvantaged context (DeCarlo Santiago et al., 2011; Goodman et al., 2005). Disadvantaged youth are exposed to higher levels of community violence (Kohen et al., 2008; Overstreet, 2000) and adverse childhood events (Lacey et al., 2020), among other significant stressors that may have a lasting impact on the mind, brain, and body (Blair & Raver, 2016; Shields, 2017). In addition to elevated risk for mental health concerns, people living in disadvantaged contexts face substantial barriers to accessing mental health care (Newacheck et al., 2003; Sonneville & Lipson, 2018; Steele et al., 2007), including financial and cost-related constraints in countries such as the United States that do not have universal health coverage (Sareen et al., 2007).

Despite robust evidence for the adverse impacts of disadvantage on mental health overall, research has only recently begun to examine associations between disadvantage and eating disorders (EDs, including anorexia nervosa [AN], bulimia nervosa [BN], binge-eating disorder [BED], and other specified feeding and eating disorders [OSFED]) or disordered eating (e.g., binge eating, body dissatisfaction). The relative dearth of research on disadvantage and EDs may reflect historical stereotypes suggesting that EDs are more common among relatively advantaged individuals (Gard & Freeman, 1996). However, results from recent studies are consistent with the broader literature on disadvantage and psychopathology in showing greater rates of EDs and disordered eating symptoms (e.g., weight/shape dissatisfaction, loss of control over eating) among individuals experiencing varied forms of disadvantage, including food insecurity (Becker et al.,

2017, 2019; Coffino et al., 2020; Lydecker et al., 2019), lower family socioeconomic status (Mikhail et al., 2021, 2023), and neighborhood poverty (Mikhail et al., 2021, 2023). Rather than experiencing elevated rates of EDs, people from relatively advantaged backgrounds may have been overrepresented in earlier research focused on clinical populations due to greater access to care for better resourced individuals (Gard & Freeman, 1996; Huryk et al., 2021; Sonneville & Lipson, 2018). An unfortunate implication is that people living in disadvantaged contexts may not only have greater rates of disordered eating, but are also currently marginalized in treatment, empirical research, and theoretical models of EDs (Mikhail & Klump, 2020).

Nevertheless, much remains unknown regarding associations between disadvantage and disordered eating. Most studies to date have focused on more narrow or proximal measures of disadvantage, such as food insecurity. Disadvantage is a multilayered construct that may have unique effects at different levels of proximity. In particular, there is robust evidence that neighborhood disadvantage (e.g., elevated poverty and reduced community resources in the area surrounding a person's home; Attar et al., 1994) significantly predicts multiple forms of psychopathology above and beyond individual/family level factors (e.g., anxiety/depression, externalizing disorders; Burt et al., 2015; Choi et al., 2021). Although neighborhood disadvantage is correlated with familial socioeconomic status (*r*'s ~ .3 to .5 with family income; Hackman et al., 2012; Roubinov et al., 2018), it is a distinct process that has unique and significant effects on mental health (Burt, 2014).

There has also been a paucity of research examining developmental changes in the impact of disadvantage or the longitudinal effects of disadvantage across the critical adolescent risk period for emergence of disordered eating. Research on other physical and mental health conditions suggests disadvantage early in life (i.e., during childhood) may have particularly robust

and persistent impacts on later health (Poulton et al., 2002; Seabrook & Avison, 2012), partially due to epigenetic changes that may be more challenging to reverse later in development (Chu et al., 2018; Loucks et al., 2016; Shields, 2017). However, the majority of research on disordered eating and disadvantage to date has focused on older adolescents and adults (e.g., Becker et al., 2017, 2019; Lydecker et al., 2019; Simone, Slaughter-Acey, et al., 2022), and little is known about how childhood disadvantage may contribute to later disordered eating. Relatedly, only one study has examined how disadvantage may interact with pubertal development to impact disordered eating risk (Mikhail et al., 2021). This is an important gap in the literature because puberty is a critical developmental stage for the emergence of disordered eating during which genetic influences come online and rates of disordered eating and EDs increase substantially (particularly for girls; Klump, 2013; Nagl et al., 2016).

The studies included in this dissertation sought to significantly expand our understanding of disordered eating in socioeconomically disadvantaged youth, with a particular focus on the under researched neighborhood context. These questions were addressed in large samples of youth representative of the state of Michigan (in study 1) or oversampled for neighborhood disadvantage (in studies 2 and 3) to circumvent the potential biases of clinical samples that may underrepresent disadvantaged young people. Analyses incorporated a deep phenotyping of disadvantage, investigation of longitudinal effects across the critical adolescent risk period, and interactions with genetic risk. All studies included youth across the full range of adolescent development (i.e., from pre-puberty/middle childhood to post-puberty/late adolescence) to assess potential changes in the impact of disadvantage across this crucial developmental period.

Study 1 expanded on a recently published paper examining how disadvantage may interact with underlying genetic vulnerabilities to impact disordered eating in youth via genotype x

environment effects. In a genotype x environment interaction, the influence of latent genetic risk on disordered eating (or another phenotype) depends on the presence of contextual factors that may amplify or obscure underlying genetic predispositions. In Mikhail et al. (2021), we found that girls living in more disadvantaged neighborhoods or families not only had higher phenotypic levels of disordered eating symptoms, but also experienced stronger and earlier expression of genetic influences on disordered eating than girls living in more advantaged contexts. However, one significant shortcoming of Mikhail et al. (2021) was that analyses only examined female twins. This is a notable limitation because boys experience different developmental patterns of ED risk across adolescence (e.g., different timing of activation of genetic influences, substantially lower risk of developing an ED post-puberty; Culbert et al., 2017; Klump, 2013; Nagl et al., 2016), such that findings in girls cannot automatically be generalized to boys. Study 1 was the first to examine phenotypic and genotype x environment associations between neighborhood and familial disadvantage and disordered eating in boys ages 8-17 (N = 3,484), as well as how these associations may differ across adrenarche (i.e., an early stage of puberty charactered by rising adrenal androgens; Auchus & Rainey, 2004), when genetic influences on disordered eating come online in boys.

Study 2 sought to address another substantial gap in the literature on disadvantage and EDs; namely, that no studies have yet examined longitudinal associations between disadvantage and disordered eating from childhood into later adolescence/emerging adulthood. Longitudinal research can provide unique insight into the timing and potential causality of disadvantage effects, as well as how changes in disadvantage over time (e.g., moving to a more affluent neighborhood) may impact developmental trajectories of disordered eating. Study 2 analyzed a large sample of girls and boys (N = 2,060 at time 1) oversampled for neighborhood disadvantage who were

assessed up to three times between the ages of 6 and 21. Multilevel growth curve models were used to examine how initial levels and change in neighborhood disadvantage impacted disordered eating trajectories from middle childhood into emerging adulthood in girls and boys. Crucially, this was the first study to longitudinally examine associations between childhood disadvantage and disordered eating, providing a significant methodological advance over earlier research that has relied on retrospective reports of childhood disadvantage provided in adulthood (e.g., Coffino et al., 2020).

Finally, study 3 sought to identify "active ingredients" of disadvantaged neighborhoods that might contribute most strongly to associations between disadvantage and disordered eating. Understanding these active ingredients is critical for advancing etiologic models of risk in disadvantaged populations and identifying points for intervention. Drawing from the same dataset as study 2, we examined neighborhood resource availability (e.g., presence of parks, schools, medical facilities, and other community resources) as reported by neighbor informants and youth-reported community violence exposure as cross-sectional and longitudinal predictors of disordered eating from middle childhood to mid/late adolescence. This was the first study to identify specific aspects of disadvantaged neighborhoods that may longitudinally predict disordered eating from childhood into the critical adolescent risk period for developing EDs.

Taken together, these studies have the potential to substantially expand our understanding of EDs and disordered eating in disadvantaged youth, with potential implications for screening, treatment, and public policy.

CHAPTER 1: DISADVANTAGE AND DISORDERED EATING IN BOYS: EXAMINING PHENOTYPIC AND GENOTYPE X ENVIRONMENT ASSOCIATIONS ACROSS DEVELOPMENT

Abstract

Background: Socioeconomic disadvantage may be a significant risk factor for disordered eating, particularly for individuals with underlying genetic risk. However, little-to-nothing is known about the impact of disadvantage on disordered eating in boys during the critical developmental risk period. Crucially, risk models developed for girls may not necessarily apply to boys, as boys show different developmental patterns of disordered eating risk (i.e., earlier activation of genetic influences during adrenarche, an early stage of puberty). This is the first study to examine phenotypic and genotype x environment (GxE) effects of disadvantage in boys. **Methods:** Analyses examined 3,484 male twins ages 8-17 (M_{age} = 12.27, SD = 2.96) from the Michigan State University Twin Registry. Disordered eating (e.g., body dissatisfaction, binge eating) was measured using the parent-report Michigan Twins Project Eating Disorder Survey. Neighborhood disadvantage was measured using a census-tract level Area Deprivation Index, and family socioeconomic status was determined from parental income and education. Adrenarche status was determined using multiple indicators, including age and Pubertal Development Scale scores. **Results:** GxE models suggested that genetic influences on disordered eating were activated earlier for boys experiencing familial or neighborhood disadvantage, with substantial genetic influences in early adrenarche, when genetic influences were low in more advantaged boys. Phenotypically, both neighborhood and familial disadvantage were associated with greater disordered eating for boys in late adrenarche, which could indicate a lasting impact of earlier activation of genetic influences on later risk. **Conclusions:** Results highlight disadvantage as a novel risk factor for disordered eating in boys, particularly those with genetic vulnerabilities.

Introduction

Despite historical stereotypes that eating disorders (EDs) primarily impact individuals from relatively advantaged backgrounds (Gard & Freeman, 1996), recent research suggests risk for EDs and related symptoms may be elevated among people experiencing socioeconomic disadvantage. While relatively few studies have examined the association between disadvantage and disordered eating, increased disordered eating among disadvantaged populations has been found in both girls and adults across multiple indicators of disadvantage, including food insecurity, neighborhood disadvantage (i.e., increased neighborhood poverty and decreased community resources), and familial disadvantage (i.e., lower household income and educational attainment) (Becker et al., 2017, 2019; Coffino et al., 2020; Hazzard et al., 2021; Lydecker et al., 2019; Mikhail, Carroll, et al., 2021). Though people from disadvantaged backgrounds are underrepresented in research and treatment settings, this disparity appears to reflect reduced access to care rather than the prevalence of EDs in the general population (Gard & Freeman, 1996; Huryk et al., 2021; Sonneville & Lipson, 2018). Preliminary studies linking disadvantage to disordered eating suggest an urgent need for additional research examining disordered eating in socioeconomically disadvantaged populations, including how the etiology of disordered eating may be similar or different for people from disadvantaged backgrounds.

There are several mechanisms through which disadvantage may increase disordered eating, including increased stress (DeCarlo Santiago et al., 2011; Goodman et al., 2005), reduced access to fresh foods such as fruits/vegetables and increased availability of highly palatable foods (e.g., fast food; Cooksey-Stowers et al., 2017; Dubowitz et al., 2012), and increased weight stigma among disadvantaged populations (Becker et al., 2021). The impact of these environmental risk factors may be further amplified in individuals with underlying genetic risk via

genotype x environment interactions (GxE). When GxE is present, the impact of latent genetic risk on a behavioral phenotype depends on the presence of environmental stressors. In some cases, genetic influences may be weaker in stressful circumstances that impede normative development (i.e., bioecological GxE; Bronfenbrenner & Ceci, 1994; Burt, 2014). Alternatively, and more commonly for internalizing phenotypes such as disordered eating (e.g., Fairweather-Schmidt & Wade, 2017; Strachan et al., 2017), stressful environmental circumstances amplify underlying genetic vulnerabilities, leading to elevated psychopathology in individuals with genetic risk (i.e., diathesis-stress GxE; Rende & Plomin, 1992).

Initial research suggests the impact of disadvantage on disordered eating may be amplified for individuals with underlying genetic vulnerabilities through diathesis-stress GxE, particularly during puberty, a developmentally sensitive risk period for the emergence of EDs (e.g., Mikhail, Anaya, et al., 2021; Nagl et al., 2016). In a recent study, our group found that phenotypic ED symptoms were greater for girls experiencing familial or neighborhood disadvantage. In addition, both forms of disadvantage were associated with stronger and earlier expression of genetic influences on disordered eating (Mikhail, Carroll, et al., 2021). Though disordered eating is strongly heritable in adulthood (with ~50% of variance in disordered eating due to genetic factors), girls from more advantaged backgrounds typically show minimal genetic influences on disordered eating prior to mid-puberty (Klump et al., 2003, 2007, 2012; O'Connor et al., 2020). However, genetic influences on disordered eating were already substantial in girls from the most disadvantaged backgrounds in pre/early puberty, suggesting much earlier expression of genetic risk in disadvantaged contexts that could ultimately lead to more disordered eating (Mikhail, Carroll, et al., 2021). The considerable stress accompanying disadvantage may exacerbate genetically-based individual differences in the stress response or emotional reactivity (Gillespie et

al., 2009), potentiating earlier expression of genetic risk for disordered eating. It is notable that effects were largely consistent across neighborhood and familial disadvantage, which are conceptually and empirically distinct (*r*'s ~ .3 to .5; Hackman et al., 2012; Mikhail, Carroll, et al., 2021; Roubinov et al., 2018), suggesting that multiple forms of disadvantage (both more proximal and distal) are associated with increased ED risk in girls.

Importantly, research to date has focused on the impact of disadvantage on disordered eating in girls (e.g., Mikhail, Carroll, et al., 2021) or adults (e.g., Becker et al., 2017, 2019; Hazzard et al., 2021; Lydecker et al., 2019), with no studies of disadvantage effects in boys during the critical developmental risk period. While disordered eating is less common in boys than girls, a significant number of boys and men do experience EDs and related symptoms (e.g., binge eating), with recent estimates indicating that over 10% of adolescent boys experience clinically significant disordered eating (Nagata et al., 2020). Disordered eating may be even more common among boys and men experiencing significant stress (Gadalla, 2009; Mitchell et al., 2016), potentially including those living in disadvantaged environments, and preliminary research suggests that food insecurity (Becker et al., 2017, 2019) and lower SES (Burke et al., 2022) are similarly associated with disordered eating in adult men and women. Notably, boys and men are less likely than girls and women to be diagnosed or receive treatment for EDs even when experiencing significant symptoms (Sonneville & Lipson, 2018). It is therefore critical to identify boys at increased risk for targeted prevention and intervention.

Crucially, boys experience different developmental patterns of ED risk than girls, and developmentally sensitive risk models based on girls (including analyses in Mikhail, Carroll, et al. (2021) discussed above) may not necessarily apply to boys. Specifically, the developmental timing of activation of genetic influences on ED risk differs across sex. Puberty can be divided

into two developmental stages: adrenarche, during which adrenal androgens (e.g., androstenedione, dehydroepiandrosterone, dehydroepiandrosterone-sulphate [DHEA-S]) increase prior to pronounced outward physical changes, and gonadarche, during which increases in gonadal hormones (e.g., estradiol, testosterone) drive the development of secondary sex characteristics (e.g., breast growth, voice changes) (Auchus & Rainey, 2003). Adrenarche typically begins before gonadarche (~age 6-8) and continues through gonadarcheal development (Guran et al., 2015). Girls do not show genetic influences on disordered eating until mid-gonadarche, well after adrenarche is underway (Klump et al., 2003, 2007, 2012; O'Connor et al., 2020). However, in boys, genetic influences start to increase during the early stages of adrenarche that precede gonadarche and are fully online when gonadarche begins (Culbert et al, 2017). Genetic influences on disordered eating may be activated in males but not females during adrenarche because males display greater sensitivity to androgens following greater exposure to testosterone prenatally, leading to unique impacts of androgens on later gene expression in males (Arnold, 2009). If disadvantage impacts disordered eating in part by leading to earlier expression of genetic risk, these developmentally sensitive effects would be expected to unfold earlier in boys than girls (i.e., in adrenarche rather than gonadarche) and could reflect potentially distinct underlying molecular mechanisms (i.e., activation by androgens rather than estrogen). It is therefore crucial to examine boys independently rather than assuming that disadvantage effects during adolescence are the same in girls and boys.

In this study, we examined whether boys living in more socioeconomically disadvantaged circumstances were at elevated risk for disordered eating. We examined both family SES and neighborhood disadvantage to investigate potential similarities and differences in the impact of disadvantage at different levels of proximity. Notably, prior research suggests that activation of

genetic influences during adrenarche/puberty may lead to lasting changes in neural organization that precede behavioral changes (Klump et al., 2018; Schulz & Sisk, 2016). If disadvantage impacts disordered eating in part through changes in gene expression that alter brain organization during adrenarche, we might expect significant GxE (i.e., elevated genetic influences on disordered eating with increasing disadvantage) in early adrenarche, but minimal phenotypic effects until late adrenarche. Conversely, we would expect smaller GxE effects (i.e., similar levels of genetic influence across disadvantage) during late adrenarche after the period of organization has ended, but greater phenotypic effects. Moderation analyses across adrenarche allowed us to examine these hypotheses regarding developmental shifts in disadvantage effects.

Methods

Participants

Primary analyses included 3,484 boys ages 8-17 (M_{age} = 12.27, *SD* = 2.96) from same-sex twin pairs from the Michigan Twins Project (MTP), a large-scale twin registry that serves as a recruitment pool for research conducted through the Michigan State University Twin Registry (MSUTR). The MSUTR is a population-based twin registry that recruits twins through birth records in collaboration with the Michigan Department of Health and Human Services (see Burt & Klump, 2013, 2019; Klump & Burt, 2006). Response rates for the MTP are similar or better than those of other twin registries (58.9% for youth under 18) and MTP twins are demographically representative of Michigan (Burt & Klump, 2019). Approximately 14% of MTP youth live in families whose income is at or below the federal poverty level (~$26,500 for a family of four; US Department of Health and Human Services, 2021), which is similar to the overall population of Michigan (Burt & Klump, 2013).

Most participants identified as white/non-Latinx (*n* = 2,948; 84.6%), followed by

Black/non-Latinx (n = 248; 7.1%), multiracial (n = 124; 3.6%), Latinx (n = 48; 1.4%), Asian American (n = 38; 1.1%), and Native American (n = 10; 0.3%). The remaining participants (n = 68; 2.0%) identified as belonging to another race/ethnicity or did not specify their race/ethnicity. Twins varied widely in family SES (combined parental income M = \$90,390, SD = \$54,410, range = \$0-\$300,000+). Similar to our prior report examining girls from the MSUTR (Mikhail, Carroll, et al., 2021), 10.9% of participants lived in neighborhoods above the national 75th percentile for disadvantage. Additional demographic information is shown in Table S1.1.

Measures

Zygosity Determination

Zygosity was determined using a well-validated physical similarity questionnaire (Lykken et al., 1990) completed by the twins' parents. This questionnaire is over 95% accurate in determining zygosity as verified through DNA/serologic testing (Lykken et al., 1990; Peeters et al., 1998).

Disordered Eating

Disordered eating was assessed using the Michigan Twins Project Eating Disorder Survey (MTP-ED; Mikhail, Carroll, et al., 2021), a nine-item parent-report questionnaire for measuring disordered eating in population-based samples. Prior research suggests parent-reported symptoms differentiate youth with and without clinical EDs (Accurso & Waller, 2021) and show similar or greater concordance with objective external measurements (e.g., BMI, clinician-reported symptoms) as adolescent-reported symptoms (Couturier et al., 2007; Steinberg et al., 2004; Swanson et al., 2014). Parent report may be particularly useful for younger boys who may have difficulty understanding disordered eating items.

The MTP-ED contains questions regarding body dissatisfaction (i.e., distress regarding

body shape), weight preoccupation (i.e., fear of gaining weight), and disordered eating behaviors (i.e., dieting, binge eating, purging). Each item is rated on a 3-point scale from 0 (not true) to 2 (certainly true). Detailed information on the reliability/validity of the MTP-ED in boys is included in Supplemental Material. In brief, in the current sample, the MTP-ED had acceptable internal consistency across age (ages 8-12: $\alpha = .77$; ages 13-17; $\alpha = .81$) and pubertal development (early adrenarche: $\alpha = .70$; early gonadarche: $\alpha = .78$; mid/late gonadarche: $\alpha = .80$), discriminated between boys with and without a parent-reported ED ($d = 1.24$, $p < .001$), and showed expected correlations with other constructs (e.g., $r = .29$, $p < .001$ with BMI; $r = .25$, $p < .001$ with internalizing symptoms).

Additional validation of the MTP-ED was conducted in 299 boys ages 7-18 and their primary caregivers from a separate, ongoing study within the MSUTR. Correlations in this independent sample were large between self-reported MTP-ED and self-reported Minnesota Eating Behavior Survey[1] (MEBS; von Ranson et al., 2005) total scores ($r = .66$, $p < .001$). As is typical in the ED literature, correlations between parent- and self-reported MTP-ED were significant but small-to-moderate in magnitude ($r = .26$, $p < .001$).

Disadvantage

Neighborhood disadvantage was measured using a well-validated (Kind & Buckingham, 2013; Singh, 2003), census-tract level Area Deprivation Index (ADI) incorporating 17 indicators of neighborhood disadvantage (e.g., unemployment rate, median home value). The ADI has been used to examine associations between neighborhood disadvantage and numerous mental and

[1] The Minnesota Eating Behavior Survey (previously known as the Minnesota Eating Disorder Inventory [M-EDI]) was adapted and reproduced by special permission of Psychological Assessment Resources, 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources. Further reproduction is prohibited without prior permission from Psychological Assessment Resources.

physical health outcomes in prior work (Burt et al., 2020; Carroll et al., 2021; Kind et al., 2014; Powell et al., 2020; Suarez et al., 2022), including our previous report on disadvantage and disordered eating in girls (Mikhail, Carroll, et al., 2021). Neighborhood disadvantage, as measured by the ADI, is correlated with poorer physical (Kind et al., 2014; Powell et al., 2020) and mental (Carroll et al., 2021; Burt et al., 2020) health, as well as higher BMI (Sheets et al., 2020) and lower physical activity (Miller et al., 2020). The ADI also has excellent internal consistency ($\alpha = .95$ in past research; Singh, 2003). The ADI score for each family was coded using publicly available data from the American Community Survey for the census-tract containing the family's address (https://www.neighborhoodatlas.medicine.wisc.edu/). Raw ADI scores were converted into percentiles relative to other families in the sample, with higher scores indicating greater neighborhood disadvantage.

Family SES was measured using a latent variable factor score incorporating mother's education level, father's education level, and combined parental income. As with the ADI, raw scores were converted into percentiles relative to other families in the sample. To maintain consistency with Mikhail, Carroll, et al. (2021), family SES was coded such that *lower* scores (i.e., lower family SES) indicate *greater* disadvantage. Importantly, neighborhood disadvantage and family SES are only moderately correlated ($r = -.47$ in the current study; only 22% variance shared), indicating that they are related but distinct (Burt, 2014; Hackman et al., 2012; Roubinov et al., 2018).

Adrenarche

Because adrenal androgens were not directly measured, age and gonadarche were used as proxy indicators of adrenarche status based on earlier research on changes in the etiology of disordered eating across adrenarche and gonadarche (Culbert et al., 2017). Gonadarche was

measured using the parent-report Pubertal Development Scale (PDS; Peterson et al., 1988), a five-item questionnaire that assesses physical markers of maturation during gonadarche. Parent-rated PDS correlates strongly with professionally rated Tanner staging and shows good psychometric properties for boys (α = .96; Koopman-Verhoeff et al., 2020). Items for boys include height changes, skin changes, body hair growth, voice deepening, and facial hair growth. Each item is rated from 1 (not yet begun) to 4 (seems completed). As in past research (Klump et al., 2003, 2012), the five items were averaged to create an overall PDS score.

We divided participants into early and late adrenarche groups based on research indicating that genetic influences on disordered eating begin to gradually increase during the period of adrenarche preceding gonadarche (i.e., early adrenarche) and are fully online when gonadarche begins (i.e., late adrenarche) (Culbert et al., 2017). In other words, the period of adrenarche preceding gonadarche onset is critical for activation of genetic influences on disordered eating in boys. Developmental studies indicate most boys begin adrenarche based on adrenal androgen levels by age 8 (i.e., the youngest age in our sample) (Guran et al., 2015; Ilondo et al., 1982). Therefore, we categorized all participants aged 12 or younger with a PDS score of 1 (i.e., no external indicators of gonadarche) as in early adrenarche (n = 495; 14.2%). Participants with a PDS score greater >1 (n = 2,723; 78.2%) or who were 13 or older and missing data on the PDS (n = 118; 3.4%) were categorized as in late adrenarche. We used a cutoff age of 13 as a proxy indicator of being in late adrenarche based on prior research indicating that over 95% of boys show evidence of gonadarcheal development (e.g., increase in testicular volume) by age 13 (Bundak et al., 2007). A small number of boys who were 13 but had a PDS score of 1 (n = 6; 0.2%) were also categorized as being in late adrenarche, which was a conservative decision in relation to our hypotheses (i.e., the difference between boys in early and late adrenarche would be

reduced if these boys were in fact in early adrenarche). Adrenarche status for the remaining 142 participants (4.1%) could not be determined because they were under age 13 and missing data on the PDS.

BMI Percentile

Age- and sex-specific BMI percentiles were calculated from parent-reported height and weight using CDC growth charts (https://www.cdc.gov/healthyweight/xls/bmi-group-calculator-us-062018-508.xlsm). Parent-reported BMI shows good concordance with measured BMI in youth, with parent-reported weight estimates deviating from measured weights by <5 pounds (Gordon & Mellor, 2015; Shields et al., 2011).

Statistical Analyses

Data Preparation

MTP-ED scores were prorated if one item was missing and marked as missing if >1 item was missing. While parent-reported BMI shows good concordance with objective measures (Gordon & Mellor, 2015; Shields et al., 2011), following Mikhail, Carroll, et al. (2021), we took a conservative approach in setting extreme BMI values <0.5th percentile or >99.5th percentile to missing. MTP-ED scores were log transformed to account for positive skew and standardized. More disadvantaged youth tend to have higher BMIs (Alvarado, 2016), and higher BMIs are associated with disordered eating (Neumark-Sztainer et al., 2007). All phenotypic and GxE analyses were therefore conducted with and without BMI percentile to directly assess its impact on results.

Phenotypic Analyses

Multilevel models (MLMs) with a random intercept to account for nesting of twins within families were used to examine phenotypic associations between disadvantage and disordered

eating. Random slopes were not estimated due to the small number of observations per group (i.e., two twins per family). Models used an identity covariance structure and maximum likelihood estimation, which makes use of all available data to produce relatively unbiased parameter estimates (Black et al., 2011). Continuous variables were z-scored. Race/ethnicity was included as a covariate because people of color are disproportionately likely to live in disadvantaged contexts due to histories of discrimination (e.g., redlining; Woods, 2012), and are also more likely to face stressors such as racism and prejudice that may increase risk for disordered eating (Mikhail & Klump, 2020). Models examined adrenarche status (coded dichotomously as 0 = early adrenarche, 1 = late adrenarche) as a moderator to examine whether phenotypic associations between disadvantage and disordered eating differ across adrenarche in boys.

GxE Analyses

Extended univariate, double moderator twin models (van der Sluis et al., 2012) were used to examine how genetic and environmental influences on disordered eating differ across disadvantage in boys, and whether these GxE effects depend on developmental stage. The double moderator twin model is depicted in Figure S1.1. This model examines additive genetic (A; i.e., genetic influences that sum across genes), shared environmental (C; i.e., environmental factors that increase similarity between co-twins, such as attending the same school), and non-shared environmental (E; i.e., environmental factors that differentiate twins raised in the same family, such as non-overlapping friend groups) influences on disordered eating, and how these influences differ across disadvantage and adrenarche. The van der Sluis (2012) model allowed us to include twins who were discordant on adrenarche status while correcting for potential biases in significance testing resulting from the correlation between adrenarche and disordered eating. All twins were concordant on disadvantage variables, as these were measured at the family level.

Because moderators are included in the means model, A, C, and E reflect the etiology of disordered eating after regressing out variance shared with the moderators. Double moderator twin models include 12 major parameters of interest: 3 initial path coefficients (a, c, e in Figure S1.1) that capture genetic/environmental influences at the lowest level of the moderators (i.e., among the least disadvantaged boys in early adrenarche), and 9 moderation coefficients that capture linear increases/decreases in the initial ACE path coefficients as a function of developmental stage (βxP, βyP, βzP in Figure S1.1), disadvantage (βxD, βyD, βzD in Figure S1.1), and their interaction (βxPD, βyPD, βzPD in Figure S1.1). Quadratic moderators were not included because the data suggested only linear effects were present. This approach is consistent with our earlier study in female twins (Mikhail, Carroll, et al., 2021), and also helps to conserve power and enhance interpretability.

The full model was fit first, with all path estimates and moderators freely estimated. Submodels were then fit based on the full model parameter estimates and confidence intervals to identify a best-fitting model. This approach allowed for identification of relevant submodels without conducting an excessive number of tests, as each model has numerous possible submodels. Best-fitting models were identified as those that had a non-significant difference in minus twice the log-likelihood (−2lnL) between the full and nested model, and minimized Akaike's Information Criterion (AIC), Bayesian Information Criterion (BIC), and sample-size adjusted BIC (SABIC). If AIC, BIC, and SABIC identified different models as best-fitting, the model that optimized two out of three fit indices was selected as best-fitting.

BMI percentile was regressed out of log-transformed MTP-ED total scores, and the resulting residuals were standardized. Neighborhood disadvantage and family SES percentiles were floored at 0, then scaled from 0-1 for interpretability. Adrenarche was coded dichotomously

(0 = early adrenarche, 1 = late adrenarche). Following prior recommendations for twin moderation models (Purcell, 2002), tables and figures report unstandardized path coefficient and moderation estimates. Unstandardized estimates are generally recommended because they reflect absolute differences in genetic/environmental influences across the moderators, while standardized estimates only capture differences in proportions of the total variance. However, standardized estimates are also reported where appropriate to facilitate interpretability.

Transparency and Openness

Data, analysis code, and research materials are available from the corresponding author upon reasonable request. This study was not preregistered.

Results

Sample Descriptives

A range of disordered eating symptoms was represented (MTP-ED score range = 0-15; possible range = 0-18), including more severe ED behaviors such as binge eating (8.5% of the sample). As expected, boys displayed greater disordered eating symptoms in late adrenarche than in early adrenarche (p <.001, d = .32). Disordered eating symptoms were also significantly associated with both neighborhood disadvantage (r = .10, p <.001) and family SES (r = -.11, p <.001) with a small effect size when examined using Pearson correlations. Importantly, relatively modest phenotypic associations between disadvantage and disordered eating do not preclude GxE, and in fact may reflect the presence of significant moderation (e.g., stronger associations for individuals with genetic vulnerabilities, and weaker/no association for individuals without genetic risk).

Phenotypic Analyses

In MLMs examining differences in associations between disadvantage and disordered

eating across adrenarche, we observed expected significant main effects of adrenarche and BMI indicating greater disordered eating in boys during late adrenarche and for boys at higher BMI percentiles. We also observed significant or trend-level interactions between adrenarche status and disadvantage for both neighborhood disadvantage and family SES (see Table 1.1). For both neighborhood disadvantage and family SES, interactions indicated that the association between disadvantage and disordered eating was stronger in late adrenarche. Specifically, in the model including BMI percentile as a covariate, the association between neighborhood disadvantage and disordered eating was significant for boys in late adrenarche (β = .08, p = .001, 95% CI [.03, .13]) but not in early adrenarche (β = -.03, p = .640, 95% CI [-.14, .09]). Similarly, when controlling for BMI, family SES was significantly associated with disordered eating for boys in late adrenarche (β = -.08, p = .001, 95% CI [-.12, -.03]), but not in early adrenarche (β = .05, p = .360, 95% CI [-.05, .15]). Results were similar (but with slightly larger effect sizes for boys in late adrenarche) in models not including BMI as a covariate. Findings were consistent with the hypothesis that phenotypic associations between disadvantage and disordered eating may be greatest in late adrenarche, following GxE during early adrenarche.

GxE Analyses

As shown in Supplemental Material (Tables S1.3-S1.4 and Figure S1.2), GxE analyses yielded very similar results with and without BMI percentile regressed out of the MTP-ED total score for family SES. However, the full GxE model of neighborhood disadvantage that did not control for BMI failed to converge, although cotwin correlations suggested a similar pattern of effects as the model that did control for BMI (see Table S1.2). Results below therefore focus on models that controlled for BMI.

For both neighborhood disadvantage and family SES, genetic influences on disordered

eating appeared to differ across disadvantage and adrenarche in the full model (see Figures 1.1 and 1.2). Specifically, for boys living in more advantaged contexts (low ADI or high family SES), genetic influences appeared substantially greater during late adrenarche than in early adrenarche. This pattern of results is consistent with previous findings suggesting greater genetic influences on disordered eating in late adrenarche in relatively advantaged boys (Culbert et al., 2017). However, for boys living in more disadvantaged circumstances (high ADI or low family SES), genetic influences on disordered eating appeared at least as large in early adrenarche as in late adrenarche. Differences in environmental influences across disadvantage and adrenarche appeared less pronounced than moderation of genetic effects in these models.

With respect to model fitting, no moderation models fit poorly for both neighborhood disadvantage and family SES, suggesting significant moderation effects (see Table 1.2). The best-fitting models for both neighborhood disadvantage and family SES retained disadvantage x adrenarche moderation of the A parameter, such that genetic influences on disordered eating were greater in late adrenarche, but only for boys living in advantaged circumstances (see Tables 1.2-1.3 and Figures 1.1-1.2). For boys in disadvantaged neighborhoods and families, genetic influences were already substantial during early adrenarche. Consequently, the estimated proportion of variance in disordered eating due to genetic factors during early adrenarche was significantly greater for boys from more disadvantaged neighborhoods (low ADI: 19% of variance due to genes; high ADI: >95% of variance due to genes)[2] and families (high SES: 35% of variance due to genes; low SES: 67% of variance due to genes). While some moderation of C

[2]A very high estimated percentage of variance due to genetic factors could reflect non-additive genetic influences. To test this possibility, we ran an additional set of analyses that modeled non-additive genetic influences and dropped shared environmental influences (i.e., ADE models). The best-fitting ADE model fit worse on all fit indices than the best fitting ACE model, suggesting that non-additive genetic influences are not a major contributor to observed effects.

and E parameters was also retained in the best-fitting models, these effects appeared relatively modest when plotted, particularly for family SES (see Figures 1.1 and 1.2). Overall, effects were consistent with the hypothesis that disadvantage may potentiate earlier expression of genetic influences on disordered eating during early adrenarche through GxE.

Discussion

This is the first study to examine phenotypic and GxE associations between multiple forms of disadvantage and disordered eating in boys, substantially extending our understanding of how disadvantage may impact disordered eating in youth. Both neighborhood disadvantage and lower family SES were associated with significantly greater phenotypic disordered eating symptoms in boys beginning in late adrenarche. Notably, effects remained significant even after controlling for BMI, indicating that the association between disadvantage and disordered eating in boys cannot be solely attributed to increased body weight and attendant weight stigma in disadvantaged environments. GxE analyses showed substantially stronger and earlier activation of genetic influences on disordered eating for boys living in disadvantaged environments during early adrenarche, when genetic influences were modest in more advantaged boys. This earlier activation of genetic influences could contribute to greater phenotypic ED symptoms later in development, reflecting a potentially lasting impact of disadvantage on ED risk in boys. Findings are novel in highlighting disadvantage as a significant risk factor for disordered eating in boys, perhaps especially for those with underlying genetic vulnerabilities.

Prior research indicates that adrenarche is a critical period for activation of genetic influences on disordered eating in relatively advantaged boys, with genetic influences increasing gradually across early adrenarche (i.e., prior to gonadarche), then remaining constant from late adrenarche/gonadarche into adulthood (Culbert et al., 2017; Klump et al., 2012). We replicated

these prior findings for boys from relatively advantaged neighborhoods and families, who showed a precipitous increase in genetic influences from early adrenarche to late adrenarche. However, for boys living in more disadvantaged circumstances, GxE analyses indicated that genetic influences on disordered eating were already substantial in early adrenarche, suggesting earlier activation of genetic influences that could increase later risk. Importantly, genetic influences did not differ across disadvantage during late adrenarche, consistent with a shift in the developmental timing of expression of genetic risk in disadvantaged environments rather than a general increase in genetic influences regardless of developmental stage. Although GxE effects emerged during early adrenarche, phenotypic associations between disadvantage and disordered eating were not apparent until late adrenarche. This pattern of effects (increased genetic activation followed by later phenotypic expression) may reflect alterations to developing neurocircuitry during key hormonal/developmental periods that have enduring effects on later behavior (i.e., organizational hormone effects; Schulz & Sisk, 2016). Similar potentially organizational impacts of risk factors for EDs during puberty have been observed previously in girls and female animals (e.g., Klump et al., 2018).

Both familial and neighborhood disadvantage are accompanied by considerable stress (e.g., stemming from financial instability, food insecurity, noise pollution, community violence, etc.) that could potentiate expression of genes relevant to vulnerability for disordered eating earlier than developmentally normative. Effects during adrenarche may involve interactions between rising androgen levels and the physiological stress response that could together lead to changes in gene expression and amplification of risk. Consistent with this possibility, a robust body of literature indicates that stress can alter gene expression and brain organization in neural circuits relevant to disordered eating (e.g., regions in the amygdala and prefrontal cortex involved

in inhibitory control and emotion regulation; McEwen, 2013), and that androgens regulate the stress response and downstream physiological changes in males (Zuloaga et al., 2020). Relatedly, stress has been shown to alter the timing of brain development, promoting earlier maturation of emotion-related circuits that may be adaptive in the short-term, but have more deleterious long-term repercussions for coping with stress and negative affect (Callaghan & Tottenham, 2016). This "stress acceleration hypothesis" is consistent with our findings of earlier activation of genetic influences in boys experiencing disadvantage. While stress is associated with increased disordered eating (Gadalla, 2009; Mitchell et al., 2016) and androgens are generally protective against disordered eating in men and boys (Culbert et al., 2014, 2020), no studies have yet examined how androgens and stress may interact to impact ED risk. Additional longitudinal research is needed to identify how the stress accompanying disadvantage may interact with androgens during development to impact gene expression and neural development in boys. Research is also needed to identify which aspects of disadvantage have the greatest impact on ED risk, and whether stressors that directly impact nutritional status (e.g., food insecurity) may have particularly pronounced effects.

This study had several strengths, including a large, population-based sample, multiple measures of disadvantage, and developmentally sensitive analyses. Nevertheless, some limitations should be noted. As in our earlier study of disadvantage effects in girls (Mikhail, Carroll, et al., 2021), we relied on a parent-report measure of disordered eating. Using a consistent outcome measure across studies allows for direct comparison between the current study and Mikhail, Carroll, et al. (2021). Our disordered eating measure also demonstrated strong psychometric properties and expected associations with other key variables (e.g., BMI, puberty, internalizing) in boys. Despite this, EDs are often accompanied by considerable shame and secrecy, and parents

may not be fully aware of all symptoms experienced by youth. Replication with self-reported symptoms is therefore needed. It would also be helpful to examine whether different symptom domains (e.g., binge eating versus body image concerns) relate to disadvantage differently. Interestingly, however, initial research in adults suggests disadvantage may be associated with increases in all types of EDs and their symptoms, rather than only select symptoms (Becker et al., 2019; Coffino et al., 2020). Relatedly, determination of adrenarcheal development relied on indirect measures (i.e., age and outward indicators of gonadarche). Though our method of measuring adrenarche is consistent with past developmental studies of EDs in boys (i.e., Culbert et al., 2017), findings would ideally be replicated using adrenal androgen levels as a more precise, continuous measure of adrenarche.

Analyses examined a population-based sample, rather than a sample enriched for disadvantage. An advantage of this approach is that the full range of disadvantage was present, allowing us to more easily detect differences between youth high and low in disadvantage. Nevertheless, effect sizes may have been larger in a sample specifically enriched for disadvantage, and future research should examine samples with larger numbers of highly disadvantaged youth. Additionally, observed associations were correlational, and causal associations between disadvantage and disordered eating cannot necessarily be inferred. Longitudinal research and research on the "active ingredients" underlying disadvantage effects is needed to continue to expand our understanding of how disadvantage may impact disordered eating for both boys and girls.

Tables

Table 1.1. *MLMs examining associations between disadvantage and disordered eating across adrenarche status*

Neighborhood Disadvantage									
BMI Percentile Not Included as a Covariate					*BMI Percentile Included as a Covariate*				
Variable	**β**	**SE**	***p***	**95% CI**	**Variable**	**β**	**SE**	***p***	**95% CI**
Intercept	-.33	.06	**<.001**	-.44, -.21	Intercept	-.30	.06	**<.001**	-.42, -.19
ADI	-.007	.06	.899	-.12, .11	ADI	-.03	.06	.640	-.14, .09
Adrenarche status	.38	.06	**<.001**	.26, .51	Adrenarche status	.36	.06	**<.001**	.24, .49
ADI x adrenarche	.12	.06	.050	.0001, .24	ADI x adrenarche	.11	.06	.078	-.01, .23
Race/ethnicity					Race/ethnicity				
Black/African American (non-Latinx)	.006	.10	.950	-.18, .19	*Black/African American (non-Latinx)*	-.04	.10	.701	-.24, .16
Latinx/Hispanic	.22	.20	.276	-.18, .62	*Latinx/Hispanic*	.03	.22	.903	-.40, .45
Asian American	.007	.23	.976	-.45, .47	*Asian American*	.06	.24	.806	-.42, .54
Native American/ American Indian	-.13	.50	.789	-1.12, .85	*Native American/ American Indian*	-.21	.48	.657	-1.14, .72
More than one race	-.008	.13	.950	-.25, .24	*More than one race*	.05	.13	.716	-.20, .29
Other/unknown	.37	.16	**.024**	.05, .69	*Other/unknown*	.50	.18	**.005**	.15, .84
					BMI percentile	.29	.02	**<.001**	.24, .33
Family Socioeconomic Status (SES)									
BMI Percentile Not Included as a Covariate					*BMI Percentile Included as a Covariate*				
Variable	**β**	**SE**	***p***	**95% CI**	**Variable**	**β**	**SE**	***p***	**95% CI**
Intercept	-.29	.06	**<.001**	-.40, -.19	Intercept	-.28	.06	**<.001**	-.39, -.17
SES	-.01	.05	.799	-.12, .09	SES	.05	.05	.360	-.05, .15
Adrenarche status	.33	.06	**<.001**	.21, .44	Adrenarche status	.32	.06	**<.001**	.20, .44
SES x adrenarche	-.09	.06	.124	-.20, .02	SES x adrenarche	-.12	.06	**.029**	-.23, -.01
Race/ethnicity					Race/ethnicity				
Black/African American (non-Latinx)	.12	.09	.158	-.05, .30	*Black/African American (non-Latinx)*	.04	.09	.674	-.14, .22
Latinx/Hispanic	.18	.19	.348	-.19, .55	*Latinx/Hispanic*	-.02	.20	.923	-.41, .37
Asian American	.08	.21	.715	-.34, .49	*Asian American*	.08	.22	.709	-.35, .52

Table 1.1 (cont'd)

Native American/ American Indian	-.39	.38	.309	-1.15, .36	*Native American/ American Indian*	-.43	.37	.237	-1.15, .28
More than one race	.03	.12	.778	-.20, .26	*More than one race*	.06	.12	.600	-.17, .29
Other/unknown	.36	.16	**.023**	.05, .68	*Other/unknown*	.50	.18	**.005**	.15, .84
					BMI percentile	.28	.02	**<.001**	.25, .32

Note: MLM = multilevel model; ADI = Area Deprivation Index; adrenarche: 0 = early adrenarche, 1 = late adrenarche; BMI = body mass index. Reference group for race/ethnicity is White. Effects significant at $p < .05$ are bolded.

Table 1.2. *Model fit comparisons for genotype x environment models across adrenarche status and disadvantage*

Model	-2lnL	χ^2 Δ (df)	*p*	AIC	BIC	SABIC
Neighborhood Disadvantage						
Full model	6551.806	—	—	6597.807	6719.546	6646.482
Nested submodels						
No moderation	6665.528	113.722 (9)	<.001	6693.529	6767.631	6723.157
Constrain all C mods	6559.144	7.338 (3)	.062	6599.144	6705.004	6641.471
Constrain all E mods	6600.034	48.228 (3)	<.001	6640.035	6745.895	6682.361
Constrain all A mods	6572.244	20.438 (3)	<.001	6612.243	6718.103	6654.570
Constrain C ADI and ADI x adrenarche mods	6553.568	1.762 (2)	.414	6595.568	6706.721	6640.011
Constrain C ADI and adrenarche mods	6553.760	1.954 (2)	.376	6595.760	6706.913	6640.203
Constrain C ADI and ADI x adrenarche mods, E adrenarche mod	6554.586	2.780 (3)	.427	6594.586	6700.447	6636.913
Constrain C ADI and adrenarche mods, E adrenarche mod	6554.272	2.466 (3)	.481	6594.271	6700.132	6636.598
Constrain C main effect and ADI and ADI x adrenarche mods, E adrenarche mod	6555.156	3.350 (4)	.501	6593.155	6693.723	6633.366
Constrain C main effect and ADI and adrenarche mods, E adrenarche mod	6554.346	2.540 (4)	.637	6592.346	6692.913	6632.556
Family Socioeconomic Status (SES)						
Full model	7410.740	—	—	7456.740	7581.386	7508.318
Nested submodels						
No moderation	7516.248	105.508 (9)	<.001	7544.248	7620.119	7575.643
Constrain all C mods	7421.048	10.308 (3)	.016	7461.048	7569.436	7505.899
Constrain all E mods	7433.930	23.190 (3)	<.001	7473.930	7582.318	7518.780
Constrain all A mods	7420.702	9.962 (3)	.019	7460.703	7569.090	7505.553
Constrain E SES mod	7411.136	0.396 (1)	.529	7455.136	7574.363	7504.472
Constrain E SES and SES x adrenarche mods	7417.478	6.738 (2)	.034	7459.479	7573.286	7506.572
Constrain E SES mod, A and C adrenarche Mods	7411.700	0.960 (3)	.811	7451.701	7560.088	7496.551
Constrain E SES mod, A adrenarche and SES x adrenarche mods, C adrenarche mod	7419.126	8.386 (4)	.078	7457.125	7560.094	7499.733
Constrain E SES mod, A adrenarche mod, C SES and SES x adrenarche mods	7419.976	9.236 (4)	.055	7457.975	7560.943	7500.583

Table 1.2 (cont'd)

Note: ADI = Area Deprivation Index; adrenarche = coded 0 for early adrenarche, 1 for late adrenarche; mod(s) = moderator(s); -2lnL = minus twice the log-likelihood; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion; full model = model with paths and all moderators; A = additive genetic variance; C = shared environmental variance; E = nonshared environmental variance. The best-fitting model description is bolded.

Table 1.3. *Unstandardized path and moderator estimates for full and best-fitting genotype x environment models*

Model	a	c	e	β_{xP}	β_{yP}	β_{zP}	β_{xD}	β_{yD}	β_{zD}	β_{xPD}	β_{yPD}	β_{zPD}
Neighborhood Disadvantage												
Full model	**.269 (.096, .442)**	-.042 (-.317, .234)	**.472 (.360, .584)**	**.510 (.280, .741)**	.253 (-.161, .668)	.059 (-.077, .194)	**.841 (.504, 1.177)**	-.106 (-.689, .476)	**-.394 (-.577, -.211)**	**-.843 (-1.293, -.393)**	.413 (-.288, 1.114)	**.499 (.273, .725)**
Best-fitting	**.244 (.085, .404)**	—	**.510 (.453, .568)**	**.597 (.419, .775)**	—	—	**.886 (.563, 1.209)**	—	**-.450 (-.550, -.350)**	**-.977 (-1.372, -.581)**	**.591 (.331, .850)**	**.587 (.499, .675)**
Family SES												
Full model	**.794 (.575, 1.013)**	.299 (-.253, .850)	**.417 (.295, .539)**	.019 (-.263, .300)	.169 (-.400, .739)	**.244 (.100, .388)**	-.337 (-.695, .021)	**-.718 (-1.389, -.046)**	-.057 (-.238, .123)	.276 (-.205, .758)	.490 (-.266, 1.247)	-.115 (-.339, .110)
Best-fitting	**.819 (.656, .983)**	**.433 (.130, .737)**	**.385 (.325, .444)**	—	—	**.278 (.185, .370)**	**-.383 (-.655, -.110)**	**-.878 (-1.294, -.462)**	—	**.316 (.087, .545)**	**.692 (.363, 1.020)**	**-.174 (-.298, -.050)**

Note: A = additive genetic influences at the lowest levels of the moderators; c = shared environmental influences at the lowest levels of the moderators; e = non-shared environmental influences at the lowest levels of the moderators; β_{xP}, β_{yP}, β_{zP} = coefficients for moderation of genetic/environmental variance by adrenarche; β_{xD}, β_{yD}, β_{zD} = coefficients for moderation of genetic/environmental variance by neighborhood disadvantage/family SES; β_{xPD}, β_{yPD}, β_{zPD} = coefficients representing changes in the moderating effects of disadvantage across adrenarche (i.e., the disadvantage x development interaction). 95% confidence intervals of parameter estimates are included in parentheses. Effects significant at $p < .05$ are bolded.

Figures

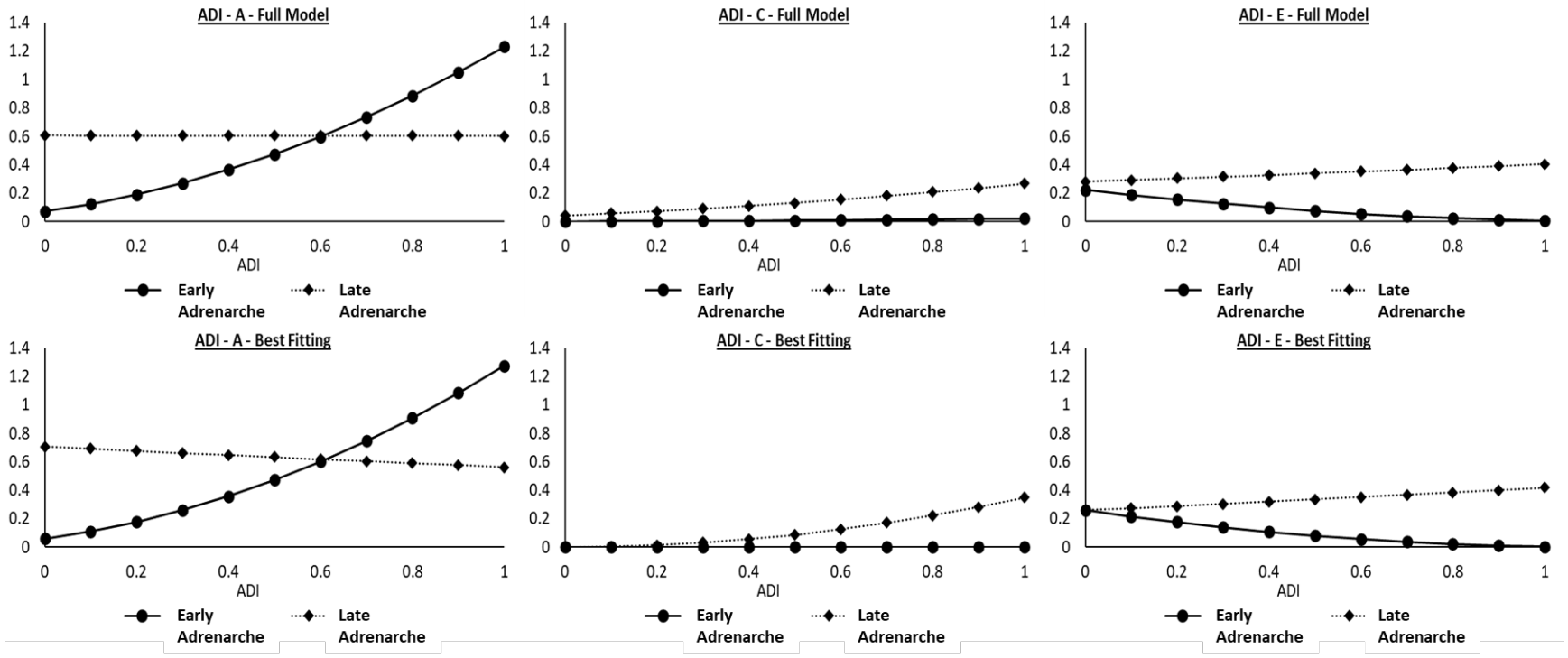


Figure 1.1. *Additive genetic (A), shared environmental (C), and non-shared environmental (E) influences on disordered eating across adrenarche status and neighborhood disadvantage*. ADI = Area Deprivation Index.

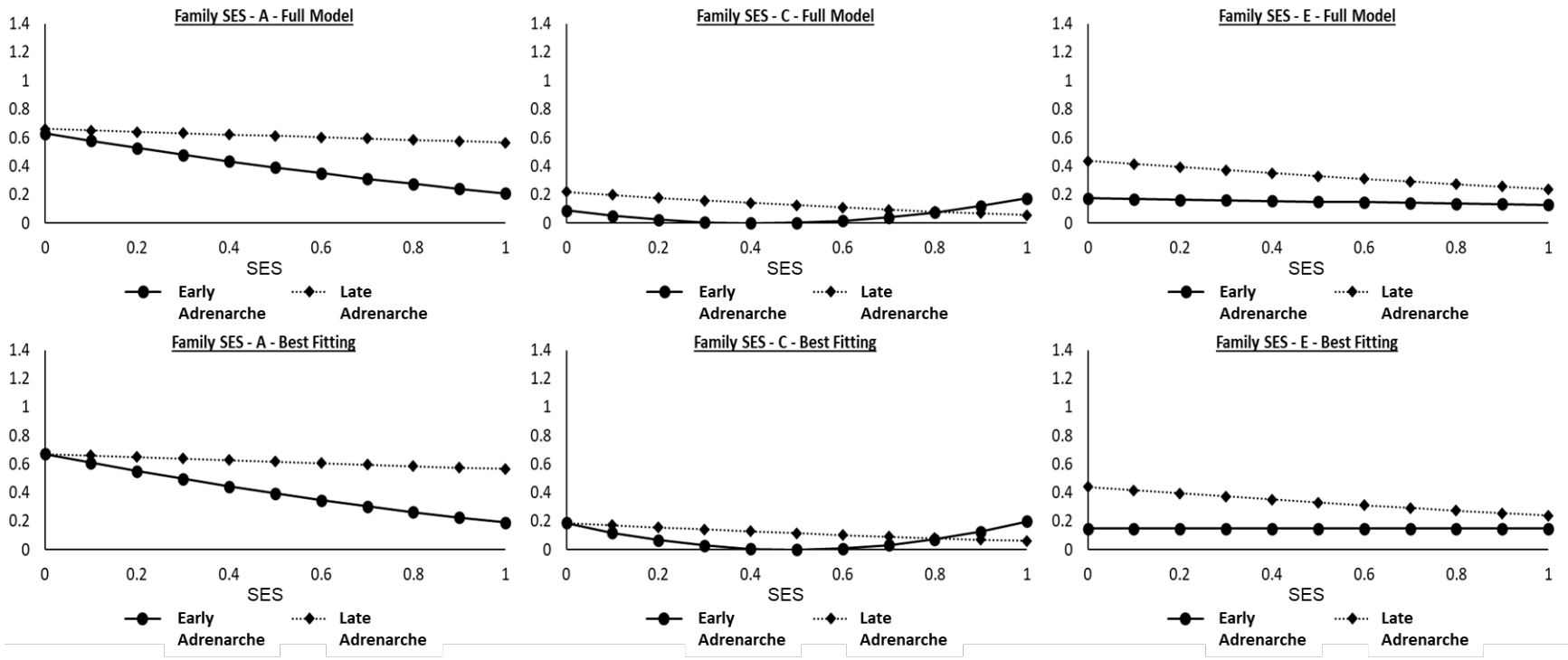


Figure 1.2. *Additive genetic (A), shared environmental (C), and non-shared environmental (E) influences on disordered eating across adrenarche status and family socioeconomic status (SES).*

CHAPTER 2: NEIGHBORHOOD DISADVANTAGE SHAPES TRAJECTORIES OF DISORDERED EATING FROM MIDDLE CHILDHOOD TO EMERGING ADULTHOOD IN FEMALE AND MALE YOUTH

Abstract

Background: Cross-sectional research suggests an association between socioeconomic disadvantage and disordered eating. However, little longitudinal research exists, and no longitudinal studies have examined the broader neighborhood context. We therefore examined how early neighborhood disadvantage impacts developmental trajectories of disordered eating from middle childhood into emerging adulthood and potential sex differences in neighborhood effects. **Methods:** Participants included a sample of girls and boys (N = 2,060 in 1,030 families at intake; M_{age} = 8.02, range = 6-11) enriched for neighborhood disadvantage from the Michigan State University Twin Registry. A subset of participants were reassessed on average 7 years after intake (n = 768) and again ~1.5 years after their first follow-up assessment (n = 380). Disordered eating (e.g., weight/shape concerns, binge eating) was measured using the Minnesota Eating Behavior Survey (MEBS), and neighborhood disadvantage was measured using a 17-indictor Census-block level Area Deprivation Index (ADI). Multilevel growth curve models that can accommodate variability in age at assessment and missing data examined how initial levels and change in neighborhood disadvantage impacted disordered eating trajectories, controlling for other demographic variables. **Results:** Youth who lived in more disadvantaged neighborhoods in middle childhood had persistently elevated disordered eating through emerging adulthood. This effect did not differ across sex and was not significantly attenuated by decreases in neighborhood disadvantage over time. Effects remained significant controlling for family income, suggesting a unique impact of the neighborhood socioeconomic context. **Conclusions:** Neighborhood disadvantage in childhood may have a lasting impact on disordered eating risk in girls and boys.

Introduction

Accumulating cross-sectional research suggests socioeconomic disadvantage is associated with increased disordered eating (e.g., Becker et al., 2019; Carroll et al., 2023; Coffino et al., 2020; Lydecker et al., 2019; Mikhail et al., 2022, 2023). Associations with disadvantage have been observed for both threshold eating disorders (EDs, including anorexia nervosa [AN], bulimia nervosa [BN], and binge-eating disorder [BED]; Coffino et al., 2020; Lydecker et al., 2019) and dimensional disordered eating symptoms (e.g., loss of control over eating, weight/shape concerns; Becker et al., 2019; Carroll et al., 2023; Mikhail et al., 2022, 2023) that may themselves cause distress/impairment (Brownstone & Bardone-Cone, 2020; Hart et al., 2020) and increase risk for future EDs (Le Grange & Loeb, 2007; Stice et al., 2009). Associations have also been observed between disordered eating and disadvantage at multiple levels of proximity, including disadvantage at the family level (e.g., food insecurity, lower family income; Becker et al., 2019; Carroll et al., 2023; Simone, Slaughter-Acey, et al., 2022) and in the broader neighborhood context (i.e., increased poverty and limited resources in the area where a person resides; Carroll et al., 2023; Mikhail et al., 2022, 2023; Simone, Slaughter-Acey, et al., 2022).

Nevertheless, several significant questions remain regarding the relationship between disadvantage and disordered eating. Almost all research to date has been cross-sectional, with few studies of associations between disadvantage and disordered eating over time. Longitudinal studies are critical to establish disadvantage as a risk factor for disordered eating rather than a mere correlate. The two longitudinal studies conducted thus far have focused on food insecurity, a proximal measure of disadvantage (Hazzard et al., 2022; Hooper et al., 2022). Both were conducted in the Project EAT sample and found that food insecurity during mid-adolescence was associated with increased binge eating (including new-onset binge eating) in young adulthood

(Hazzard et al., 2022; Hooper et al., 2022). These findings suggest at least some forms of disadvantage may longitudinally predict disordered eating risk.

However, there has been no longitudinal research on other forms of disadvantage, including disadvantage in the neighborhood context. This is an important gap because neighborhood disadvantage is associated with disordered eating above and beyond family-level factors in youth (Carroll et al., 2023), with perhaps particularly strong associations for boys and young adult men (Mikhail et al., 2023; Simone, Slaughter-Acey, et al., 2022). Neighborhood disadvantage is accompanied by a wide range of stressors, including increased exposure to community violence, decreased access to a broad range of resources (e.g., grocery stores, green spaces), and other structural features (e.g., noise pollution, toxicant exposure) that could have enduring impacts on psychological wellbeing and ED risk, but are not captured by more proximal measures of disadvantage like food insecurity. Consistent with a unique and lasting impact of the neighborhood context, neighborhood disadvantage longitudinally predicts risk for forms of psychopathology closely related to EDs (i.e., anxiety, depression) in youth even after accounting for family-level factors, with the strongest effects for youth who lived in disadvantaged neighborhoods the longest (King et al., 2022). Research on neighborhood disadvantage specifically is therefore important to fully understand how different forms and proximity of disadvantage may longitudinally impact ED risk.

There are also some important methodological limitations of existing research on disadvantage and disordered eating. The few longitudinal studies conducted on food insecurity and disordered eating thus far began assessing participants when they had already entered adolescence, which is the peak risk period for ED onset (Hazzard et al., 2022; Hooper et al., 2022; Nagl et al., 2016). Nothing is therefore yet known about how disadvantage in childhood before

EDs typically onset may impact later risk. This is a particularly notable limitation because early disadvantage may have an especially pronounced impact on later health. For example, research has shown a lingering association between childhood family socioeconomic status (SES) and both physical (e.g., cardiovascular, metabolic) and mental (e.g., substance abuse) health in adulthood even when controlling for adult SES (Poulton et al., 2002).

Existing research has also relied on pre/post designs that cannot fully capture how dynamic trajectories of disadvantage and disordered eating may interact over development. A youth's experience of neighborhood disadvantage may change over time as families relocate and neighborhoods evolve (e.g., through gentrification). The direction and magnitude of changes in neighborhood socioeconomic status may differ significantly even for neighborhoods within the same city (Delmelle, 2016), highlighting the importance of modeling individual trajectories of neighborhood disadvantage exposure and their impact on disordered eating over time.

Finally, no studies have examined sex differences in the longitudinal effects of disadvantage in youth. This is a critical gap because there are notable sex differences in trajectories of disordered eating, with substantially larger increases for females than males during puberty (Klump et al., 2017; Nagl et al., 2016). Moreover, although boys and girls show similar associations between disadvantage and disordered eating in childhood, recent work from our group (Mikhail et al., in press) and others (Simone, Slaughter-Acey, et al., 2022) indicates disadvantage may be more strongly associated with disordered eating and EDs in males in adulthood, suggesting a potential divergence in the impact of disadvantage in females and males over time. Understanding sex differences in the longitudinal impact of disadvantage on disordered eating can help inform how intersecting identities may shape risk and refine screening and prevention efforts.

The current study therefore sought to examine the longitudinal impact of early neighborhood disadvantage on trajectories of disordered eating from middle childhood through emerging adulthood, as well as potential sex differences in neighborhood effects. Analyses were conducted in a large sample of youth enriched for neighborhood disadvantage (N = 2,060 at intake; M_{age} = 8.02). A subsample of youth living in disadvantaged neighborhoods at intake (i.e., neighborhoods with ≥10.5% of residents below the federal poverty line) were reassessed on average 7 years later and again ~1.5 years after their first follow-up assessment, when they were in later adolescence or emerging adulthood. We examined how initial levels and change in neighborhood disadvantage impacted initial levels and change in disordered eating across adolescence. These analyses allowed us to investigate whether early neighborhood disadvantage had lingering effects on disordered eating later in development and the extent to which changes in disadvantage over time might attenuate (for youth moving to less disadvantaged contexts) or accentuate (for youth experiencing increasing disadvantage) early disadvantage effects. We conducted all analyses within a sex-differences framework, which allowed us to examine whether longitudinal neighborhood disadvantage effects differ between girls and boys.

Methods

Transparency and Openness

The current study represents secondary data analysis from a larger, ongoing study (described in greater detail below). Current analyses were not pre-registered. Data and research materials related to the current analyses are available from the corresponding author upon request. We report all data exclusions and manipulations.

Participants

Analyses included 2,060 twins in 1,030 families (48.7% female, 51.3% male) who were in middle childhood at intake (M_{age} = 8.02; range = 6-11; SD = 1.49). Participants were recruited

through the Michigan State University Twin Registry (MSUTR; Burt & Klump, 2013, 2019; Klump & Burt, 2006) for the *Twin Study of Behavioral and Emotional Development in Children* (TBED-C; Burt & Klump, 2019). The TBED-C consists of a population-based arm representative of Michigan (51.3% of the overall sample) and an under-resourced arm drawn from Census blocks in which ≥10.5% of residents had incomes at or below the federal poverty line at intake (48.7% of the overall sample). Consent was provided by participants' parents/guardians and study procedures were approved by the University of Michigan (protocol HUM00163965) and Michigan State University (protocol 04–887) Institutional Review Boards.

A subset of TBED-C participants from disadvantaged Census blocks (i.e., participants from the original under-resourced arm or the population-based arm who were living in Census blocks with poverty rates ≥10.5% at intake) were reassessed twice, on average 7 years after the initial assessment (M = 6.78 years after time 1; SD = 1.98; range = .97-13.50; n = 768) and again approximately 1.5 years later (M = 1.36 years after time 2; SD = .48; range = .67-3.50; n = 380), as part of the ongoing *Michigan Twin Neurogenetics Study* (MTwiNS). Varied ages at intake and different reassessment schedules across participants allowed us to capture the full developmental period from 6-21 even though every participant was not assessed at each age included in the study (see Table S2.1 for the number of participants by age at each timepoint).

Participant demographics at all three timepoints are reported in Table 2.1. Almost the full possible spectrum of neighborhood disadvantage was represented at intake (neighborhood disadvantage percentiles = 2-99), with a mean level of neighborhood disadvantage slightly above that of Michigan overall (M = 57.24th percentile relative to all Census blocks in Michigan; SD = 22.67). Consistent with the MTwiNS sampling method, mean neighborhood disadvantage was higher at time 2 (M = 61.57st percentile; SD = 20.94; range = 6-99) and time 3 (M = 60.48th

percentile; *SD* = 21.61; range = 12-99) relative to time 1 (*p*s <.001). Note that a range of neighborhood disadvantage was represented at times 2 and 3 despite twins being selected for reassessment based on living in modestly-to-severely disadvantaged neighborhoods at time 1 because some twins may have moved to less disadvantaged neighborhoods or lived in neighborhoods that experienced gentrification over time, consistent with neighborhood disadvantage as a dynamic variable (see Figures S2.1-S2.2 for individual trajectories of neighborhood disadvantage and disordered eating over time).

Measures

Disordered Eating

Disordered eating was assessed using the Minnesota Eating Behavior Survey[3] total score (MEBS; von Ranson et al., 2005). The MEBS is a 30-item true/false self-report questionnaire that measures several disordered eating symptom domains, including weight preoccupation (e.g., worry about gaining weight), body dissatisfaction (e.g., thinking body parts are too big), compensatory behavior (e.g., fasting or vomiting to control weight/shape), and binge eating (e.g., feeling a loss of control over eating). Analyses focused on the total score because it has the best psychometric properties across sex and adolescent development (Culbert et al., 2014, 2017; Klump et al., 2007; von Ranson et al., 2005) and captures the full range of core ED symptoms.

Importantly, the MEBS total score has previously shown good psychometric properties and validity in children as young as six (Culbert et al., 2017). The MEBS total score has adequate internal consistency (α's > .77) in both girls and boys from age six into young adulthood (Culbert

[3] The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory [M-EDI]) was adapted and reproduced by special permission of Psychological Assessment Resources, 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources.

et al., 2014, 2017; Klump et al., 2007; von Ranson et al., 2005), and its factor structure is consistent across sex (Luo et al., 2016), indicating it captures a similar construct in girls and boys. The MEBS is also strongly correlated with other established disordered eating measures across adolescence in both girls and boys (e.g., *r*'s ≥ .77 with the Eating Disorder Examination Questionnaire; Klump et al., 2012) and successfully discriminates between youth with and without an ED diagnosis (von Ranson et al., 2005). As in past research using the MEBS with young children (Culbert et al., 2017), research assistants read items aloud to participants below age 8 to ensure adequate comprehension.

Neighborhood Disadvantage

Neighborhood disadvantage was assessed using a Census-block level Area Deprivation Index (ADI; Kind & Buckingham, 2013; Singh, 2003). The ADI is a well-validated (Kind & Buckingham, 2013; Singh, 2003) measure of neighborhood disadvantage that incorporates 17 socioeconomic indicators (e.g., unemployment rate, median home value; see Table S2.2 for all items) measured at the Census-block level (i.e., the smallest United States Census Bureau geographic unit). The ADI has excellent internal consistency ($\alpha = .95$ in past research; Singh, 2003). Consistent with past research (Burt, 2014; Hackman et al., 2012; Roubinov et al., 2018), ADI scores and family income were only moderately correlated in this sample ($r = -.40$; 16% of variance shared), indicating they are related but distinct. Neighborhood disadvantage as measured by the ADI is associated with poorer physical and mental health (Burt et al., 2020; Hu et al., 2018; Kind et al., 2014). The ADI score for each family was coded using publicly available data from the American Community Survey for the Census block containing the family's address (https://www.neighborhoodatlas.medicine.wisc.edu/). ADI scores were entered into models as percentiles relative to all Census blocks in Michigan.

Covariates

Pubertal Stage. Disordered eating tends to increase across puberty, especially for girls (Klump et al., 2017). A youth's trajectory of disordered eating over time could therefore depend on their pubertal stage at baseline (e.g., youth who had already passed through puberty at intake might have smaller increases in disordered eating from their initial level). Consistent with past research suggesting puberty may onset earlier for youth in disadvantaged neighborhoods (Acker et al., 2023), partial correlations between the ADI and pubertal stage that controlled for age were significant for both girls ($r = .08$, $p = .011$) and boys ($r = .11$, $p < .001$) at time 1 (but not later timepoints). We therefore covaried time 1 pubertal stage in all analyses to account for the impact of initial pubertal stage on initial levels and subsequent trajectories of disordered eating.

Pubertal stage was assessed using mother-reported scores on the Pubertal Development Scale (PDS; Petersen et al., 1988), a five-item questionnaire that assesses markers of physical maturation during puberty (e.g., growth spurts, breast development in girls, voice changes in boys). Parent-reported scores on the PDS have good internal consistency for girls and boys ($\alpha \geq .91$) and high correlations with professionally assessed Tanner stages that are as high or higher than correlations for self-report (Koopman-Verhoeff et al., 2020). PDS scores were used to classify pubertal stage on a scale from 1 = pre-pubertal to 5 = post-pubertal based on previously established guidelines (Carskadon & Acebo, 1993; Petersen et al., 1988).

Family Income. Youths' mother reported their annual family income from the following options: <$10,000, $10,000-$15,000, $15,000-$20,000, $20,000-25,000, $25,000-$30,000, $30,000-$40,000, $40,000-$50,000, and >$50,000. Income was examined continuously in analyses.

Body Mass Index (BMI). Youth living in disadvantaged neighborhoods tend to have

higher BMIs (Alvarado et al., 2016), and higher BMIs are in turn associated with greater disordered eating, in part because youth with higher BMIs face greater weight stigma (Vartanian & Porter, 2016). It is important to understand whether neighborhood disadvantage is associated with greater disordered eating even if these effects are ultimately mediated by increased BMI and weight stigma, as disordered eating and body dissatisfaction are distressing and impairing for people regardless of weight (Darby et al., 2007). Nevertheless, it is also informative for etiologic models to understand whether neighborhood disadvantage impacts disordered eating independent of BMI. We therefore examined whether observed effects remained significant when covarying time 1 age- and sex-specific BMI percentile based on CDC growth charts (https://www.cdc.gov/healthyweight/xls/bmi-group-calculator-us-062018-508.xlsm) calculated from researcher-measured height and weight.

Statistical Analyses

Data Preparation and General Analytic Approach

MEBS scores were log transformed due to positive skew (however, log transforming had minimal impact on results; see Tables S2.3-S2.4). In the case of missing data on the MEBS, raw scores were prorated if ≤10% of items were missing and marked as missing otherwise. To place effects on a more easily interpretable scale, ADI scores were divided by 10, such that an increase of 1 on the ADI in the model corresponded to a decile increase in neighborhood disadvantage.

Analyses were conducted in Mplus version 8 (Muthén & Muthén, 1998-2021) using robust full information likelihood estimation, which makes use of all available data while accounting for missingness (Enders & Bandalos, 2001). Because the full three-level model failed to converge (likely due to the small number of observations nested within each family, i.e., only two individuals per cluster), we instead used the Mplus "complex two level random" option to

account for clustering at both the family and participant levels when calculating standard errors.

Multilevel Growth Curve Models

The effects of neighborhood disadvantage on trajectories of disordered eating from middle childhood to emerging adulthood were examined using multivariate multilevel growth curve models (MGCM; Kaplan et al., 2009; see Figure 2.1). The MGCM consists of two components: a within-person model and a between-person model. At a within-person level, the MGCM models how time-varying variables (i.e., disordered eating and neighborhood disadvantage) change with increasing age for each individual participant. Random effects on the intercept and slope capture individual differences in initial levels of disordered eating and neighborhood disadvantage (i.e., intercept effects) and in the rate of change of these variables over time (i.e., slope effects). In other words, the MGCM models linear change in neighborhood disadvantage and disordered eating over time for each participant, with participant-specific intercepts and slopes. We centered the model at age 6 (i.e., the youngest age in the sample), such that intercepts represent the predicted levels of neighborhood disadvantage and disordered eating for each participant at age 6. Note that because the MGCM models a unique linear trajectory for each participant, it can accommodate the fact that participants began the study at different ages and had different numbers of follow-up assessments and intervals between assessments. This makes the MGCM more appropriate for the current analyses than other longitudinal models (e.g., cross-lagged panel model), particularly given that we would expect developmental change in disordered eating over time.

At the between-person level, the MGCM examines how individual differences in the intercept and slope of neighborhood disadvantage and disordered eating relate to each other, controlling for other person-level covariates. A significant effect of the intercept of neighborhood

disadvantage on the intercept of disordered eating ($I_1 \rightarrow I_2$ in Figure 2.1) indicates that initial levels of neighborhood disadvantage are associated with initial levels of disordered eating. A significant effect of the intercept of neighborhood disadvantage on the slope of disordered eating ($I_1 \rightarrow S_2$ in Figure 2.1) indicates that initial levels of neighborhood disadvantage predict the rate of change in disordered eating across time. Finally, a significant effect of the slope of neighborhood disadvantage on the slope of disordered eating ($S_1 \rightarrow S_2$ in Figure 2.1) indicates that changes in neighborhood disadvantage over time impact the rate of change in disordered eating (e.g., an increase in disadvantage over time may be associated with a faster rate of increase in disordered eating).

Sex Differences. To identify a best-fitting model, we first examined whether parameters could be constrained to equality across sex. To reduce the number of models necessary to fit, we initially constrained categories of parameters (i.e., all neighborhood disadvantage parameters, all disordered eating parameters, and all disadvantage to disordered eating parameters), then tested individual parameters within a category if constraining all parameters in that category worsened model fit. The constrained model was preferred if the Satorra-Bentler scaled change in chi-square (which is recommended for model comparisons using the cluster robust estimator in Mplus; Satorra, 2010) was non-significant and Akaike's Information Criterion (AIC), Bayesian Information Criterion (BIC), and sample-size adjusted BIC (SABIC) were lower for the constrained model. If AIC, BIC, and SABIC identified different models as best-fitting, the model that optimized two out of three fit indices was selected as best-fitting.

Covariates. All analyses controlled for the impact of time 1 pubertal stage on the intercept and slope of disordered eating. Consistent with prior research on the lasting impact of discriminatory housing practices (Roscigno et al., 2009), youth of color tended to live in much

more highly disadvantaged neighborhoods in our sample (d = .82 between white youth and youth of color at time 1). There is also some evidence of potential differences in developmental trajectories of disordered eating in people with different racial and ethnic identities (Mikhail & Klump, 2021; Simone, Telke, et al., 2022). We therefore covaried parent-reported racial/ethnic identity at intake (using binary variables indicating whether the participant was White, African American, Asian American, Hispanic/Latinx, Native American, or another racial/ethnic identity) as a predictor of the intercept and slope of neighborhood disadvantage and the intercept and slope of disordered eating in all models. Unfortunately, sample sizes of participants from different racial/ethnic groups were too small to examine race/ethnicity as a moderator of disadvantage effects rather than a covariate. We also conducted secondary analyses additionally controlling for time 1 family income and BMI percentile to examine whether neighborhood effects persisted above and beyond these factors.

Results

Sex Differences in Effects

Constraining ADI parameters across sex produced an improvement in model fit on all fit indices and a non-significant change in chi-square (see Table 2.2), indicating no significant sex differences in the mean or variance of initial levels of neighborhood disadvantage and change in neighborhood disadvantage over time. However, consistent with prior research (Klump et al., 2017; Nagl et al., 2016), model fit indices suggested significant sex differences in developmental trajectories of disordered eating. Specifically, the best-fitting model allowed girls and boys to differ in their variability in initial levels of disordered eating, mean change in disordered eating over time, and intercept-slope covariance (i.e., the association between initial levels and change in disordered eating). Despite these sex differences in the overall trajectory of disordered eating, all

associations between ADI parameters and disordered eating parameters could be constrained to equality across sex without a decrement in model fit (see Table 2.2). This indicates that although there were sex differences in mean trajectories of disordered eating, there were no significant sex differences in the impact of neighborhood disadvantage (and change in neighborhood disadvantage) on disordered eating.

Based on these results, the final model interpreted below constrained all parameters to equality in girls and boys except the intercept variance, slope mean, and intercept-slope covariance for disordered eating, which were allowed to vary across sex.

Interpretation of Model Parameters

On average, participants experienced a small but significant decrease in neighborhood disadvantage over time (unstandardized β = -.02, p = .007, 95% CI [-.03, -.003]; see Table 2.3). However, variances for both the intercept and slope of neighborhood disadvantage were also significant, indicating variability across youth in both initial levels and change in neighborhood disadvantage. A significant intercept-slope covariance indicated that youth who lived in more disadvantaged neighborhoods at age 6 tended to have steeper decreases in neighborhood disadvantage over time.

Although girls and boys had the same predicted mean level of disordered eating at age 6, disordered eating tended to increase more rapidly across development for girls (unstandardized β = .05, p = .001, 95% CI [.02, .08]) relative to boys (unstandardized β = .02, p = .295, 95% CI [-.02, .06]) (see Table 2.3). Both girls and boys also showed significant variability around these mean slopes, indicating significant individual differences in changes in disordered eating over time within each sex. Interestingly, the intercept-slope covariance was negative for both girls and boys, suggesting youth with greater initial disordered eating tended to have smaller increases in

disordered eating over time.

Greater initial neighborhood disadvantage was associated with greater initial disordered eating for both girls and boys (i.e., there was a significant effect of the intercept of neighborhood disadvantage on the intercept of disordered eating; unstandardized β = .03, p = .006, 95% CI [.01, .06]) (see Table 2.3). However, initial levels of neighborhood disadvantage were not associated with the rate of change in disordered eating over time (unstandardized β = .00, p = .899, 95% CI [-.004, .003]). Change in neighborhood disadvantage over time was also not significantly associated with change in disordered eating, though the effect was in the expected direction (i.e., increases in neighborhood disadvantage over time associated with more rapid increases in disordered eating; unstandardized β = .04, p = .078, 95% CI [-.004, .08]).

Overall, results indicate that youth who lived in more disadvantaged neighborhoods in middle childhood had contemporaneously elevated disordered eating relative to their more advantaged peers. Because the rate of change in disordered eating did not differ across levels of neighborhood disadvantage, youth who lived in disadvantaged neighborhoods in middle childhood continued to have higher levels of disordered eating into emerging adulthood (see Figure 1.2). In other words, the impact of living in a disadvantaged neighborhood early in life was persistent over time, with youth who had lived in such neighborhoods displaying a consistent elevation in disordered eating that did not attenuate over development. The pattern of effects remained unchanged when controlling for time 1 BMI percentile and family income (see Tables S2.5-S2.8). The effect of initial neighborhood disadvantage also remained significant and was similar in magnitude if analyses were constrained to participants in the under-resourced TBED-C arm at intake (see Tables S2.9-S2.10), suggesting effects generalize to youth across the full spectrum of neighborhood disadvantage (including those in the most disadvantaged contexts).

Discussion

This was the first study to examine longitudinal associations between neighborhood disadvantage and disordered eating in girls and boys, as well as the first to investigate sex differences in the longitudinal effects of any form of disadvantage on disordered eating across the critical adolescent risk period. We found that early neighborhood disadvantage was associated with persistently elevated disordered eating from middle childhood through emerging adulthood, with no significant sex differences in neighborhood disadvantage effects. Neighborhood disadvantage in middle childhood predicted significantly greater disordered eating even when accounting for BMI, family income, and changes in neighborhood disadvantage over time, suggesting early life deprivation in the neighborhood context may uniquely and persistently impact disordered eating risk. Overall, results significantly extend our understanding of the association between disadvantage and disordered eating and highlight the neighborhood context as a critical factor shaping disordered eating risk in youth.

A particularly striking aspect of our findings was that youth who lived in disadvantaged neighborhoods in middle childhood continued to show increased disordered eating many years later into adolescence/emerging adulthood, even if they subsequently lived in more advantaged neighborhoods. These findings are consistent with research on other phenotypes suggesting disadvantage during childhood can have a lasting impact on health across the lifespan. Socioeconomic disadvantage in childhood longitudinally predicts internalizing disorders (e.g., anxiety, depression; Melchior et al., 2010; Najman et al., 2010) and substance use (Non et al., 2016) in later adolescence and adulthood even when controlling for potential confounds (e.g., parental substance use), and the impact of childhood disadvantage on other health outcomes remains evident even when individuals attain a higher socioeconomic status in adulthood (Poulton

et al., 2002). One possible reason may be that youth with limited access to important community resources early in development (e.g., early education programs, effective and respectful healthcare, positive social and recreational opportunities) may experience lingering impacts of this deprivation even when resources increase later on (e.g., feeling less prepared for advanced classes, experiencing chronic health conditions or mistrust of the medical system, fewer established supportive social connections) (Seabrook & Avison, 2012). There is also evidence that early disadvantage may have a lasting biological impact through epigenetic changes (i.e., changes in gene expression in response to environmental circumstances) that tend to emerge early in development and persist into adulthood (Chu et al., 2018; Loucks et al., 2016; Shields, 2017). While epigenetic changes are theoretically malleable throughout life, some research indicates early disadvantage may have epigenetic consequences that are not reversed by changes in socioeconomic status in adulthood (Austin et al., 2018; Kundakovic & Champagne, 2015). Despite these findings, it is crucial to note that many youth are resilient even when experiencing disadvantage (Burt et al., 2021) and interventions later in development can be effective for individuals from disadvantaged backgrounds (Radunz et al., 2021), particularly when they integrate an understanding of a person's history and holistic needs (Frayn et al., 2022).

Findings were significant independent of family income and BMI, suggesting neighborhood disadvantage may contribute to disordered eating through mechanisms above and beyond those most commonly examined in studies of disadvantage and EDs (e.g., food insecurity, weight stigma; Becker et al., 2021; Hazzard et al., 2022; Hooper et al., 2022). Disadvantaged neighborhoods are often characterized by structural features (e.g., noise pollution, physical disorder, community violence) that can substantially increase psychological stress (Cutrona et al., 2006). Poverty-related stress is in turn longitudinally associated with increased anxiety and

depressive symptoms in youth (DeCarlo Santiago et al., 2011) and could also be a risk factor for disordered eating. Reduced resources at the community level (e.g., fewer medical services, grocery stores, and recreational facilities) could further amplify the impact of neighborhood stressors by limiting availability of fresh foods, opportunities for positive youth development (e.g., activities that might help reduce negative affect and improve self-esteem), and early intervention for youth showing psychological distress. Untangling the contributions of these factors in future research could help identify which specific aspects of disadvantage may put youth at greatest risk and where public policy interventions could potentially have the greatest impact.

This study had several strengths, including longitudinal data collection spanning the critical adolescent risk period for development of disordered eating, well-validated measures of neighborhood disadvantage and disordered eating, and a relatively large sample of girls and boys enriched for neighborhood disadvantage. However, some limitations should also be noted. Follow-up data were only available for a subset of youth, which somewhat reduced power to detect longitudinal effects. Because youth were assessed at a maximum of three timepoints, we modeled change in disordered eating over time as a linear trajectory, as we did not have sufficient data to model a more complex pattern of change. While this approach captured some important aspects of the development of disordered eating during adolescence (e.g., increases over time that are greater for girls than boys), it is likely an overly simplified model of change across time. It would therefore be ideal to replicate findings in other samples that have more sampling points during adolescence and can accommodate more complex modeling approaches. In addition, youth had variable ages at initial assessment and lengths of time between follow-up assessments. Although our modeling approach could accommodate this variability in sampling, it would be

ideal to have assessment data for every participant across the full developmental range examined in the study (i.e., age 6 through emerging adulthood). Finally, while we were able to examine longitudinal associations, this study was observational rather than experimental in nature. Though neighborhood disadvantage effects remained significant after accounting for a range of covariates (e.g., pubertal status, race/ethnicity, family income, BMI), the current study cannot definitively establish causality.

Constraints on Generality

Although we had a strong sampling frame (i.e., birth records) to generate a representative sample, participants were predominantly White (reflecting the overall Michigan population), and we did not explicitly weight for demographic factors. Sample sizes were also too small to examine potential interactions between neighborhood disadvantage and racial identity. Analyses were conducted with youth in the United States, and findings may not necessarily generalize to youth in other countries and non-Western cultural contexts.

Conclusion

Despite these limitations, the current study significantly extends existing research on disadvantage and disordered eating in youth by suggesting that experiences of neighborhood disadvantage in childhood can have a lasting impact on disordered eating risk. Results also add to a broader literature suggesting that early-life socioeconomic disadvantage at both more proximal (e.g., family) and distal (e.g., neighborhood) levels is associated with a wide range of negative psychological and physiological health outcomes in youth (e.g., Carroll et al., 2023; Choi et al., 2021; DeCarlo Santiago et al., 2011). These accumulating findings suggest that in addition to increased screening and intervention at the individual level, community-wide interventions at the neighborhood level warrant investigation for their potential to bolster youth mental health

transdiagnostically.

Tables

Table 2.1. *Descriptive statistics for participant demographics by wave*

	Time 1 (N = 2,060)		Time 2 (N = 768)		Time 3 (N = 380)	
Participant Characteristics	**Mean (SD) or % of Sample (N)**	**Range**	**Mean (SD) or % of Sample (N)**	**Range**	**Mean (SD) or % of Sample (N)**	**Range**
Sex						
Female	1,003 (48.7%)	—	362 (47.1%)	—	177 (46.6%)	—
Male	1,057 (51.3%)	—	406 (52.9%)	—	203 (53.4%)	—
Age	8.02 (1.49)	6-11	14.77 (2.27)	7-21	15.63 (2.33)	10-20
Pubertal stage	1.30 (.66)	1-5	3.60 (1.13)	1-5	3.84 (1.11)	1-5
Race/ethnicity						
White (non-Latinx)	1,682 (81.7%)	—	600 (78.1%)	—	280 (73.7%)	—
Black/African American	196 (9.5%)	—	98 (12.8%)	—	62 (16.3%)	—
Latinx/Hispanic	16 (0.8%)	—	8 (1.0%)	—	4 (1.1%)	—
Asian American	16 (0.8%)	—	4 (0.5%)	—	2 (0.5%)	—
Native American/American Indian	22 (1.1%)	—	10 (1.3%)	—	2 (0.5%)	—
Other/Unknown	128 (6.2%)	—	48 (6.3%)	—	30 (7.9%)	—
Combined parental income						
Less than $10,000	54 (2.6%)	—	10 (1.3%)	—	3 (0.8%)	—
$10,000-$15,000	74 (3.6%)	—	18 (2.3%)	—	4 (1.1%)	—
$15,000-$20,000	72 (3.5%)	—	16 (2.1%)	—	6 (1.6%)	—
$20,000-$25,000	74 (3.6%)	—	18 (2.3%)	—	10 (2.6%)	—
$25,000-$30,000	110 (5.3%)	—	14 (1.8%)	—	8 (2.1%)	—
$30,000-$40,000	166 (8.1%)	—	52 (6.8%)	—	24 (6.3%)	—
$40,000-$50,000	278 (13.5%)	—	65 (8.5%)	—	32 (8.4%)	—
Over $50,000	1,184 (57.5%)	—	518 (67.5%)	—	254 (66.8%)	—
Unknown	48 (2.3%)	—	57 (7.4%)	—	39 (10.3%)	—
Mother's education level						
Less than high school	40 (1.9%)	—	10 (1.3%)	—	10 (2.6%)	—

Table 2.1 (cont'd)

High school graduate	134 (6.5%)	—	52 (6.8%)	—	20 (5.3%)	—
Trade school	88 (4.3%)	—	Not assessed	—	Not assessed	—
Some college	464 (22.5%)	—	156 (20.3%)	—	86 (22.6%)	—
Associate's degree	284 (13.8%)	—	123 (16.0%)	—	52 (13.7%)	—
Bachelor's degree	610 (29.6%)	—	238 (31.0%)	—	120 (31.6%)	—
Advanced graduate degree (e.g., master's, PhD, MD)	344 (16.7%)	—	136 (17.7%)	—	54 (14.2%)	—
Unknown	96 (4.7%)	—	53 (6.9%)	—	37 (9.7%)	—
Father's education level						
Less than high school	106 (5.1%)	—	24 (3.1%)	—	8 (2.1%)	—
High school graduate	394 (19.1%)	—	100 (13.0%)	—	34 (9.0%)	—
Trade school	100 (4.9%)	—	Not assessed	—	Not assessed	—
Some college	470 (22.8%)	—	118 (15.4%)	—	52 (13.7%)	—
Associate's degree	210 (10.2%)	—	71 (9.2%)	—	30 (7.9%)	—
Bachelor's degree	468 (22.7%)	—	127 (16.5%)	—	54 (14.2%)	—
Advanced graduate degree (e.g., master's, PhD, MD)	260 (12.6%)	—	88 (11.5%)	—	36 (9.5%)	—
Unknown	52 (2.5%)	—	240 (31.3%)	—	166 (43.7%)	—
Area Deprivation Index (ADI) percentile rank relative to all Census blocks in Michigan	57.24 (22.67)	2-99	61.57 (20.94)	6-99	60.48 (21.61)	12-99
MEBS total score	5.48 (4.19)	0-22	7.72 (4.69)	0-24.67	6.93 (4.58)	0-22

Note: MEBS = Minnesota Eating Behavior Survey.

Table 2.2. *Model fit comparisons constraining parameters across sex for the neighborhood disadvantage and disordered eating multilevel growth curve model*

Model	AIC	BIC	SABIC	-2lnL	Scaling Correction Factor	$\chi^2\Delta$ (df)	*p*
Full model	9363.25	10055.62	9693.39	9135.25	6.38	—	—
Constraining ADI Parameters							
Constrain all ADI parameters	9360.24	10022.24	9675.90	9142.24	6.52	2.08 (5)	.838
Constraining DE Parameters							
Constrain all DE parameters	9369.06	10031.07	9684.73	9151.06	6.63	16.15 (5)	.006
Constrain DE intercept factor mean	9362.43	10048.72	9689.67	9136.43	6.43	1.20 (1)	.273
Constrain DE intercept factor variance	9368.00	10054.30	9695.25	9142.00	6.43	7.16 (1)	.007
Constrain DE slope factor mean	9366.41	10052.70	9693.65	9140.40	6.43	6.76 (1)	.009
Constrain DE slope factor variance	9362.15	10048.44	9689.40	9136.15	6.42	.74 (1)	.389
Constrain DE intercept-slope covariance	9364.64	10050.94	9691.89	9138.64	6.43	3.43 (1)	.064
Constraining ADI → DE Association Parameters							
Constrain all ADI → DE parameters	9360.45	10034.60	9681.90	9138.45	6.52	3.14 (3)	.370
Constrain all parameters except DE intercept factor variance and DE slope factor mean	9359.58	9985.14	9657.87	9153.58	6.84	8.77 (11)	.643
Constrain all parameters except DE intercept factor variance, DE slope factor mean, and DE intercept-slope covariance	**9354.82**	**9986.46**	**9656.00**	**9146.82**	**6.78**	**5.25 (10)**	**.874**

Note: ADI = Area Deprivation Index; DE = disordered eating; -2lnL = minus twice the log-likelihood; $\chi^2\Delta$ = chi square change; df = degrees of freedom; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion. Dashes indicate parameters are not applicable. The best-fitting model is bolded.

Table 2.3. *Estimates from the best-fitting neighborhood disadvantage and disordered eating multilevel growth curve model*

Parameter	Unstandardized Estimate	SE	*p*	95% CI
Best-Fitting Model in Girls				
Neighborhood disadvantage (ADI)				
Intercept factor mean	5.42	.07	**<.001**	5.29, 5.55
Intercept factor residual variance	4.44	.18	**<.001**	4.09, 4.79
Slope factor mean	-.02	.01	**.016**	-.03, -.003
Slope factor residual variance	.02	.004	**<.001**	.02, .03
Intercept-slope covariance	-.08	.02	**<.001**	-.12, -.04
Disordered eating (DE)				
Intercept factor mean	1.34	.09	**<.001**	1.17, 1.51
Intercept factor residual variance	.47	.06	**<.001**	.35, .59
Slope factor mean	.05	.02	**.001**	.02, .08
Slope factor residual variance	.01	.001	**<.001**	.003, .01
Intercept-slope covariance	-.04	.01	**<.001**	-.05, -.03
Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.03	.01	**.006**	.01, .06
ADI intercept factor → DE slope factor	.00	.002	.899	-.004, .003
ADI slope factor → DE slope factor	.04	.02	.078	-.004, .08
Best-Fitting Model in Boys				
Neighborhood disadvantage (ADI)				
Intercept factor mean	5.42	.07	**<.001**	5.29, 5.55
Intercept factor residual variance	4.44	.18	**<.001**	4.09, 4.79
Slope factor mean	-.02	.01	**.016**	-.03, -.003
Slope factor residual variance	.02	.004	**<.001**	.02, .03
Intercept-slope covariance	-.08	.02	**<.001**	-.12, -.04
Disordered eating (DE)				
Intercept factor mean	1.34	.01	**<.001**	1.17, 1.51
Intercept factor residual variance	.32	.05	**<.001**	.23, .41
Slope factor mean	.02	.02	.295	-.02, .06
Slope factor residual variance	.01	.001	**<.001**	.003, .01
Intercept-slope covariance	-.03	.01	**<.001**	-.04, -.02
Associations between ADI and DE parameters				

Table 2.3 (cont'd)

ADI intercept factor → DE intercept factor	.03	.01	**.006**	.01, .06
ADI intercept factor → DE slope factor	.00	.002	.899	-.004, .003
ADI slope factor → DE slope factor	.04	.02	.078	-.004, .08

Note: T1 = time 1; ADI = Area Deprivation Index.

Figures

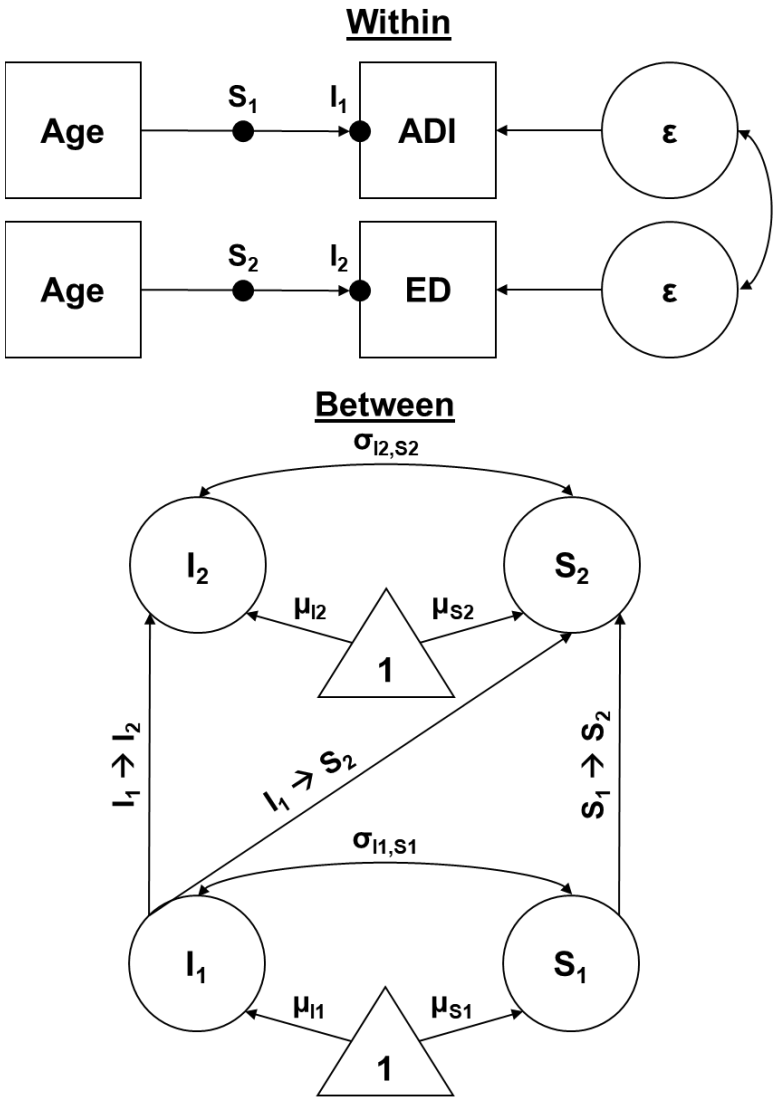


Figure 2.1. *Multilevel growth curve model.* ADI = area deprivation index; DE = disordered eating; I_1 = predicted initial ADI score at age 6 for an individual; S_1 = rate of linear change in ADI with increasing age for an individual; I_2 = predicted initial DE at age 6 for an individual; S_2 = rate of linear change in DE with increasing age for an individual; μ_{I1}, μ_{I2} = predicted initial ADI and DE at age 6 on average across the sample; μ_{S1}, μ_{S2} = mean rates of linear change for ADI and DE with increasing age across the sample; $\sigma_{I1,S1}$ = covariance between initial ADI score and change in ADI over time; $\sigma_{I2,S2}$ = covariance between initial DE and change in DE over time; $I_1 \rightarrow I_2$ =

Figure 2.1 (cont'd)

association between predicted ADI score at age 6 and predicted DE at age 6; $I_1 \rightarrow S_2$ = association between predicted ADI score at age 6 and the rate of change in DE over time; $S_1 \rightarrow S_2$ = association between change in ADI and change in DE over time. Filled dots in the within-person model (labeled S_1, I_1, S_2, and I_2) indicate the presence of random effects. Covariates are not depicted in the figure for simplicity.

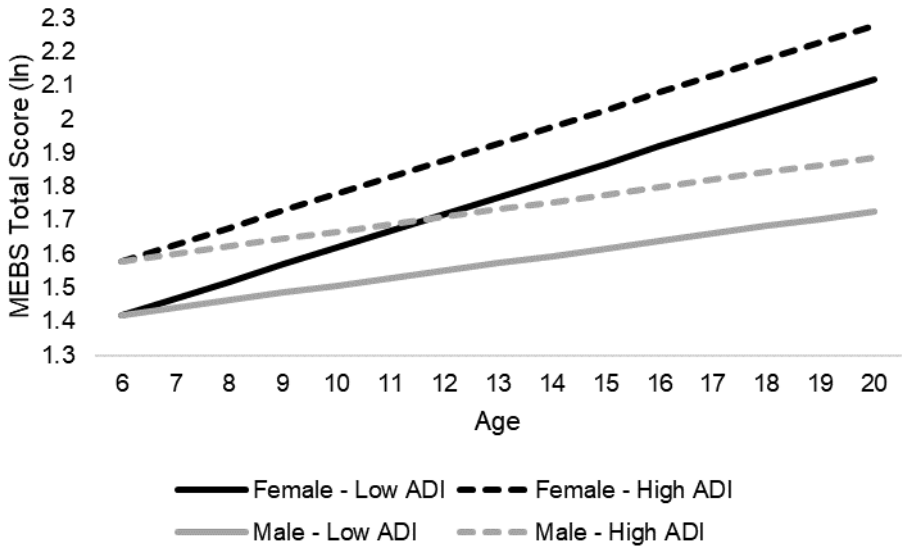


Figure 2.2. *Mean trajectories of disordered eating from middle childhood to young adulthood across sex and neighborhood disadvantage.* ADI = area deprivation index; MEBS = Minnesota Eating Behavior Survey; low ADI = 25th percentile for neighborhood disadvantage in Michigan at age 6; high ADI = 75th percentile for neighborhood disadvantage in Michigan at age 6.

CHAPTER 3: IDENTIFYING ACTIVE INGREDIENTS OF THE CROSS-SECTIONAL AND LONGITUDINAL ASSOCIATIONS BETWEEN NEIGHBORHOOD DISADVANTAGE AND DISORDERED EATING

Abstract

Background: Emerging research suggests neighborhood disadvantage (i.e., poverty in the neighborhood context) is associated with increased disordered eating (DE; e.g., binge eating, body dissatisfaction). However, the specific aspects of neighborhood disadvantage that most strongly predict DE remain unknown. **Methods:** Participants included girls and boys oversampled for neighborhood disadvantage from the Michigan State University Twin Registry (intake $N = 2{,}060$; $M_{age} = 8.02$), a subset of whom were reassessed on average ~7.5 years later (follow-up $N = 770$; $M_{age} = 15.50$). Using structural equation modeling, we examined community violence exposure and neighborhood resources (e.g., parks, medical services, recreational facilities) as cross-sectional and longitudinal predictors of DE, controlling for family income and other demographic factors. **Results:** Associations between neighborhood characteristics and DE did not significantly differ across sex. Cross-sectionally, community violence exposure was associated with increased DE at both intake and follow-up. Longitudinally, community violence exposure at intake predicted greater DE at follow-up indirectly through its association with greater intake DE. Although not associated with DE cross-sectionally, fewer neighborhood resources at intake predicted a greater increase in DE from intake to follow-up. **Conclusions:** Neighborhood factors in middle childhood may leave an enduring imprint on DE risk. Community violence exposure and associated trauma may contribute to early increases in disordered eating that are sustained over development. While the effects of community resources may be less immediately evident, their absence may increase risk for DE over time. Both neighborhood resources and stressors should be incorporated into etiologic models and public policy related to DE.

Introduction

Youth living in disadvantaged neighborhoods characterized by increased poverty and decreased structural resources (such as schools, medical facilities, and recreational/community organizations) are at elevated risk for a wide range of mental health concerns. Prior research has found that neighborhood disadvantage is associated with anxiety/depression (Choi et al., 2021; Xue et al., 2005), externalizing disorders (Pearson et al., 2022), and social difficulties (DeCarlo Santiago, 2011) in young people. In contrast to historical stereotypes (Gard & Freeman, 1996), emerging research also points to associations between neighborhood disadvantage and eating disorders (EDs; e.g., anorexia nervosa [AN], bulimia nervosa [BN], binge-eating disorder [BED]) and disordered eating symptoms (e.g., binge eating, body dissatisfaction) in youth (Burnette et al., 2023).

Though research in this area remains limited, the few studies conducted thus far suggest neighborhood disadvantage may be associated with disordered eating in both girls and boys even when controlling for family-level factors (e.g., family income; Carroll et al., 2023; Mikhail et al., under review). Material neighborhood disadvantage (including availability of food outlets) is not consistently associated with food insecurity after controlling for individual/family demographic factors (Allard et al., 2017; Carter et al., 2012; Denney et al., 2018; Kirkpatrick & Tarasuk, 2010; Santarossa et al., 2021), suggesting neighborhood disadvantage effects may also be independent of food insecurity. In other words, the neighborhood context may have a unique impact on disordered eating above and beyond the more proximal forms of disadvantage most often studied. These preliminary findings are particularly salient given extensive evidence that individuals living in disadvantaged communities are less likely to be able to access health care (including behavioral health care; Kirby & Kaneda, 2005; VanderWielen et al., 2015) and may have needs for

additional services and advocacy beyond the traditional scope of treatments designed in more advantaged contexts (Rose & Thompson, 2012). Consequently, youth in disadvantaged neighborhoods may not only be at higher risk for EDs but may also be less likely to receive timely and effective treatment, potentially leading to significant impairments in physical and psychological wellbeing.

While research in this area is increasing, relatively few studies have been conducted overall, and many important gaps remain in our understanding of when and why neighborhood disadvantage is linked to disordered eating. Most notably, almost nothing is known about the "active ingredients" of neighborhood disadvantage that may increase disordered eating risk. Understanding these active ingredients is critical for advancing etiologic models of risk in disadvantaged populations and identifying points for intervention.

Neighborhood disadvantage is associated with multiple factors that are linked to negative mental health outcomes in youth and may act as active ingredients to increase disordered eating. Disadvantaged neighborhoods are less likely to contain structural resources, including green spaces, community centers, medical services, and recreational facilities, that help support healthy physical and psychological youth development (Henry et al., 2014; Pearson et al., 2022; Timonen et al., 2021; Zhang et al., 2020). Positive youth development theory suggests the presence of supportive resources in the community ("community assets") is important to facilitate healthy socioemotional development in young people (e.g., social competencies, positive identity, sources of support and connection, and a sense of personal self-efficacy; Shek et al., 2022). Conversely, youth living in neighborhoods with a dearth of resources are at increased risk for several negative outcomes related to disordered eating, including anxiety/depression (Li et al., 2021), being diagnosed with a mental health condition (Shen, 2022), decreased wellbeing (Liu et al., 2018),

and poorer metabolic outcomes (e.g., poorer diabetes control; Bergmann et al., 2022). Limited neighborhood resources could impact disordered eating through mechanisms shared with other mental health concerns (e.g., increased negative affect, decreased social support, lower overall self-esteem) as well as mechanisms more specific to disordered eating (e.g., fewer safe areas for movement and recreation contributing to lower body esteem). However, no research has yet examined associations between neighborhood resources and disordered eating in youth.

Simultaneously, individuals living in disadvantaged neighborhoods are exposed to elevated stressors, among the most salient of which is community violence. Research suggests youth living in disadvantaged neighborhoods in the United States are at substantially elevated risk for witnessing community violence, with youth in disadvantaged urban neighborhoods approximately three times as likely to be exposed to deadly gun violence compared to youth in advantaged neighborhoods (Kravitz-Wirtz et al., 2022). Though there have been fewer studies on disadvantage and violence exposure in rural regions, extant research suggests neighborhood disadvantage is also associated with higher rates of violence in rural communities (Lee at al., 2003). The reasons for associations between neighborhood disadvantage and community violence are complex and include increased individual-level economic distress (Benson et al., 2003), feelings of powerlessness and threat (Ross et al., 2001), cycles of victimization and violent offending (Berg & Loeber, 2011), fewer supportive social organizations (Slocum et al., 2013), and decreased social connection and informal neighborhood social control (Henry et al., 2014).

Importantly, witnessing or experiencing violence (Brady, 2008) and trauma more generally (Brewerton, 2015; Trottier & MacDonald, 2017; Zelkowitz et al., 2022) are associated with increased ED risk. While there has been much less research regarding exposure to violence in the broader community, there is emerging evidence that such exposure may also contribute to

disordered eating. In the two studies that have examined this question explicitly, community violence exposure (defined as witnessing or directly experiencing at least one community violence event in the past year, such as being shot or shot at or getting beaten up or mugged) was associated with disordered eating symptoms in both girls and boys cross-sectionally (J. Isaksson et al., 2023) and across a one-year follow-up period (M. Isaksson et al., 2024). Associations between community violence exposure and disordered eating may be mediated by increased trauma symptoms relevant to disordered eating, including dissociation from one's body and emotion regulation difficulties (M. Isaksson et al., 2024; Lev-Ari et al., 2021). However, it is unknown how community violence exposure may be associated with disordered eating across longer follow-up periods or among youth living in disadvantaged neighborhoods where violence may occur more often. Because the two studies conducted thus far have used dichotomous measures of violence exposure (i.e., any exposure versus none), it is also unclear whether the frequency of violence exposure may linearly increase risk.

Current Study

The goal of the current study was therefore to better understand the active ingredients of neighborhood disadvantage effects by examining (limited) neighborhood resources and community violence exposure as potential predictors of disordered eating in youth. We investigated this question in a large sample of girls and boys enriched for neighborhood disadvantage. Youth were followed longitudinally from middle childhood to mid/late adolescence, allowing us to examine the impact of different facets of neighborhood disadvantage both cross-sectionally and over time. Notably, this is the first study to identify specific aspects of disadvantaged neighborhoods that may longitudinally predict disordered eating from childhood into the critical adolescent risk period for developing EDs. We incorporated a deep phenotyping

of neighborhood characteristics, including neighbor informant reports of neighborhood resources and a well-validated, multi-scale dimensional measure of community violence exposure. Because our sample included both girls and boys (based on sex recorded on original birth certificates; hereafter referred to as sex), we were also able to examine sex differences in associations between aspects of neighborhood disadvantage and disordered eating across development.

Methods

Participants

Analyses included 2,060 participants (48.7% girls, 51.3% boys) nested in 1,030 families recruited through the Michigan State University Twin Registry (MSUTR; Burt & Klump, 2013, 2019; Klump & Burt, 2006) for the *Twin Study of Behavioral and Emotional Development in Children* (TBED-C; Burt & Klump, 2019). The TBED-C consists of a population-based arm representative of the state of Michigan (51.3% of the overall sample) and an under-resourced arm drawn from Census blocks in which ≥10.5% of residents had incomes at or below the federal poverty line at intake (48.7% of the overall sample). At their intake assessment, participants ranged in age from 6-10 (M_{age} = 8.02, *SD* = 1.49), although a handful of pairs had turned 11 by the time they participated.

A subset of TBED-C participants from disadvantaged Census blocks (i.e., participants from the original under-resourced arm or the population-based arm living in Census blocks with poverty rates ≥10.5% at intake) were reassessed once or twice, on average 7 years after the initial assessment (wave 2; *M* = 6.78 years after intake; *SD* = 1.98; range = .97-13.50) and again approximately 1.5 years later (wave 3; *M* = 1.36 years after wave 2; *SD* = .48; range = .67-3.50), as part of the ongoing *Michigan Twin Neurogenetics Study* (MTwiNS). For the current analyses, we included follow-up data from wave 3 for participants who had completed their wave 3 assessment (*n* = 380; 49.4% of participants with follow-up data) and from wave 2 for participants

who had not yet completed their wave 3 assessment but were at least 10 years old at wave 2 (which corresponds to the youngest age at wave 3) (n = 390; 50.6%). This allowed us to maximize the sample size at follow-up while ensuring most participants had entered the peak adolescent risk period for disordered eating (i.e., were at least 10 years old). Importantly, variables included in the current study were assessed in the same manner at wave 2 and wave 3. Time from intake to follow-up did not significantly differ between participants whose data were drawn from their wave 2 assessment (M = 7.52, SD = 1.79) and those whose data were from their wave 3 assessment (M = 7.47, SD = 1.71) (d = .03, p = .708), as participants who had not yet completed wave 3 were likely among the last to complete wave 2. Global neighborhood disadvantage as measured by a Census-block level Area Deprivation Index (ADI; Kind & Buckingham, 2018; Singh, 2003) was also equivalent between participants whose follow-up data were drawn from wave 2 and those whose follow-up data were drawn from wave 3 (d = .04, p = .610).

Consistent with the MTwiNS recruitment strategy, participants with follow-up data had greater levels of global neighborhood disadvantage at intake than participants without follow-up data (d = .38, p <.001), but did not significantly differ from participants without follow-up data on time 1 age or sex (ps >.10). However, global neighborhood disadvantage did not differ between participants with and without follow-up data who were in the under-resourced arm at intake (d = .04, p = .560). We therefore conducted sensitivity analyses including only youth in the under-resourced arm at intake to ensure results generalized to the more disadvantaged youth in the sample and were not skewed by the greater level of disadvantage in the follow-up sample relative to the full sample at intake. Additional demographic information at intake and follow-up is included in Table 3.1.

Measures

Disordered Eating

Disordered eating at intake and follow-up was assessed using the total score from the Minnesota Eating Behavior Survey[4] (MEBS; von Ranson et al., 2005). The MEBS is a 30-item self-report questionnaire that assesses several domains of disordered eating using a true/false format, including weight preoccupation (e.g., worry about gaining weight), body dissatisfaction (e.g., thinking body parts are too big), compensatory behavior (e.g., vomiting to control weight/shape), and binge eating (e.g., feeling a loss of control over eating).

Analyses focused on the total score because this overall measure of disordered eating has the best psychometric properties across sex and child/adolescent development (Culbert et al., 2014, 2017; Klump et al., 2007; von Ranson et al., 2005) and captures the full spectrum of disordered eating symptoms. The MEBS total score has adequate internal consistency (α's > .77) in both girls and boys from age six into emerging adulthood (Culbert et al., 2014, 2017; Klump et al., 2007; von Ranson et al., 2005), and its factor structure is consistent across sex (Luo et al., 2016), indicating it captures a similar construct in both girls and boys. The MEBS is also strongly correlated with other established disordered eating symptom measures (e.g., *r*'s ≥ .77 with the Eating Disorder Examination Questionnaire [Fairburn & Beglin, 1994] across puberty in both girls and boys; Klump et al., 2012) and discriminates between girls with and without an ED diagnosis (von Ranson et al., 2005). As in past research that has used the MEBS with young children (Culbert et al., 2017), research assistants read items aloud to participants aged six and

[4] The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory [M-EDI]) was adapted and reproduced by special permission of Psychological Assessment Resources, 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources.

seven to ensure adequate comprehension.

Neighborhood Resource Availability

Neighborhood resource availability was assessed using informant reports provided by neighbors living in each family's Census tract. To obtain neighbor reports, mailings were sent to 10 randomly chosen households within a family's Census tract, with one adult at each address invited to complete the questionnaire (see Burt et al., 2020 for additional details). Participant demographic characteristics were generally similar at intake (63.2% women; M_{age} (*SD*) = 52.61 (15.52); 80.6% White, 11.6% Black/African American, 7.8% other racial/ethnic identity) and follow-up (66.2% women; M_{age} (*SD*) = 56.56 (16.46); 88.9% White, 6.7% Black/African American, 4.3% other racial/ethnic identity). There were a total of 1,880 independent neighbor reports at intake (mean = 4.49 reports per Census tract, *SD* = 1.63, range = 1-10) within Census tracts containing 1,514 youth (73.5% of the full sample). At the time of the current data draw, there were 713 neighbor reports at follow-up (mean = 2.92 reports per Census tract, *SD* = 1.52, range = 1-10) within Census tracts containing 526 youth (68.3% of the follow-up sample). Because recruitment of neighbor informants focused on under-resourced neighborhoods at intake, global neighborhood disadvantage was greater among youth participants with versus without neighbor informant reports at intake in the full sample ($d = .47$, $p < .001$). However, global neighborhood disadvantage did not differ between participants with versus without neighbor informant reports at follow-up ($d = .08$, $p = .330$) or in participants with versus without neighbor informant reports within the under-resourced arm at intake ($d = .08$, $p = .610$).

Neighbor informants reported on neighborhood resources using the 13-item true/false Resource Availability subscale of the Neighborhood Matters Scale questionnaire (NMS; Henry et al., 2014). Prior research has found the kinds of built neighborhood characteristics assessed by the

NMS are significantly associated with increased rates of other behavioral health concerns (e.g., conduct problems) in youth even after controlling for demographic characteristics (e.g., age, sex) (Burt et al., 2020; Pearson et al., 2022). The Resource Availability subscale assesses the presence/absence of multiple resources in the community, including medical services, recreational and religious facilities, public transportation, grocery stores, and green spaces (i.e., parks). We conducted random effects models to examine whether neighbor reports in the same Census tract were significantly more similar than expected by chance. A significant proportion of the variance in reports of neighborhood resources could be attributed to the Census tract at both intake (19.1% of variance; 95% CI = .15-.24) and follow-up (29.6% of variance; 95% CI = .22-.39). Following guidance from the scale developers (Henry et al., 2014), we did not calculate internal consistency for the Resource Availability subscale because the presence of one resource in a neighborhood does not necessarily guarantee the presence of others (e.g., a neighborhood may have a school or religious services but lack grocery stores or medical facilities). However, Henry et al. (2014) found the items on the Resource Availability subscale form a single factor, indicating it captures a unitary underlying construct.

Community Violence Exposure

Community violence exposure was measured using the 27-item KID-SAVE (Flowers et al., 2000). Consistent with author recommendations (Flowers et al., 2000), the KID-SAVE was administered via clinical interview at intake to ensure comprehension in younger children and via self-report questionnaire at follow-up. The KID-SAVE assesses the frequency (rated on a 3-point scale from "never" to "a lot") at which youth are exposed to violence in their neighborhood, including indirect violence (e.g., "I have heard about drive-by shootings in my neighborhood"), direct violence (e.g., "someone has pulled a gun on me"), and physical/verbal abuse (e.g.,

"someone has threatened to beat me up"). To ensure a comprehensive assessment of community violence exposure, the current study used the KID-SAVE total score capturing frequency of exposure to all forms of violence in one's neighborhood. While the KID-SAVE also assesses the perceived impact of violence exposure for each item (rated from "not at all upsetting" to "very upsetting"), we focused on frequency because we hypothesized violence exposure may have a deleterious impact even if not perceived as overtly upsetting in the moment. This is also consistent with the limited prior research on community violence exposure and disordered eating, which has focused on the occurrence of exposure rather than its perceived impact (J. Isaksson et al., 2023; M. Isaksson et al., 2024). Nevertheless, frequency and impact scores were highly correlated (r = .81 at intake and .76 at follow-up), and results were identical if impact scores were used instead of frequency scores (see Tables S3.1-S3.2). Past research has shown excellent internal consistency (α = .91), test-retest reliability (r = .86 over three weeks), and construct validity (i.e., significant correlations with posttraumatic stress symptoms) of the KID-SAVE frequency total score in children (Flowers et al., 2000).

Covariates

Individuals with marginalized racial identities are more likely to live in disadvantaged neighborhoods due to the ongoing impact of redlining and other discriminatory housing practices (Bailey et al., 2017; Egede et al., 2023). People with marginalized racial identities may also experience prejudice and other environmental stressors that could increase ED risk (Mikhail & Kump, 2021). We therefore included racial identity (identified at intake) as a covariate in analyses. We initially tried to fit models with all racial identities in our sample included individually (i.e., without combining any groups); however, this led to model convergence issues, likely due to the small number of participants with racial identities other than Black and White at

follow-up (e.g., only 4 Asian-American participants). Racial identity was therefore coded as White, Black/African American, or other person of color (including participants who identified their race as Native American, Asian American, Latinx/Hispanic, Pacific Islander, or another race) in analyses.

Family income at intake and follow-up was also included as a covariate to understand the impact of neighborhood factors above and beyond family-level resources. Youth's parents reported their annual family income as <$10,000, $10,000-$15,000, $15,000-$20,000, $20,000-25,000, $25,000-$30,000, $30,000-$40,000, $40,000-$50,000, or >$50,000. Family income was included as a continuous variable (coded from 0 = <$10,000 to 7 = >$50,000) in analyses.

General Analytic Approach

In the case of missing data on individual scales, raw scores were prorated if ≤10% of items were missing and marked as missing otherwise. Because skew for the MEBS was <1 at both intake and follow-up for girls and boys, scores were not log transformed. All analyses were conducted in Mplus version 8 (Muthén & Muthén, 1998-2021) with robust full information maximum likelihood estimation to account for missing data (Enders & Bandalos, 2001) and the "complex" option to control for clustering of twins within families. Models allowed for correlations between predictors that were not connected by direct effects. Statistical significance was determined using the percentile bootstrapping method with 1000 random samples with replacement (Falk, 2018; Preacher & Hayes, 2008); paths were deemed significant if the 95% confidence interval did not contain 0. Based on recommendations in the literature, the fit of the final model was deemed adequate if RMSEA was <0.08 (Browne & Cudeck, 1993) and TLI ≥ 0.95 *or* SRMR ≤ 0.09 (Hu & Bentler, 1999). Unstandardized effects are reported in figures and in the text, with standardized estimates and values for all covariates in the models included in Table

S3.3.

Structural Equation Model

The structural equation model is shown in Figure 3.1. Cross-sectionally, the model estimates whether neighborhood resources and community violence exposure are associated with disordered eating at the same timepoint, covarying age, racial identity, and family income (which are not shown in Figure 3.1 for legibility). Longitudinally, the model estimates whether neighborhood resources and community violence exposure at intake are associated with disordered eating at follow-up, either directly or indirectly through intake disordered eating. A significant direct longitudinal path in this model implies that a neighborhood variable at intake predicts the degree of increase in disordered eating from childhood to adolescence. A significant indirect longitudinal path implies that the increase in disordered eating associated with a neighborhood variable at intake persists over time, leading to increased disordered eating at follow-up.

Examination of Sex Differences

The full model was first estimated separately in girls and boys. We then tested which model paths could be constrained to equality across sex without worsening model fit. The constrained model was preferred if the Satorra-Bentler scaled change in chi-square (Satorra, 2010) was non-significant and Akaike's Information Criterion (AIC), Bayesian Information Criterion (BIC), and sample-size adjusted BIC (SABIC) were lower for the constrained model. If AIC, BIC, and SABIC identified different models as best-fitting, the model that optimized two out of three fit indices was selected as best-fitting. Correlations between predictors without direct paths in the model and variable means and variances were allowed to freely vary across sex because testing equality of these parameters was not central to study hypotheses.

Results

Cross-sectional and Longitudinal Associations between Neighborhood Factors and Disordered Eating

As shown in Table 3.2, the best-fitting model constrained all parameters across sex except the associations between age and identifying as a person of color other than Black/African American with disordered eating at intake. Among girls only, youth who identified as a person of color other than Black/African American had greater disordered eating at intake than White youth. Age was also negatively associated with disordered eating at intake in girls only, with a small effect size. Although somewhat counterintuitive, a slight decrease in disordered eating across middle childhood in girls has been observed in prior research (Davison et at., 2003; Evans et al., 2013; Knez et al., 2006), and disordered eating in girls increased from intake to follow-up as expected (d = .52, p <.001).

Fit of the final model was good (RMSEA = .008, 95% CI [.000, .027]; TLI = .979; SRMR = .020). Greater community violence exposure was associated with significantly greater concurrent disordered eating at intake (unstandardized b = .21, 95% CI [.15, .27]) and follow-up (unstandardized b = .23, 95% CI [.11, .36]) in both girls and boys (see Figure 3.1). There was no direct effect from community violence exposure at intake to disordered eating at follow-up, indicating that community violence exposure at intake did not predict change in disordered eating from childhood to adolescence. However, community violence exposure at intake significantly *indirectly* predicted disordered eating at follow-up in girls and boys through its effect on intake disordered eating (unstandardized b = .03, 95% CI [.01, .05]). This indicates that the elevation in disordered eating associated with community violence exposure at intake persisted into adolescence. Conversely, while neighborhood resources were not significantly associated with

contemporaneous disordered eating at intake or follow-up, greater resource availability at intake directly predicted lower disordered eating at follow-up (unstandardized b = -.41, 95% CI [-.72, -.11]). This suggests that youth with lower neighborhood resources at intake had greater increases in disordered eating from middle childhood to mid/late adolescence.

Results were very similar in the under-resourced arm at intake, except the indirect path from intake community violence exposure to follow-up disordered eating was only significant in girls (albeit positive in both sexes) (see Tables S3.4-S3.5). This difference is likely attributable to the smaller sample size combined with the fact that disordered eating at intake was more strongly associated with disordered eating at follow-up in girls than boys in this subsample.

Follow-Up Analyses – Associations between Global Neighborhood Disadvantage and Disordered Eating Through Neighborhood Factors

In follow-up analyses, we sought to investigate whether the associations between neighborhood factors and disordered eating observed in this study might contribute to previously observed relationships between global neighborhood disadvantage in middle childhood and disordered eating in childhood and later adolescence (Mikhail et al., under review). This would provide further evidence of these factors as potential active ingredients linking neighborhood disadvantage to disordered eating. This model examined whether global neighborhood disadvantage as measured by the ADI at intake was associated with disordered eating at intake and follow-up through its relationship with neighborhood resources and/or community violence exposure (see Figure S3.1).

The best-fitting model constrained all parameters to equality across sex except the associations between age and identifying as a person of color other than Black/African American with disordered eating at intake (as in the model above), the direct path between community

violence exposure and contemporaneous disordered eating at follow-up (which was stronger in girls), and the direct path between the ADI at intake and disordered eating at follow-up after accounting for neighborhood variables (which was stronger in boys) (see Table S3.6). Fit of the final model was good (RMSEA = .000, 95% CI [.000, .017]; TLI = 1.000; SRMR = .017).

All paths that were significant in primary analyses remained significant in this model (see Table S3.7 and Figure S3.1). The ADI was significantly indirectly associated with disordered eating through its relationship with community violence exposure at intake in girls and boys (unstandardized b = .06, 95% CI [.04, .09]). This effect suggests neighborhood disadvantage is associated with greater contemporaneous disordered eating in middle childhood in part because disadvantaged neighborhoods experience more community violence. The ADI at intake was also indirectly associated with disordered eating at follow-up through intake neighborhood resources (unstandardized b = .03, 95% CI [.003, .06]) and intake community violence exposure (via the association between violence exposure and disordered eating at intake; unstandardized b = .01, 95% CI [.001, .02]) in girls and boys. This suggests living in a disadvantaged neighborhood in middle childhood may be associated with increased disordered eating in adolescence in part because such neighborhoods tend to have more community violence and fewer resources.

Discussion

This was the first study to examine "active ingredients" of neighborhood disadvantage that may contribute to disordered eating cross-sectionally at multiple points in development and prospectively across the critical adolescent risk period. Consistent with prior research (Mikhail et al., 2021, 2023, under review), associations between neighborhood characteristics and disordered eating were generally similar across sex. Greater community violence exposure was associated with significantly greater contemporaneous disordered eating in both middle childhood and

mid/late adolescence for girls and boys. Although community violence exposure did not predict the degree of change in disordered eating from childhood to adolescence, increased disordered eating associated with community violence exposure at intake endured to predict persistently elevated disordered eating in adolescence. Conversely, while neighborhood resources were not associated with disordered eating at the same timepoint, lower neighborhood resource availability in middle childhood prospectively predicted greater increases in disordered eating from childhood to adolescence. Neighborhood factors were associated with disordered eating independent of proximal disadvantage (i.e., family income) and contributed to relationships between global neighborhood disadvantage and disordered eating. Altogether, results significantly extend our understanding of when and why neighborhood disadvantage may lead to disordered eating in youth.

The significant association between community violence exposure and disordered eating observed in this study is consistent with the well-established relationship between trauma and EDs (Brady, 2008; Brewerton, 2015; Trottier & MacDonald, 2017). It is also consistent with the limited prior research on associations between community violence and disordered eating cross-sectionally and across a brief follow-up period (J. Isaksson et al., 2023, M. Isaksson et al., 2024). The pattern of effects observed in this study suggests community violence exposure may contribute to relatively immediate increases in disordered eating (i.e., significant cross-sectional effects) that are then sustained across development (i.e., significant indirect longitudinal effects). The impact of early violence exposure may therefore remain evident even when violence exposure decreases over time (as was true for many youth in this sample; $r = .23$ between community violence exposure at intake and follow-up). This pattern is similar to that observed in our earlier study of global neighborhood disadvantage effects on trajectories of disordered eating,

in which early disadvantage predicted initial elevations in disordered eating that persisted over time even for youth who later transitioned to more advantaged neighborhoods (Mikhail et al., under review).

Community violence exposure in youth alters connectivity between brain regions involved in emotion regulation and interoception, including the insula, hippocampus, and amygdala (Dark et al., 2020; Reda et al., 2021; Saxbe et al., 2018). These neural changes could in turn contribute to difficulties regulating negative emotions or processing internal sensations (particularly normative physiological fluctuations that may be reminiscent of fight/flight sensations experienced during violence exposure), both of which are known risk factors for disordered eating and EDs (Brockmeyer et al., 2014; Haedt-Matt & Keel, 2011; Zucker & Bulik, 2020). Repeated violence exposure and consequent activation of the physiological stress response could also contribute to dysregulation of the HPA axis, further amplifying ED risk (Lo Sauro et al., 2008). Additional research is needed to investigate these and other possible mechanisms of community violence effects on disordered eating.

This was the first study to show an association between neighborhood resource availability and disordered eating in youth. Limited neighborhood resources appeared to have a delayed effect on disordered eating, with a significant longitudinal but not cross-sectional association. In contrast to acute stressors like violence exposure, it may take some time for the effects of insufficient resource availability to fully manifest. Indeed, other studies have found delayed effects of neighborhood deprivation on cognitive (Elías Alvarado, 2016; Sampson et al., 2008) and health-related (Jimenez et al., 2019) outcomes. Research suggests socioeconomic deprivation often has a cumulative effect, meaning the impact of early life disadvantage can become compounded over time (Seabrook & Avison, 2012). For example, youth with less access

to positive social and recreational activities in middle childhood may have fewer friends or mentors in adolescence (Schaefer et al., 2011), which could lead to increased loneliness, negative affect, and subsequent mental health concerns. Youth in neighborhoods with few mental health services may be unable to access early intervention when they initially show signs of disordered eating or other mental health conditions, leading to worsening symptoms over time. Importantly, like community violence exposure, the presence of neighborhood resources was relatively dynamic in our sample (r = .27 between intake and follow-up). This implies the impact of childhood neighborhood deprivation was evident in adolescence even though many youth may have transitioned to better resourced neighborhoods later on. Early intervention in under-resourced neighborhoods may therefore be key to prevent disordered eating and other negative outcomes in youth.

Our findings have potential implications for research, policy, and treatment of disordered eating in disadvantaged youth. Some effects of neighborhood-level deprivation may not be immediately apparent, and longitudinal research that can capture distal and cumulative impacts is needed to fully understand the influence of contextual disadvantage on disordered eating. Assessment for youth with EDs should include questions about violence exposure and histories of contextual deprivation, and treatment approaches should be sensitive to the impact of these factors. Lack of trauma-informed care may lead to poorer outcomes (Rodríguez et al., 2005) and even iatrogenic effects (Brewerton, 2019). To holistically address a youth or family's concerns, treatment providers should also be prepared to help families identify and connect with other needed resources (e.g., affordable grocery stores, transportation systems, safe recreational spaces). Notably, such "wraparound" approaches have been found to be more efficacious and cost-effective than traditional approaches for youth with complex behavioral health concerns in past

research (Olson et al., 2021). Though there is limited systematic study of wraparound approaches for treating EDs in youth, initial evidence suggests they may also be efficacious (Darwish et al., 2006; Pehlivan et al., 2022).

This study had several strengths, including a relatively large sample of boys and girls enriched for neighborhood disadvantage, longitudinal analyses that spanned the critical adolescent risk period for developing EDs, and the ability to simultaneously model the impact of multiple neighborhood processes over time. However, some limitations should also be noted. As is common in longitudinal research, sample size was smaller at follow-up than intake, somewhat reducing power to detect longitudinal effects and cross-sectional effects at follow-up. Follow-up did not occur on the same schedule for all youth and the age range at follow-up was relatively wide. While analyses controlled for age, models may not have fully captured the nuances of effects of neighborhood disadvantage at different developmental stages.

Youth self-reported their frequency of community violence exposure, while neighborhood resources were reported by neighbor informants. This method variance could have contributed to the stronger cross-sectional associations between community violence exposure and disordered eating. However, youth-reported community violence exposure was significantly correlated with global neighborhood disadvantage as measured by the ADI (rs = .20-.27, ps <.001) and neighbor-reported fear of crime (rs = .19-.24, ps <.001). This suggests reports of violence exposure were tied to objective neighborhood characteristics rather than simply reflecting youth characteristics.

Although representative of Michigan with respect to race/ethnicity, the sample had relatively few youth of color, which prevented us from examining whether the impact of certain aspects of neighborhood disadvantage might be even more pronounced for youth with marginalized racial/ethnic identities. Prior research in adults suggests socioeconomic

disadvantage may in some cases be more strongly associated with disordered eating for individuals from marginalized racial/ethnic groups (Mikhail et al., in press), perhaps because the social and material impacts of disadvantage are amplified by racial discrimination (Williams, 2002). While we were not able to examine moderation of disadvantage effects by race/ethnicity, it is worth mentioning that Black youth lived in substantially more disadvantaged neighborhoods as measured by the ADI than White youth on average at both intake and follow-up (*d*s = .85-1.28). Black youth are therefore disproportionately likely to be impacted by neighborhood disadvantage even if associations between neighborhood characteristics and disordered eating are similar across race/ethnicity.

Finally, our study was exclusively quantitative in nature, and complementary qualitative research is needed to fully understand the nuances of how and why different aspects of neighborhood disadvantage may impact disordered eating in youth. In addition to the increased insights that can be gained, young people report many benefits of being included in participatory mental health research, including recognition, skill-building, mentorship, and ability to have a meaningful impact (Watson et al., 2023; Zeal et al., 2013). Such benefits may be particularly pronounced for youth experiencing neighborhood disadvantage and other forms of marginalization.

Tables

Table 3.1. *Participant descriptive statistics at intake and follow-up*

	Intake (N = 2,060)		Follow-Up (N = 770)	
Participant Characteristics	**Mean (SD) or N (% of Sample)**	**Range**	**Mean (SD) or N (% of Sample)**	**Range**
Sex				
Female	1,003 (48.7%)	—	366 (47.5%)	—
Male	1,057 (51.3%)	—	404 (52.5%)	—
Age	8.02 (1.49)	6-11	15.50 (2.10)	10-21
Race/ethnicity				
White (non-Latinx)	1,682 (81.7%)	—	604 (78.4%)	—
Black/African American	196 (9.5%)	—	96 (12.5%)	—
Latinx/Hispanic	16 (0.8%)	—	8 (1.0%)	—
Asian American	16 (0.8%)	—	4 (0.5%)	—
Native American/American Indian	22 (1.1%)	—	10 (1.3%)	—
Pacific Islander	6 (0.3%)		6 (0.8%)	
Other/Unknown	122 (5.9%)	—	42 (5.5%)	—
Combined parental income				
Less than $10,000	54 (2.6%)	—	7 (0.9%)	—
$10,000-$15,000	74 (3.6%)	—	12 (1.6%)	—
$15,000-$20,000	72 (3.5%)	—	16 (2.1%)	—
$20,000-$25,000	74 (3.6%)	—	18 (2.3%)	—
$25,000-$30,000	110 (5.3%)	—	12 (1.6%)	—
$30,000-$40,000	166 (8.1%)	—	44 (5.7%)	—
$40,000-$50,000	278 (13.5%)	—	58 (7.5%)	—
Over $50,000	1,184 (57.5%)	—	509 (66.1%)	—
Unknown	48 (2.3%)	—	94 (12.2%)	—
Mother's education level				
Less than high school	40 (1.9%)	—	15 (1.9%)	—
High school graduate	134 (6.5%)	—	44 (5.7%)	—
Trade school	88 (4.3%)	—	Not assessed	—

Table 3.1 (cont'd)

Some college	464 (22.5%)	—	161 (20.9%)	—
Associate's degree	284 (13.8%)	—	125 (16.2%)	—
Bachelor's degree	610 (29.6%)	—	239 (31.0%)	—
Advanced graduate degree (e.g., master's, PhD, MD)	344 (16.7%)	—	133 (17.3%)	—
Unknown	96 (4.7%)	—	53 (6.9%)	—
Father's education level				
Less than high school	106 (5.1%)	—	24 (3.1%)	—
High school graduate	394 (19.1%)	—	102 (13.2%)	—
Trade school	100 (4.9%)	—	Not assessed	—
Some college	470 (22.8%)	—	126 (16.4%)	—
Associate's degree	210 (10.2%)	—	73 (9.5%)	—
Bachelor's degree	468 (22.7%)	—	141 (18.3%)	—
Advanced graduate degree (e.g., master's, PhD, MD)	260 (12.6%)	—	86 (11.2%)	—
Unknown	52 (2.5%)	—	218 (28.3%)	—
Area Deprivation Index (ADI) percentile rank relative to all Census blocks in Michigan (Possible range = 1-99)				
All participants	57.24 (22.67)	2-99	59.98 (21.31)	6-99
Female	56.46 (22.99)	4-99	59.20 (21.85)	6-99
Male	57.99 (22.33)	2-99	60.64 (20.86)	12-99
Exposure to community violence (Possible range = 0-54)				
All participants	3.07 (3.52)	0-33	2.28 (3.58)	0-29
Female	2.64 (3.13)	0-28	1.90 (2.88)	0-17
Male	3.49 (3.81)	0-33	2.60 (4.05)	0-29

Table 3.1 (cont'd)

Neighborhood resources				
(Possible range = 0-13)				
All participants	10.48 (1.47)	5-13	10.58 (1.84)	3.67-13
Female	10.48 (1.50)	5-13	10.58 (1.74)	5-13
Male	10.49 (1.44)	6-13	10.57 (1.92)	3.67-13
MEBS total score				
(Possible range = 0-30)				
All participants	5.48 (4.19)	0-22	7.41 (4.69)	0-24.14
Female	5.32 (4.26)	0-22	8.40 (4.88)	0.67-24.14
Male	5.64 (4.12)	0-22	6.57 (4.35)	0-21.67

Note. MEBS = Minnesota Eating Behavior Survey.

Table 3.2. *Model fit comparisons constraining parameters across sex for cross-sectional and longitudinal effects*

Model	AIC	BIC	SABIC	χ^2 (df)	Scaling Correction Factor	$\chi^2\Delta$ (df)	*p*
Full model	58536.79	59482.87	58949.12	12.18 (12)	1.0825	—	—
Constrain all parameters	58535.54	59397.15	58911.05	37.62 (27)	1.1149	25.20 (15)	.047
T1 associations							
Constrain T1 ECV → T1 DE	58537.70	59478.15	58947.58	14.26 (13)	1.1290	1.73 (1)	.188
Constrain T1 NR → T1 DE	58535.56	59476.01	58945.44	12.88 (13)	1.0839	0.70 (1)	.403
Constrain T1 age → T1 DE	58543.09	59483.54	58952.96	19.77 (13)	1.0871	7.27 (1)	.007
Constrain Black racial identity → T1 DE	58535.38	59475.83	58945.26	12.39 (13)	1.1123	0.41 (1)	.522
Constrain other POC identity → T1 DE	58539.29	59479.74	58949.17	16.34 (13)	1.0829	4.14 (1)	.042
Constrain T1 INC → T1 DE	58534.79	59475.24	58944.67	11.97 (13)	1.1024	0.01 (1)	.920
FU associations							
Constrain FU ECV → FU DE	58537.23	59477.68	58947.10	14.23 (13)	1.0985	1.89 (1)	.169
Constrain FU NR → FU DE	58535.66	59476.11	58945.54	13.27 (13)	1.0601	1.11 (1)	.292
Constrain FU age → FU DE	58536.91	59477.36	58946.79	14.13 (13)	1.0841	1.93 (1)	.165
Constrain Black racial identity → FU DE	58535.35	59475.80	58945.23	12.97 (13)	1.0606	0.71 (1)	.399
Constrain other POC identity → FU DE	58535.15	59475.60	58945.03	12.41 (13)	1.0923	0.30 (1)	.584
Constrain FU INC → FU DE	58535.57	59476.02	58945.45	13.22 (13)	1.0570	1.04 (1)	.308
T1 → FU associations							
Constraint T1 DE → FU DE	58537.30	59477.75	58947.18	14.48 (13)	1.0843	2.27 (1)	.132
Constrain T1 ECV → FU DE	58534.92	59475.37	58944.80	12.30 (13)	1.0830	0.12 (1)	.729
Constrain T1 NR → FU DE	58535.01	59475.46	58944.88	12.63 (13)	1.0614	0.28 (1)	.597
Constrain all parameters except T1 age → T1 DE, other POC identity → T1 DE	**58527.26**	**59400.13**	**58907.68**	**26.68 (25)**	**1.1116**	**14.47 (13)**	**.342**

Table 3.2 (cont'd)

Note. T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; ECV = exposure to community violence; NR = neighborhood resources; POC = person of color; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion; χ^2 **Δ** = change in chi-square; df = degrees of freedom. The best-fitting model is bolded.

Figures

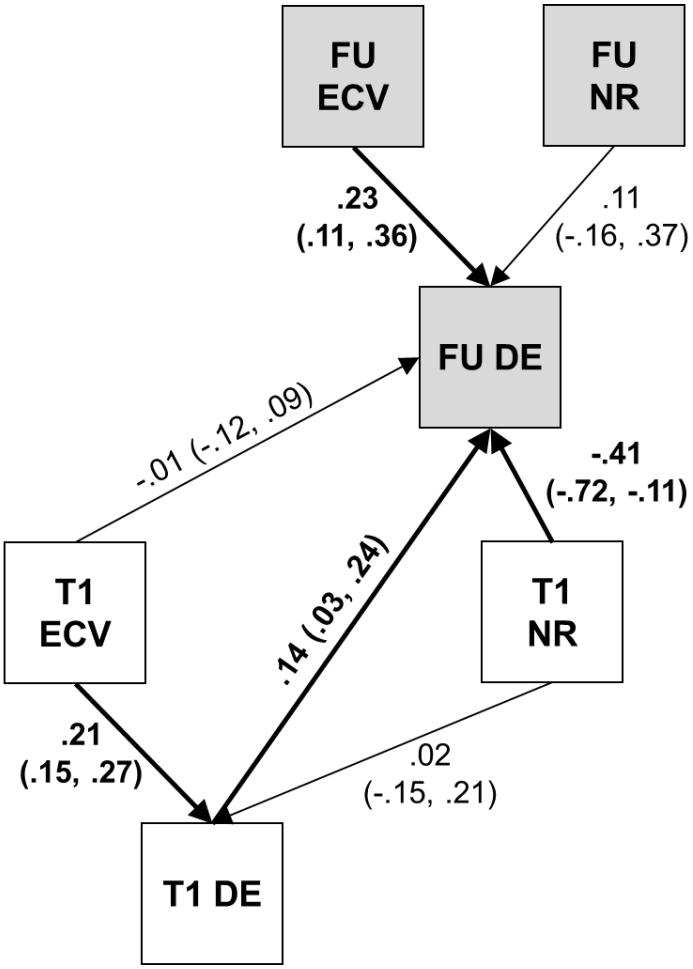


Figure 3.1. *Best-fitting model for cross-sectional and longitudinal associations between neighborhood factors and disordered eating.* T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; ECV = exposure to community violence; NR = neighborhood resources. White boxes represent variables at T1 and grey boxes represent variables at FU. Significant paths are bolded. 95% confidence intervals are included in parentheses. Covariates are not depicted for legibility.

GENERAL DISCUSSION

The studies in this dissertation significantly expanded on prior work on disadvantage and EDs through developmentally sensitive, longitudinal, and genetically informed analyses, with a particular focus on disadvantage in the broader neighborhood context. Study 1 showed that genetic influences on disordered eating were activated earlier for boys living in disadvantaged families or neighborhoods. In combination with Mikhail et al. (2021), the results of study 1 suggest that disadvantage may potentiate earlier expression of genetic risk for disordered eating in both girls and boys relative to typical sex-specific developmental patterns. Study 2 indicated that neighborhood disadvantage may lead to early elevations in disordered eating for both girls and boys that are maintained over development, even when youth later transition to more advantaged contexts. This suggests a profound and persistent impact of childhood disadvantage on disordered eating risk. Finally, study 3 identified limited resource availability and exposure to community violence as two specific neighborhood characteristics that may contribute to elevated disordered eating in youth. The results of this study point to a role for both neighborhood assets and stressors in shaping disordered eating risk. Altogether, the findings from these three studies underscore a significant and enduring impact of neighborhood disadvantage on disordered eating in youth.

Crucially, the studies included in this dissertation highlight disadvantage in the broader neighborhood context as an important environmental factor shaping disordered eating risk. Prior research on disadvantage and EDs has tended to focus on relatively narrow indices of disadvantage closely tied to the food environment, such as food insecurity. While such factors undoubtedly play an important role in disordered eating risk for individuals from socioeconomically disadvantaged backgrounds (Hazzard et al., 2020), a more holistic assessment of the socioeconomic context is needed to fully capture the diverse and intersecting ways disadvantage at different levels of proximity may impact risk. According to Bronfenbrenner's

bioecological model, development is shaped by reciprocal and repeated interactions between a person and their environment that cannot be fully understood without considering both more proximal (e.g., family environment) and distal (e.g., neighborhoods, communities, cultures) contextual factors (Bronfenbrenner & Morris, 2007). Like the development of persons, the development of EDs and disordered eating likely involves complex interactions across multiple levels of analysis that are shaped by the broader social and economic context. Consideration of the neighborhood, community, and even more distal social contexts (e.g., state or national level policies that may shape economic circumstances and opportunity) is crucial to avoid overlooking factors that may have a profound impact on youth and their risk for disordered eating. Relatedly, the types of environmental stressors that increase risk for disordered eating are likely not limited to those that directly impact eating and body image but may also include factors that more generally influence a person's emotions, sense of safety (e.g., community violence exposure), self-esteem, and sense of belonging (e.g., community organizations and supports).

A consistent finding across all three studies was that disadvantage early in development (i.e., during middle childhood/pre-puberty) may have a particularly significant impact on disordered eating risk that persists over time. These findings add to a growing body of evidence that early deprivation may have a unique and lasting impact on later physical and mental health (Galobardes et al., 2004; Heidinger & Willson, 2022; Poulton et al., 2002). The enduring impact of disadvantage early in development may be attributable in part to the earlier activation of genetic influences on disordered eating observed in study 1 and Mikhail et al. (2021) for youth living in disadvantaged contexts. Childhood and adolescence are critical periods for neural development, and genetic influences that normatively come online during puberty are thought to organize brain development in a manner that shapes later behavioral responses to physiological

and environmental cues in adulthood (Schulz & Sisk, 2016). According to the "stress acceleration hypothesis" (Callaghan & Tottenham, 2016), youth living in adverse circumstances may experience accelerated development of certain neural pathways that serve an adaptive function for navigating challenging environments (e.g., affect signaling, stress responding, threat conditioning). While these developmental differences may be adaptive and promote survival in the short term, they may be associated with tradeoffs that impede flexible coping over the long term (Callaghan & Tottenham, 2016). Importantly, the neural circuits that may be most strongly impacted by early developmental stress (e.g., amygdala, prefrontal cortex) have also been implicated in disordered eating and EDs (Bartholdy et al., 2019; Donnelly et al., 2018; Oberndorfer et al., 2011; Steward et al., 2022). Disadvantage-mediated changes to these neural pathways during development could therefore contribute to later disordered eating risk. Future neurobiological research (e.g., neuroimaging, neuroepigenetics) is needed to explore these possibilities.

An important additional area for future research is qualitative studies to complement the kinds of quantitative analyses included in this dissertation. Quantitative analyses are valuable because they can capture data from large numbers of participants to detect risk factors and potential moderators at the population level. At the same time, they cannot capture all the nuances of individuals' lived experiences and risk overlooking influences that researchers did not think to measure or include. Many youth find it empowering to share their lived experiences and play an active role in shaping research and understanding about their lives (Watson et al., 2023; Zeal et al., 2013), and this may be particularly true for youth with histories of marginalization. It is also important for future quantitative and qualitative research to consider the ways in which personal, family, and community strengths and vulnerabilities may reciprocally interact to shape risk over

time. While this dissertation largely focused on neighborhood disadvantage as an under-studied contextual factor, future research would ideally incorporate indices of disadvantage at multiple levels of proximity as well as individual and community resiliency factors that could serve as moderators of disadvantage effects.

While most EDs onset for the first time in adolescence and emerging adulthood (Nagl et al., 2016; Klump, 2013), they are not restricted to this developmental stage. Many individuals experience EDs and disordered eating well into adulthood (Mangweth-Matzek et al., 2014, 2016) and certain developmental events later in adulthood may serve as triggers for ED onset or relapse (e.g., menopause; Baker & Runfola, 2016). Additional research is therefore needed to examine how long the effects of early disadvantage may persist into adulthood and whether there are other sensitive periods during which socioeconomic deprivation may have especially prominent and lasting consequences. Relatedly, although representative of Michigan, the youth included in the studies in this dissertation were predominantly White. More research is needed to understand how racial and socioeconomic marginalization could potentially interact to further increase disordered eating risk (e.g., through intersectional experiences of racism, classism, and economic deprivation; Homan et al., 2021).

Despite the need for additional research in these areas, the findings from this dissertation have important implications for research, screening, treatment, and public policy related to EDs. It is critical to include youth from socioeconomically diverse backgrounds in ED research and consider risk factors that may disproportionately impact disadvantaged youth in etiologic models. Screening for disordered eating should be incorporated in the settings where youth from disadvantaged backgrounds are most likely to present for care, such as pediatrician's offices (Walton et al., 2021). Simultaneously, youth and families presenting for ED treatment should be

screened for socioeconomic stressors in the family and neighborhood context that may be contributing to disordered eating or could impede treatment progress. Youth from disadvantaged backgrounds are less likely to be able to access evidence-based treatment, and efforts are urgently needed to both increase the number of providers serving under-resourced youth and ensure interventions fully meet their specific needs (Accurso, Buckelew, et al., 2021; Accurso, Mu, et al., 2021). In addition to facilitating effective and respectful care at the individual level, it is also imperative for the ED field to work to advance public policy that can enhance access to care for disadvantaged youth (e.g., increasing Medicaid coverage for comprehensive ED care) and shape communities to foster healthy youth development at the population level.

BIBLIOGRAPHY

Accurso, E. C., Buckelew, S. M., & Snowden, L. R. (2021). Youth insured by Medicaid with restrictive eating disorders—underrecognized and underresourced. *JAMA Pediatrics*, *175*(10), 999-1000.

Accurso, E. C., Mu, K. J., Landsverk, J., & Guydish, J. (2021). Adaptation to family-based treatment for Medicaid-insured youth with anorexia nervosa in publicly-funded settings: Protocol for a mixed methods implementation scale-out pilot study. Journal of Eating Disorders, *9*, 1-11.

Accurso, E. C., & Waller, G. (2021). Concordance between youth and caregiver report of eating disorder psychopathology: Development and psychometric properties of the Eating Disorder-15 for Parents/Caregivers (ED-15-P). *International Journal of Eating Disorders*, *54*(7), 1302-1306.

Acker, J., Mujahid, M., Aghaee, S., Gomez, S., Shariff-Marco, S., Chu, B.,...Kubo, A. (2023). Neighborhood racial and economic privilege and timing of pubertal onset in girls. *Journal of Adolescent Health*, *72*(3), 419-427.

Allard, S. W., Wathen, M. V., Shaefer, H. L., & Danziger, S. K. (2017). Neighborhood food infrastructure and food security in metropolitan Detroit. *Journal of Consumer Affairs*, *51*(3), 566-597.

Alvarado, S. E. (2016). Neighborhood disadvantage and obesity across childhood and adolescence: Evidence from the NLSY children and young adults cohort (1986–2010). *Social Science Research*, *57*, 80-98.

Arnold, A. P. (2009). The organizational–activational hypothesis as the foundation for a unified theory of sexual differentiation of all mammalian tissues. *Hormones and Behavior*, *55*(5), 570-578.

Attar, B. K., Guerra, N. G., & Tolan, P. H. (1994). Neighborhood disadvantage, stressful life events and adjustments in urban elementary-school children. *Journal of Clinical Child Psychology*, *23*(4), 391-400.

Auchus, R. J., & Rainey, W. E. (2004). Adrenarche–physiology, biochemistry and human disease. *Clinical Endocrinology*, *60*(3), 288-296.

Austin, M. K., Chen, E., Ross, K. M., McEwen, L. M., Maclsaac, J. L., Kobor, M. S., & Miller, G. E. (2018). Early-life socioeconomic disadvantage, not current, predicts accelerated epigenetic aging of monocytes. *Psychoneuroendocrinology*, *97*, 131-134.

Bailey, Z. D., Krieger, N., Agénor, M., Graves, J., Linos, N., & Bassett, M. T. (2017). Structural racism and health inequities in the USA: Evidence and interventions. *The Lancet*, *389*(10077), 1453-1463.

Baker, J. H., & Runfola, C. D. (2016). Eating disorders in midlife women: A perimenopausal eating disorder? *Maturitas*, *85*, 112-116.

Bartholdy, S., O'Daly, O. G., Campbell, I. C., Banaschewski, T., Barker, G., Bokde, A. L.,...Stedman, A. (2019). Neural correlates of failed inhibitory control as an early marker of disordered eating in adolescents. *Biological Psychiatry*, *85*(11), 956-965.

Becker, C. B., Middlemas, K., Gomez, F., & Kilpela, L. S. (2021). An exploratory examination of internalized weight stigma in a sample living with food insecurity. *Body Image*, *37*, 238-245.

Becker, C. B., Middlemass, K. M., Gomez, F., & Martinez-Abrego, A. (2019). Eating disorder pathology among individuals living with food insecurity: A replication study. *Clinical Psychological Science*, *7*(5), 1144-1158.

Becker, C. B., Middlemass, K., Taylor, B., Johnson, C., & Gomez, F. (2017). Food insecurity and eating disorder pathology. *International Journal of Eating Disorders*, *50*(9), 1031-1040.

Benson, M. L., Fox, G. L., DeMaris, A., & Van Wyk, J. (2003). Neighborhood disadvantage, individual economic distress and violence against women in intimate relationships. *Journal of Quantitative Criminology*, *19*, 207-235.

Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin*, *88*(3), 588-606.

Berg, M. T., & Loeber, R. (2011). Examining the neighborhood context of the violent offending-victimization relationship: A prospective investigation. *Journal of Quantitative Criminology*, *27*, 427-451.

Bergmann, K. R., Nickel, A., Hall, M., Cutler, G., Abuzzahab, M. J., Bretscher, B.,...Hester, G. Z. (2022). Association of neighborhood resources and race and ethnicity with readmissions for diabetic ketoacidosis at US children's hospitals. *JAMA Network Open*, *5*(5), e2210456-e2210456.

Black, A. C., Harel, O., & Betsy McCoach, D. (2011). Missing data techniques for multilevel data: Implications of model misspecification. *Journal of Applied Statistics*, *38*(9), 1845-1865.

Blair, C., & Raver, C. C. (2016). Poverty, stress, and brain development: New directions for prevention and intervention. *Academic Pediatrics*, *16*(3), S30-S36.

Brady, S. S. (2008). Lifetime family violence exposure is associated with current symptoms of eating disorders among both young men and women. *Journal of Traumatic Stress*, *21*(3), 347-351.

Brewerton, T. D. (2015) Stress, trauma, and adversity as risk factors in the development of eating disorders. In L. Smolak & M. Levine (Eds). *Wiley Handbook of Eating Disorders* (pp. 445-460). Guilford Press, New York.

Brewerton, T. D. (2019). An overview of trauma-informed care and practice for eating disorders. *Journal of Aggression, Maltreatment & Trauma*, *28*(4), 445-462.

Brockmeyer, T., Skunde, M., Wu, M., Bresslein, E., Rudofsky, G., Herzog, W., & Friederich, H. C. (2014). Difficulties in emotion regulation across the spectrum of eating disorders. *Comprehensive Psychiatry*, *55*(3), 565-571.

Bronfenbrenner, U., & Ceci, S. J. (1994). Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review*, *101*(4), 568-586.

Bronfenbrenner, U., & Morris, P. A. (2007). The bioecological model of human development. In W. Damon & R. M. Lerner (Eds.), *Handbook of child psychology* (pp. 793-828). John Wiley & Sons, Inc.

Browne, M. W. & Cudeck, R. (1993). Alternative ways of assessing model fit. In: K. A. Bollen & J. S. Long (Eds.), *Testing Structural Equation Models* (pp. 136–162). Beverly Hills, CA: Sage.

Brownstone, L. M., & Bardone-Cone, A. M. (2020). Subjective binge eating: A marker of disordered eating and broader psychological distress. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, 1-9.

Bundak, R., Darendeliler, F., Gunoz, H., Bas, F., Saka, N., & Neyzi, O. (2007). Analysis of puberty and pubertal growth in healthy boys. *European Journal of Pediatrics*, *166*(6), 595-600.

Burke, N. L., Hazzard, V. M., Schaefer, L. M., Simone, M., O'Flynn, J. L., & Rodgers, R. F. (2022). Socioeconomic status and eating disorder prevalence: At the intersections of gender identity, sexual orientation, and race/ethnicity. *Psychological Medicine*, 1-11.

Burnette, C. B., Burt, S. A., & Klump, K. L. (2023). The ignored role of disadvantage in eating disorders. *Trends in Molecular Medicine*.

Burt, S. A. (2014). Research review: The shared environment as a key source of variability in child and adolescent psychopathology. *Journal of Child Psychology and Psychiatry*, *55*(4), 304-312.

Burt, S. A., Clark, D. A., Pearson, A. L., Klump, K. L., & Neiderhiser, J. M. (2020). Do neighborhood social processes moderate the etiology of youth conduct problems? *Psychological Medicine*, *50*(9), 1519-1529.

Burt, S. A., & Klump, K. L. (2013). The Michigan state university twin registry (MSUTR): An update. *Twin Research and Human Genetics*, *16*(1), 344-350.

Burt, S. A., & Klump, K. L. (2019). The Michigan State University Twin Registry (MSUTR): 15 years of twin and family research. *Twin Research and Human Genetics*, *22*(6), 741-745.

Burt, S. A., Klump, K. L., Vazquez, A. Y., Shewark, E. A., & Hyde, L. W. (2021). Identifying patterns of youth resilience to neighborhood disadvantage. *Research in Human Development*, *18*(3), 181-196.

Burt, S. A., Pearson, A. L., Carroll, S., Klump, K. L., & Neiderhiser, J. M. (2020). Child antisocial behavior is more environmental in origin in disadvantaged neighborhoods: Evidence across residents' perceptions and geographic scales in two samples. *Journal of Abnormal Child Psychology*, *48*(2), 265-276.

Burt, S. A., Thaler, D., Shewark, E. A., Pearson, A. L., Anaya, C., Tomlinson, R. C.,...Lonstein, J. S. (2023). Illuminating associations between parenting and deleterious neighborhood characteristics via exhaustive modeling. *Journal of Marriage and Family*, *85*(1), 153-172.

Callaghan, B. L., & Tottenham, N. (2016). The stress acceleration hypothesis: Effects of early-life adversity on emotion circuits and behavior. *Current Opinion in Behavioral Sciences*, *7*, 76-81.

Carroll, S. L., Clark, D. A., Hyde, L. W., Klump, K. L., & Burt, S. A. (2021). Continuity and change in the genetic and environmental etiology of youth antisocial behavior. *Behavior Genetics*, 1-12.

Carroll, S. L., Shewark, E. A., Mikhail, M. E., Thaler, D. J., Pearson, A. L., Klump, K. L., & Burt, S. A. (2023). Identifying the 'active ingredients' of socioeconomic disadvantage for youth outcomes in middle childhood. *Development and Psychopathology*, 1-9.

Carskadon, M. A., & Acebo, C. (1993). A self-administered rating scale for pubertal development. *Journal of Adolescent Health*, *14*(3), 190-195.

Carter, M. A., Dubois, L., Tremblay, M. S., & Taljaard, M. (2012). Local social environmental factors are associated with household food insecurity in a longitudinal study of children. *BMC Public Health*, *12*(1), 1-11.

Choi, J. K., Teshome, T., & Smith, J. (2021). Neighborhood disadvantage, childhood adversity, bullying victimization, and adolescent depression: A multiple mediational analysis. *Journal of Affective Disorders*, *279*, 554-562.

Chu, S. H., Loucks, E. B., Kelsey, K. T., Gilman, S. E., Agha, G., Eaton, C. B.,...Huang, Y. T. (2018). Sex-specific epigenetic mediators between early life social disadvantage and adulthood BMI. *Epigenomics*, *10*(6), 707-722.

Coffino, J. A., Grilo, C. M., & Udo, T. (2020). Childhood food neglect and adverse experiences associated with DSM-5 eating disorders in US national sample. *Journal of Psychiatric Research*, *127*, 75-79.

Cooksey-Stowers, K., Schwartz, M. B., & Brownell, K. D. (2017). Food swamps predict obesity rates better than food deserts in the United States. *International Journal of Environmental Research and Public Health*, *14*(11), 1366.

Couturier, J., Lock, J., Forsberg, S., Vanderheyden, D., & Yen, H. L. (2007). The addition of a parent and clinician component to the eating disorder examination for children and adolescents. *International Journal of Eating Disorders*, *40*(5), 472-475.

Culbert, K. M., Burt, S. A., & Klump, K. L. (2017). Expanding the developmental boundaries of etiologic effects: The role of adrenarche in genetic influences on disordered eating in males. *Journal of Abnormal Psychology*, *126*(5), 593-606.

Culbert, K. M., Burt, S. A., Sisk, C. L., Nigg, J. T., & Klump, K. L. (2014). The effects of circulating testosterone and pubertal maturation on risk for disordered eating symptoms in adolescent males. *Psychological Medicine*, *44*(11), 2271-2286.

Culbert, K. M., Shope, M. M., Sisk, C. L., & Klump, K. L. (2020). Low testosterone is associated with dysregulated eating symptoms in young adult men. *International Journal of Eating Disorders*, *53*(9), 1469-1479.

Cutrona, C. E., Wallace, G., & Wesner, K. A. (2006). Neighborhood characteristics and depression: An examination of stress processes. *Current Directions in Psychological Science*, *15*(4), 188-192.

Darby, A., Hay, P., Mond, J., Rodgers, B., & Owen, C. (2007). Disordered eating behaviours and cognitions in young women with obesity: Relationship with psychological status. *International Journal of Obesity*, *31*(5), 876-882.

Dark, H. E., Harnett, N. G., Goodman, A. M., Wheelock, M. D., Mrug, S., Schuster, M. A., ...Knight, D. C. (2020). Violence exposure, affective style, and stress-induced changes in resting state functional connectivity. *Cognitive, Affective, & Behavioral Neuroscience*, *20*, 1261-1277.

Darwish, A., Salmon, G., Ahuja, A., & Steed, L. (2006). The community intensive therapy team: Development and philosophy of a new service. *Clinical Child Psychology and Psychiatry*, *11*(4), 591-605.

Davison, K. K., Markey, C. N., & Birch, L. L. (2003). A longitudinal examination of patterns in girls' weight concerns and body dissatisfaction from ages 5 to 9 years. *International Journal of Eating Disorders*, *33*(3), 320-332.

DeCarlo Santiago, C., Wadsworth, M. E., & Stump, J. (2011). Socioeconomic status, neighborhood disadvantage, and poverty-related stress: Prospective effects on psychological syndromes among diverse low-income families. *Journal of Economic Psychology*, *32*(2), 218-230.

Delmelle, E. C. (2016). Mapping the DNA of urban neighborhoods: Clustering longitudinal sequences of neighborhood socioeconomic change. *Annals of the American Association of Geographers*, *106*(1), 36-56.

Denney, J. T., Kimbro, R. T., & Sharp, G. (2018). Neighborhoods and food insecurity in households with young children: A disadvantage paradox? *Social Problems*, *65*(3), 342-359.

Donnelly, B., Touyz, S., Hay, P., Burton, A., Russell, J., & Caterson, I. (2018). Neuroimaging in bulimia nervosa and binge eating disorder: A systematic review. *Journal of Eating Disorders*, *6*, 1-24.

Dubowitz, T., Ghosh-Dastidar, M., Eibner, C., Slaughter, M. E., Fernandes, M., Whitsel, E. A.,...Escarce, J. J. (2012). The Women's Health Initiative: The food environment, neighborhood socioeconomic status, BMI, and blood pressure. *Obesity*, *20*(4), 862-871.

Egede, L. E., Walker, R. J., Campbell, J. A., Linde, S., Hawks, L. C., & Burgess, K. M. (2023). Modern day consequences of historic redlining: Finding a path forward. *Journal of General Internal Medicine*, 1-4.

Elías Alvarado, S. (2016). Delayed disadvantage: Neighborhood context and child development. *Social Forces*, *94*(4), 1847-1877.

Enders, C. K., & Bandalos, D. L. (2001). The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Structural Equation Modeling*, *8*(3), 430-457.

Evans, E. H., Tovée, M. J., Boothroyd, L. G., & Drewett, R. F. (2013). Body dissatisfaction and disordered eating attitudes in 7-to 11-year-old girls: Testing a sociocultural model. *Body Image*, *10*(1), 8-15.

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders*, *16*(4), 363-370.

Fairweather-Schmidt, A. K., & Wade, T. D. (2017). Weight-related peer-teasing moderates genetic and environmental risk and disordered eating: Twin study. *The British Journal of Psychiatry*, *210*(5), 350-355.

Falk, C. F. (2018). Are robust standard errors the best approach for interval estimation with non-normal data in structural equation modeling? *Structural Equation Modeling*, *25*(2), 244-266.

Flowers, A. L., Hastings, T. L., & Kelley, M. L. (2000). Development of a screening instrument for exposure to violence in children: The KID-SAVE. *Journal of Psychopathology and Behavioral Assessment*, *22*, 91-104.

Frayn, M., Trainor, C., Lin, M., Pitts, A., Drexler, S. A., Patarinski, A. G. G., & Juarascio, A. (2022). Patient perceptions of the relationship between food insecurity and eating disorder treatment: A qualitative exploration. *International Journal of Eating Disorders*, *55*(3), 332-342.

Gadalla, T. M. (2009). Eating disorders in men: A community-based study. *International Journal of Men's Health*, *8*(1).

Galobardes, B., Lynch, J. W., & Davey Smith, G. (2004). Childhood socioeconomic circumstances and cause-specific mortality in adulthood: Systematic review and interpretation. *Epidemiologic Reviews*, *26*(1), 7-21.

Gard, M. C., & Freeman, C. P. (1996). The dismantling of a myth: A review of eating disorders and socioeconomic status. *International Journal of Eating Disorders*, *20*(1), 1-12.

Garvin, E., Branas, C., Keddem, S., Sellman, J., & Cannuscio, C. (2013). More than just an eyesore: Local insights and solutions on vacant land and urban health. *Journal of Urban Health*, *90*, 412-426.

Gillespie, C. F., Phifer, J., Bradley, B., & Ressler, K. J. (2009). Risk and resilience: Genetic and environmental influences on development of the stress response. *Depression and Anxiety*, *26*(11), 984-992.

Goodman, E., McEwen, B. S., Dolan, L. M., Schafer-Kalkhoff, T., & Adler, N. E. (2005). Social disadvantage and adolescent stress. *Journal of Adolescent Health*, *37*(6), 484-492.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A research note. *Journal of Child Psychology and Psychiatry*, *38*(5), 581-586.

Goodnight, J. A., Lahey, B. B., Van Hulle, C. A., Rodgers, J. L., Rathouz, P. J., Waldman, I. D., & D'Onofrio, B. M. (2012). A quasi-experimental analysis of the influence of neighborhood disadvantage on child and adolescent conduct problems. *Journal of Abnormal Psychology*, *121*(1), 95-108.

Gordon, N. P., & Mellor, R. G. (2015). Accuracy of parent-reported information for estimating prevalence of overweight and obesity in a race-ethnically diverse pediatric clinic population aged 3 to 12. *BMC Pediatrics*, *15*(1), 1-11.

Guran, T., Firat, I., Yildiz, F., Kaplan Bulut, I., Dogru, M., & Bereket, A. (2015). Reference values for serum dehydroepiandrosterone-sulphate in healthy children and adolescents with emphasis on the age of adrenarche and pubarche. *Clinical Endocrinology*, *82*(5), 712-718.

Hackman, D. A., Betancourt, L. M., Brodsky, N. L., Hurt, H., & Farah, M. J. (2012). Neighborhood disadvantage and adolescent stress reactivity. *Frontiers in Human Neuroscience*, *6*, 277.

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin*, *137*(4), 660-681.

Hart, L. M., Gordon, A. R., Sarda, V., Calzo, J. P., Sonneville, K. R., Samnaliev, M., & Austin, S. B. (2020). The association of disordered eating with health-related quality of life in US young adults and effect modification by gender. *Quality of Life Research*, 1-13.

Hazzard, V. M., Barry, M. R., Leung, C. W., Sonneville, K. R., Wonderlich, S. A., & Crosby, R. D. (2021). Food insecurity and its associations with bulimic-spectrum eating disorders, mood disorders, and anxiety disorders in a nationally representative sample of US adults. *Social Psychiatry and Psychiatric Epidemiology*, 1-8.

Hazzard, V. M., Hooper, L., Larson, N., Loth, K. A., Wall, M. M., & Neumark-Sztainer, D. (2022). Associations between severe food insecurity and disordered eating behaviors from adolescence to young adulthood: Findings from a 10-year longitudinal study. *Preventive Medicine*, *154*, 106895.

Hazzard, V. M., Loth, K. A., Hooper, L., & Becker, C. B. (2020). Food insecurity and eating disorders: A review of emerging evidence. *Current Psychiatry Reports*, *22*, 1-9.

Heidinger, L. S., & Willson, A. E. (2022). The lasting imprint of childhood disadvantage: Cumulative histories of exposure to childhood adversity and trajectories of psychological distress in adulthood. *Longitudinal and Life Course Studies*, *13*(1), 121-144.

Henry, D., Gorman-Smith, D., Schoeny, M., & Tolan, P. (2014). "Neighborhood matters": Assessment of neighborhood social processes. *American Journal of Community Psychology*, *54*(3-4), 187-204.

Homan, P., Brown, T. H., & King, B. (2021). Structural intersectionality as a new direction for health disparities research. *Journal of Health and Social Behavior*, *62*(3), 350-370.

Hooper, L., Mason, S. M., Telke, S., Larson, N., & Neumark-Sztainer, D. (2022). Experiencing household food insecurity during adolescence predicts disordered eating and elevated body mass index 8 years later. *Journal of Adolescent Health*, *70*(5), 788-795.

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives, *Structural Equation Modeling*, *6*, 1-55.

Hu, J., Kind, A. J., & Nerenz, D. (2018). Area deprivation index predicts readmission risk at an urban teaching hospital. *American Journal of Medical Quality*, *33*(5), 493-501.

Huryk, K. M., Drury, C. R., & Loeb, K. L. (2021). Diseases of affluence? A systematic review of the literature on socioeconomic diversity in eating disorders. *Eating Behaviors*, 101548.

Ilondo, M. M., Vanderschueren-Lodeweyckx, M., Vlietinck, R., Pizarro, M., Malvaux, P., Eggermont, E., & Eeckels, R. (1982). Plasma androgens in children and adolescents. *Hormones*, *16*(2), 61-77.

Isaksson, J., Isaksson, M., Stickley, A., Vermeiren, R., Koposov, R., Schwab-Stone, M., & Ruchkin, V. (2023). Community violence exposure and eating disorder symptoms among Belgian, Russian and US adolescents: Cross-country and gender perspectives. *Child Psychiatry & Human Development*, 1-10.

Isaksson, M., Isaksson, J., Schwab-Stone, M., & Ruchkin, V. (2024). Longitudinal associations between community violence exposure, posttraumatic stress symptoms, and eating disorder symptoms. *Journal of Eating Disorders*, *12*(1), 6.

Jimenez, M. P., Wellenius, G. A., Subramanian, S. V., Buka, S., Eaton, C., Gilman, S. E., & Loucks, E. B. (2019). Longitudinal associations of neighborhood socioeconomic status with cardiovascular risk factors: A 46-year follow-up study. *Social Science & Medicine*, *241*, 112574.

Kaplan, D., Kim, J.-S., & Kim, S.-Y. (2009). Multilevel latent variable modeling: Current research and recent developments. In R. E. Millsap & A. Maydeu-Olivares (Eds.), *The Sage handbook of quantitative methods in psychology* (pp. 592–612). Sage Publications Ltd.

Karas, D. (2015). Highway to inequity: the disparate impact of the interstate highway system on poor and minority communities in American cities. *New Visions for Public Affairs*, *7*(April), 9-21.

Kind, A. J., & Buckingham, W. R. (2018). Making neighborhood-disadvantage metrics accessible—the neighborhood atlas. *The New England Journal of Medicine*, *378*(26), 2456-2458.

Kind, A. J., Jencks, S., Brock, J., Yu, M., Bartels, C., Ehlenbach, W.,...Smith, M. (2014). Neighborhood socioeconomic disadvantage and 30-day rehospitalization: A retrospective cohort study. *Annals of Internal Medicine*, *161*(11), 765-774.

King, C., Huang, X., & Dewan, N. A. (2022). Continuity and change in neighborhood disadvantage and adolescent depression and anxiety. *Health & Place*, *73*, 102724.

Kirby, J. B., & Kaneda, T. (2005). Neighborhood socioeconomic disadvantage and access to health care. *Journal of Health and Social Behavior*, *46*(1), 15-31.

Kirkpatrick, S. I., & Tarasuk, V. (2010). Assessing the relevance of neighbourhood characteristics to the household food security of low-income Toronto families. *Public Health Nutrition*, *13*(7), 1139-1148.

Klump, K. L. (2013). Puberty as a critical risk period for eating disorders: A review of human and animal studies. *Hormones and Behavior*, *64*(2), 399-410.

Klump, K. L., & Burt, S. A. (2006). The Michigan State University Twin Registry (MSUTR): Genetic, environmental and neurobiological influences on behavior across development. *Twin Research and Human Genetics*, *9*(6), 971-977.

Klump, K. L., Culbert, K. M., & Sisk, C. L. (2017). Sex differences in binge eating: Gonadal hormone effects across development. *Annual Review of Clinical Psychology*, *13*, 183-207.

Klump, K. L., Culbert, K. M., Slane, J. D., Burt, S. A., Sisk, C. L., & Nigg, J. T. (2012). The effects of puberty on genetic risk for disordered eating: Evidence for a sex difference. *Psychological Medicine*, *42*(3), 627-637.

Klump, K. L., Fowler, N., Mayhall, L., Sisk, C. L., Culbert, K. M., & Burt, S. A. (2018). Estrogen moderates genetic influences on binge eating during puberty: Disruption of normative processes? *Journal of Abnormal Psychology*, *127*(5), 458–470.

Klump, K. L., McGue, M., & Iacono, W. G. (2003). Differential heritability of eating attitudes and behaviors in prepubertal versus pubertal twins. *International Journal of Eating Disorders*, *33*(3), 287-292.

Klump, K. L., Perkins, P. S., Burt, S. A., McGue, M. A. T. T., & Iacono, W. G. (2007). Puberty moderates genetic influences on disordered eating. *Psychological Medicine*, *37*(5), 627-634.

Knez, R., Munjas, R., Petrovečki, M., Paučić-Kirinčić, E., & Peršić, M. (2006). Disordered eating attitudes among elementary school population. *Journal of Adolescent Health*, *38*(5), 628-630

Kohen, D. E., Leventhal, T., Dahinten, V. S., & McIntosh, C. N. (2008). Neighborhood disadvantage: Pathways of effects for young children. *Child Development*, *79*(1), 156-169.

Koopman-Verhoeff, M. E., Gredvig-Ardito, C., Barker, D. H., Saletin, J. M., & Carskadon, M. (2020). Classifying pubertal development using child and parent report: Comparing the Pubertal Development Scales to Tanner staging. *Journal of Adolescent Health*, *66*, 597-602.

Kravitz-Wirtz, N., Bruns, A., Aubel, A. J., Zhang, X., & Buggs, S. A. (2022). Inequities in community exposure to deadly gun violence by race/ethnicity, poverty, and neighborhood disadvantage among youth in large US cities. *Journal of Urban Health*, *99*(4), 610-625.

Kundakovic, M., & Champagne, F. A. (2015). Early-life experience, epigenetics, and the developing brain. *Neuropsychopharmacology*, *40*(1), 141-153.

Lacey, R. E., Howe, L. D., Kelly-Irving, M., Bartley, M., & Kelly, Y. (2020). The clustering of adverse childhood experiences in the Avon Longitudinal Study of Parents and Children: Are gender and poverty important? *Journal of Interpersonal Violence*, 0886260520935096.

Le Grange, D., & Loeb, K. L. (2007). Early identification and treatment of eating disorders: Prodrome to syndrome. *Early Intervention in Psychiatry*, *1*(1), 27-39.

Lee, M. R., Maume, M. O., & Ousey, G. C. (2003). Social isolation and lethal violence across the metro/nonmetro divide: The effects of socioeconomic disadvantage and poverty concentration on homicide. *Rural Sociology*, *68*(1), 107-131.

Lev-Ari, L., Zohar, A. H., & Bachner-Melman, R. (2021). Eating for numbing: A community-based study of trauma exposure, emotion dysregulation, dissociation, body dissatisfaction and eating disorder symptoms. *PeerJ*, *9*, e11899.

Li, X., Fu, Q., Leigh, I., & Humphrey, D. (2021). A latent class analysis of perceived neighborhood conditions associated with mental disorders among children in the United States. *Child Psychiatry & Human Development*, *52*, 355-364.

Liu, S. R., Kia-Keating, M., Santacrose, D. E., & Modir, S. (2018). Linking profiles of neighborhood elements to health and related outcomes among children across the United States. *Health & Place*, *53*, 203-209.

Lo Sauro, C., Ravaldi, C., Cabras, P. L., Faravelli, C., & Ricca, V. (2008). Stress, hypothalamic-pituitary-adrenal axis and eating disorders. *Neuropsychobiology*, *57*(3), 95-115.

Loucks, E. B., Huang, Y. T., Agha, G., Chu, S., Eaton, C. B., Gilman, S. E.,...Kelsey, K. T. (2016). Epigenetic mediators between childhood socioeconomic disadvantage and mid-life body mass index: The New England family study. *Psychosomatic Medicine*, *78*(9), 1053.

Luo, X., Donnellan, M. B., Burt, S. A., & Klump, K. L. (2016). The dimensional nature of eating pathology: Evidence from a direct comparison of categorical, dimensional, and hybrid models. *Journal of Abnormal Psychology*, *125*(5), 715-726.

Lydecker, J. A., & Grilo, C. M. (2019). Food insecurity and bulimia nervosa in the United States. *International Journal of Eating Disorders*, *52*(6), 735-739.

Lykken, D. T., Bouchard, T. J., McGue, M., & Tellegen, A. (1990). The Minnesota twin family registry: Some initial findings. *Acta Geneticae Medicae et Gemellologiae: Twin Research*, *39*(1), 35-70.

Mangweth-Matzek, B., Hoek, H. W., Rupp, C. I., Lackner-Seifert, K., Frey, N., Whitworth, A. B.,...Kinzl, J. (2014). Prevalence of eating disorders in middle-aged women. *International Journal of Eating Disorders*, *47*(3), 320-324.

Mangweth-Matzek, B., Kummer, K. K., & Pope, H. G. (2016). Eating disorder symptoms in middle-aged and older men. *International Journal of Eating Disorders*, *49*(10), 953-957.

Mayer, V. L., Hillier, A., Bachhuber, M. A., & Long, J. A. (2014). Food insecurity, neighborhood food access, and food assistance in Philadelphia. *Journal of Urban Health*, *91*, 1087-1097.

McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation: Central role of the brain. *Physiological Reviews*, *87*(3), 873-904.

McEwen, B. S. (2013). The brain on stress: Toward an integrative approach to brain, body, and behavior. *Perspectives on Psychological Science*, *8*(6), 673-675.

Melchior, M., Chastang, J. F., Walburg, V., Arseneault, L., Galéra, C., & Fombonne, E. (2010). Family income and youths' symptoms of depression and anxiety: A longitudinal study of the French GAZEL Youth cohort. *Depression and Anxiety*, *27*(12), 1095-1103.

Mikhail, M. E., Ackerman, L. S., Anaya, C., Culbert, K. M., Burt, S. A., & Klump, K. L. (In press). Associations between household income and disordered eating differ across sex and racial identity in a population-based sample of adults. *International Journal of Eating Disorders*.

Mikhail, M. E., Anaya, C., Culbert, K. M., Sisk, C. L., Johnson, A., & Klump, K. L. (2021). Gonadal hormone influences on sex differences in binge eating across development. *Current Psychiatry Reports*, *23*(11), 1-10.

Mikhail, M. E., Carroll, S. L., Clark, D. A., O'Connor, S., Burt, S. A., & Klump, K. L. (2021). Context matters: Neighborhood disadvantage is associated with increased disordered eating and earlier activation of genetic influences in girls. *Journal of Abnormal Psychology*, *130*(8), 875–885.

Mikhail, M. E., Carroll, S. L., Clark, D. A., O'Connor, S. M., Culbert, K. M., Burt, S. A., & Klump, K. L. (2023). Disadvantage and disordered eating in boys: Examining phenotypic and genotype × environment associations across development. *Journal of Psychopathology and Clinical Science*, *132*(1), 51–62.

Mikhail, M. E., & Klump, K. L. (2021). A virtual issue highlighting eating disorders in people of black/African and Indigenous heritage. *International Journal of Eating Disorders*, *54*(3), 459-467.

Mikhail, M. E., Klump, K. L., Hyde, L. W., & Burt, S. A. (Under review). Neighborhood disadvantage shapes trajectories of disordered eating from middle childhood to emerging adulthood in female and male youth.

Miller, A., Pohlig, R. T., & Reisman, D. S. (2020). Social and physical environmental factors in daily stepping activity in those with chronic stroke. *Topics in Stroke Rehabilitation*, 1-9.

Mitchell, K. S., Porter, B., Boyko, E. J., & Field, A. E. (2016). Longitudinal associations among posttraumatic stress disorder, disordered eating, and weight gain in military men and women. *American Journal of Epidemiology*, *184*(1), 33-47.

Mond, J., Hall, A., Bentley, C., Harrison, C., Gratwick-Sarll, K., & Lewis, V. (2014). Eating-disordered behavior in adolescent boys: Eating disorder examination questionnaire norms. *International Journal of Eating Disorders*, *47*(4), 335-341.

Muthén, L.K. and Muthén, B.O. (1998-2021). Mplus version 8.6. Los Angeles, CA: Muthén & Muthén.

Nagata, J. M., Ganson, K. T., & Murray, S. B. (2020). Eating disorders in adolescent boys and young men: An update. *Current Opinion in Pediatrics*, *32*(4), 476-481.

Nagl, M., Jacobi, C., Paul, M., Beesdo-Baum, K., Höfler, M., Lieb, R., & Wittchen, H. U. (2016). Prevalence, incidence, and natural course of anorexia and bulimia nervosa among adolescents and young adults. *European Child & Adolescent Psychiatry*, *25*(8), 903-918.

Najman, J. M., Hayatbakhsh, M. R., Clavarino, A., Bor, W., O'Callaghan, M. J., & Williams, G. M. (2010). Family poverty over the early life course and recurrent adolescent and young adult anxiety and depression: A longitudinal study. *American Journal of Public Health*, *100*(9), 1719-1723.

Neumark-Sztainer, D., & Hannan, P. J. (2000). Weight-related behaviors among adolescent girls and boys: Results from a national survey. *Archives of Pediatrics & Adolescent Medicine*, *154*(6), 569-577.

Neumark-Sztainer, D. R., Wall, M. M., Haines, J. I., Story, M. T., Sherwood, N. E., & van den Berg, P. A. (2007). Shared risk and protective factors for overweight and disordered eating in adolescents. *American Journal of Preventive Medicine*, *33*(5), 359-369.

Newacheck, P. W., Hung, Y. Y., Jane Park, M., Brindis, C. D., & Irwin Jr, C. E. (2003). Disparities in adolescent health and health care: Does socioeconomic status matter? *Health Services Research*, *38*(5), 1235-1252.

Non, A. L., Román, J. C., Gross, C. L., Gilman, S. E., Loucks, E. B., Buka, S. L., & Kubzansky, L. D. (2016). Early childhood social disadvantage is associated with poor health behaviours in adulthood. *Annals of Human Biology*, *43*(2), 144-153.

Oberndorfer, T. A., Kaye, W. H., Simmons, A. N., Strigo, I. A., & Matthews, S. C. (2011). Demand-specific alteration of medial prefrontal cortex response during an inhibition task in recovered anorexic women. *International Journal of Eating Disorders*, *44*(1), 1-8.

O'Connor, S. M., Culbert, K. M., Mayhall, L. A., Burt, S. A., & Klump, K. L. (2020). Differences in genetic and environmental influences on body weight and shape concerns across pubertal development in females. *Journal of Psychiatric Research*, *121*, 39-46.

Olson, J. R., Benjamin, P. H., Azman, A. A., Kellogg, M. A., Pullmann, M. D., Suter, J. C., & Bruns, E. J. (2021). Systematic review and meta-analysis: Effectiveness of wraparound care coordination for children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, *60*(11), 1353-1366.

Overstreet, S. (2000). Exposure to community violence: Defining the problem and understanding the consequences. *Journal of Child and Family Studies*, *9*(1), 7-25.

Pearson, A. L., Shewark, E. A., & Burt, S. A. (2022). Associations between neighborhood built, social, or toxicant conditions and child externalizing behaviors in the Detroit metro area: A cross-sectional study of the neighborhood 'exposome'. *BMC Public Health*, *22*(1), 1-13.

Peeters, H., Van Gestel, S., Vlietinck, R., Derom, C., & Derom, R. (1998). Validation of a telephone zygosity questionnaire in twins of known zygosity. *Behavior Genetics*, *28*(3), 159-163.

Pehlivan, M. J., Miskovic-Wheatley, J., Le, A., Maloney, D., Research Consortium, N. E. D., Touyz, S., & Maguire, S. (2022). Models of care for eating disorders: Findings from a rapid review. *Journal of Eating Disorders*, *10*(1), 166.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, *17*(2), 117-133.

Poulton, R., Caspi, A., Milne, B. J., Thomson, W. M., Taylor, A., Sears, M. R., & Moffitt, T. E. (2002). Association between children's experience of socioeconomic disadvantage and adult health: A life-course study. *The Lancet*, *360*(9346), 1640-1645.

Powell, W. R., Buckingham, W. R., Larson, J. L., Vilen, L., Yu, M., Salamat, M. S.,...Kind, A. J. (2020). Association of neighborhood-level disadvantage with Alzheimer Disease neuropathology. *JAMA Network Open*, *3*(6), e207559-e207559.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, (3), 879-891.

Purcell, S. (2002). Variance components models for gene–environment interaction in twin analysis. *Twin Research and Human Genetics*, *5*(6), 554-571.

Radunz, M., Pritchard, L., Steen, E., Williamson, P., & Wade, T. D. (2021). Evaluating evidence-based interventions in low socio-economic-status populations. *International Journal of Eating Disorders*, *54*(10), 1887-1895.

Reda, M. H., Marusak, H. A., Ely, T. D., van Rooij, S. J. H., Stenson, A. F., Stevens, J. S.,...Jovanovic, T. (2021). Community violence exposure is associated with hippocampus–insula resting state functional connectivity in urban youth. *Neuroscience*, *468*, 149-157.

Reiss, F. (2013). Socioeconomic inequalities and mental health problems in children and adolescents: A systematic review. *Social Science & Medicine*, *90*, 24-31.

Rende, R., & Plomin, R. (1992). Diathesis-stress models of psychopathology: A quantitative genetic perspective. *Applied and Preventive Psychology*, *1*(4), 177-182.

Ridley, M., Rao, G., Schilbach, F., & Patel, V. (2020). Poverty, depression, and anxiety: Causal evidence and mechanisms. *Science*, *370*(6522).

Rodríguez, M., Pérez, V., & García, Y. (2005). Impact of traumatic experiences and violent acts upon response to treatment of a sample of Colombian women with eating disorders. *International Journal of Eating Disorders*, *37*(4), 299-306.

Roscigno, V. J., Karafin, D. L., & Tester, G. (2009). The complexities and processes of racial housing discrimination. *Social Problems*, *56*(1), 49-69.

Rose, V. K., & Thompson, L. M. (2012). Space, place and people: A community development approach to mental health promotion in a disadvantaged community. *Community Development Journal*, *47*(4), 604-611.

Ross, C. E., Mirowsky, J., & Pribesh, S. (2001). Powerlessness and the amplification of threat: Neighborhood disadvantage, disorder, and mistrust. *American Sociological Review*, 568-591.

Roubinov, D. S., Hagan, M. J., Boyce, W. T., Adler, N. E., & Bush, N. R. (2018). Family socioeconomic status, cortisol, and physical health in early childhood: The role of advantageous neighborhood characteristics. *Psychosomatic Medicine*, *80*(5), 492-501.

Rucker, D. D., Preacher, K. J., Tormala, Z. L., & Petty, R. E. (2011). Mediation analysis in social psychology: Current practices and new recommendations. *Social and Personality Psychology Compass*, *5*(6), 359-371.

Salom, C. L., Williams, G. M., Najman, J. M., & Alati, R. (2014). Does early socio-economic disadvantage predict comorbid alcohol and mental health disorders? *Drug and Alcohol Dependence*, *142*, 146-153.

Sampson, R. J., Sharkey, P., & Raudenbush, S. W. (2008). Durable effects of concentrated disadvantage on verbal ability among African-American children. *Proceedings of the National Academy of Sciences*, *105*(3), 845-852.

Santarossa, S., Hill, A. B., Sitarik, A. R., Taylor, M., Hawkins, S., Scher, K.,...Joseph, C. L. (2022). Food insecurity in Detroit: exploring the relationship between patient-reported food insecurity and proximity to healthful grocery stores. *Public Health Nutrition*, *25*(4), 954-963.

Sareen, J., Jagdeo, A., Cox, B. J., Clara, I., ten Have, M., Belik, S. L.,...Stein, M. B. (2007). Perceived barriers to mental health service utilization in the United States, Ontario, and the Netherlands. *Psychiatric Services*, *58*(3), 357-364.

Satorra, A. (2000). Scaled and adjusted restricted tests in multi-sample analysis of moment structures. In Heijmans, R.D.H., Pollock, D.S.G. & Satorra, A. (eds.), *Innovations in multivariate statistical analysis*. A Festschrift for Heinz Neudecker (pp.233-247). London: Kluwer Academic Publishers.

Saxbe, D., Khoddam, H., Piero, L. D., Stoycos, S. A., Gimbel, S. I., Margolin, G., & Kaplan, J. T. (2018). Community violence exposure in early adolescence: Longitudinal associations with hippocampal and amygdala volume and resting state connectivity. *Developmental Science*, *21*(6), e12686.

Seabrook, J. A., & Avison, W. R. (2012). Socioeconomic status and cumulative disadvantage processes across the life course: Implications for health outcomes. *Canadian Review of Sociology/Revue canadienne de sociologie*, *49*(1), 50-68.

Schaefer, D. R., Simpkins, S. D., Vest, A. E., & Price, C. D. (2011). The contribution of extracurricular activities to adolescent friendships: New insights through social network analysis. *Developmental Psychology*, *47*(4), 1141.

Schulz, K. M., & Sisk, C. L. (2016). The organizing actions of adolescent gonadal steroid hormones on brain and behavioral development. *Neuroscience & Biobehavioral Reviews*, *70*, 148-158.

Sheets, L. R., Kelley, L. E. H., Scheitler-Ring, K., Petroski, G. F., Barnett, Y., Barnett, C.,... Parker, J. C. (2020). An index of geospatial disadvantage predicts both obesity and unmeasured body weight. *Preventive Medicine Reports*, *18*, 101067.

Shek, D. T., Dou, D., Zhu, X., & Chai, W. (2019). Positive youth development: Current perspectives. *Adolescent Health, Medicine and Therapeutics*, 131-141.

Shen, Y. (2022). Race/ethnicity, built environment in neighborhood, and children's mental health in the US. *International Journal of Environmental Health Research*, *32*(2), 277-291.

Shields, A. E. (2017). Epigenetic signals of how social disadvantage "gets under the skin": A challenge to the public health community. *Epigenomics*, *9*(3), 223–229.

Shields, M., Gorber, S. C., Janssen, I., & Tremblay, M. S. (2011). Obesity estimates for children based on parent-reported versus direct measures. *Health Reports*, *22*(3), 47.

Simanek, A. M., Meier, H. C., D'Aloisio, A. A., & Sandler, D. P. (2021). Objective and subjective childhood socioeconomic disadvantage and incident depression in adulthood: A longitudinal analysis in the Sister Study. *Social Psychiatry and Psychiatric Epidemiology*, 1-10.

Simone, M., Slaughter-Acey, J., Hazzard, V. M., Eisenberg, M., & Neumark-Sztainer, D. (2022). Exploring the intersection of multiple social determinants of health and disordered eating behaviors in a population-based sample in the United States. *International Journal of Eating Disorders*, *55*(11), 1589-1602.

Simone, M., Telke, S., Anderson, L. M., Eisenberg, M., & Neumark-Sztainer, D. (2022). Ethnic/racial and gender differences in disordered eating behavior prevalence trajectories among women and men from adolescence into adulthood. *Social Science & Medicine*, *294*, 114720.

Singh, G. K. (2003). Area deprivation and widening inequalities in US mortality, 1969–1998. *American Journal of Public Health*, *93*(7), 1137-1143.

Sivak, C. J., Pearson, A. L., & Hurlburt, P. (2021). Effects of vacant lots on human health: A systematic review of the evidence. *Landscape and Urban Planning*, *208*, 104020.

Slocum, L. A., Rengifo, A. F., Choi, T., & Herrmann, C. R. (2013). The elusive relationship between community organizations and crime: An assessment across disadvantaged areas of the South Bronx. *Criminology*, *51*(1), 167-216.

Smink, F. R., Van Hoeken, D., & Hoek, H. W. (2012). Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports*, *14*(4), 406-414.

Sonneville, K. R., & Lipson, S. K. (2018). Disparities in eating disorder diagnosis and treatment according to weight status, race/ethnicity, socioeconomic background, and sex among college students. *International Journal of Eating Disorders*, *51*(6), 518-526.

Stansfeld, S. A., & Matheson, M. P. (2003). Noise pollution: non-auditory effects on health. *British Medical Bulletin*, *68*(1), 243-257.

Steele, L., Dewa, C., & Lee, K. (2007). Socioeconomic status and self-reported barriers to mental health service use. *The Canadian Journal of Psychiatry*, *52*(3), 201-206.

Steinberg, E., Tanofsky-Kraff, M., Cohen, M. L., Elberg, J., Freedman, R. J., Semega-Janneh, M.,...Yanovski, J. A. (2004). Comparison of the child and parent forms of the Questionnaire on Eating and Weight Patterns in the assessment of children's eating-disordered behaviors. *International Journal of Eating Disorders*, *36*(2), 183-194.

Steward, T., Martínez-Zalacaín, I., Mestre-Bach, G., Sánchez, I., Riesco, N., Jiménez-Murcia, S.,...Fernandez-Aranda, F. (2022). Dorsolateral prefrontal cortex and amygdala function during cognitive reappraisal predicts weight restoration and emotion regulation impairment in anorexia nervosa. *Psychological Medicine*, *52*(5), 844-852.

Stice, E., Marti, C. N., Shaw, H., & Jaconis, M. (2009). An 8-year longitudinal study of the natural history of threshold, subthreshold, and partial eating disorders from a community sample of adolescents. *Journal of Abnormal Psychology*, *118*(3), 587-597.

Strachan, E., Duncan, G., Horn, E., & Turkheimer, E. (2017). Neighborhood deprivation and depression in adult twins: Genetics and gene x environment interaction. *Psychological Medicine*, *47*(4), 627-638.

Suarez, G. L., Burt, S. A., Gard, A. M., Burton, J., Clark, D. A., Klump, K. L., & Hyde, L. W. (2022). The impact of neighborhood disadvantage on amygdala reactivity: Pathways through neighborhood social processes. *Developmental Cognitive Neuroscience*, 101061.

Swanson, S. A., Aloisio, K. M., Horton, N. J., Sonneville, K. R., Crosby, R. D., Eddy, K. T.,...Micali, N. (2014). Assessing eating disorder symptoms in adolescence: Is there a role for multiple informants? *International Journal of Eating Disorders*, *47*(5), 475-482.

van der Sluis, S., Posthuma, D., & Dolan, C. V. (2012). A note on false positives and power in GxE modelling of twin data. *Behavior Genetics*, *42*(1), 170-186.

Thomas, K. S., Williams, M. O., & Vanderwert, R. E. (2021). Disordered eating and internalizing symptoms in preadolescence. *Brain and Behavior*, *11*(1), e01904.

Timonen, J., Niemelä, M., Hakko, H., Alakokkare, A., & Räsänen, S. (2021). Associations between adolescents' social leisure activities and the onset of mental disorders in young adulthood. *Journal of Youth and Adolescence*, *50*(9), 1757-1765.

Tortorella, A., Menculini, G., Moretti, P., Attademo, L., Balducci, P. M., Bernardini, F.,...Erfurth, A. (2022). New determinants of mental health: The role of noise pollution. A narrative review. *International Review of Psychiatry*, 1-14.

Trottier, K., & MacDonald, D. E. (2017). Update on psychological trauma, other severe adverse experiences and eating disorders: state of the research and future research directions. *Current Psychiatry Reports*, *19*, 1-9.

U.S. Census Bureau. *U.S. Census Bureau quickfacts: Michigan; United States*. (2022, July 1). Retrieved April 29, 2023 from https://www.Census.gov/quickfacts/fact/table/MI,US/PST045221

VanderWielen, L. M., Gilchrist, E. C., Nowels, M. A., Petterson, S. M., Rust, G., & Miller, B. F. (2015). Not near enough: Racial and ethnic disparities in access to nearby behavioral health care and primary care. *Journal of Health Care for the Poor and Underserved*, *26*(3), 1032-1047.

Vartanian, L. R., & Porter, A. M. (2016). Weight stigma and eating behavior: A review of the literature. *Appetite*, *102*, 3-14.

von Ranson, K. M., Klump, K. L., Iacono, W. G., & McGue, M. (2005). The Minnesota Eating Behavior Survey: A brief measure of disordered eating attitudes and behaviors. *Eating Behaviors*, *6*(4), 373-392.

Homan, P., Brown, T. H., & King, B. (2021). Structural intersectionality as a new direction for health disparities research. *Journal of Health and Social Behavior*, *62*(3), 350-370

Watson, R., Burgess, L., Sellars, E., Crooks, J., McGowan, R., Diffey, J.,...McMellon, C. (2023). A qualitative study exploring the benefits of involving young people in mental health research. *Health Expectations*.

Williams, D. R. (2002). Racial/ethnic variations in women's health: The social embeddedness of health. *American Journal of Public Health*, *92*(4), 588-597.

Woods, L. L. (2012). The Federal Home Loan Bank Board, redlining, and the national proliferation of racial lending discrimination, 1921–1950. *Journal of Urban History*, *38*(6), 1036-1059.

Xue, Y., Leventhal, T., Brooks-Gunn, J., & Earls, F. J. (2005). Neighborhood residence and mental health problems of 5-to 11-year-olds. *Archives of General Psychiatry*, *62*(5), 554 -563.

Zeal, M., & Terry, J. (2013). Knowing what I can do and who I can be: Youth identify transformational benefits of participatory action research. *Journal of Ethnographic & Qualitative Research*, *8*(1), 42-55.

Zelkowitz, R. L., Zerubavel, N., Zucker, N. L., & Copeland, W. E. (2021). Longitudinal associations of trauma exposure with disordered eating: lessons from the Great Smoky Mountains study. *Eating Disorders*, *29*(3), 208-225.

Zhang, Y., Mavoa, S., Zhao, J., Raphael, D., & Smith, M. (2020). The association between green space and adolescents' mental well-being: A systematic review. *International Journal of Environmental Research and Public Health*, *17*(18), 6640.

Zucker, N. L., & Bulik, C. M. (2020). On bells, saliva, and abdominal pain or discomfort: Early aversive visceral conditioning and vulnerability for anorexia nervosa. *International Journal of Eating Disorders*, *53*(4), 508-512.

Zuloaga, D. G., Heck, A. L., De Guzman, R. M., & Handa, R. J. (2020). Roles for androgens in mediating the sex differences of neuroendocrine and behavioral stress responses. *Biology of sex Differences*, *11*(1), 1-18.

APPENDIX A: SUPPLMENTAL MATERIAL FOR CHAPTER 1

Additional Information Regarding the Reliability and Validity of the MTP-ED in Boys

The MTP-ED was previously validated in a large, population-based sample of female twins (N = 2,922; Mikhail, Carroll, et al., 2021). For the current study, the MTP-ED was further validated in boys. The MTP-ED had acceptable internal consistency across age (ages 8-12: α = .77; ages 13-17; α = .81) and pubertal development (early adrenarche: α = .70; early gonadarche: α = .78; mid/late gonadarche: α = .80) in boys.

Exploratory factor analysis (EFA) with orthogonal varimax rotation yielded a single factor with an eigenvalue above 1 (factor 1 eigenvalue = 2.87, factor 2 eigenvalue = .31), suggesting that all MTP-ED items loaded on a single factor. Confirmatory factor analysis (CFA) of the nine MTP-ED items showed adequate fit for a single latent factor model in the full sample (RMSEA = .071; CFI = .941, TLI = .922, SRMR = .036). An alternative two-factor model suggested by the EFA that placed purging and dieting on a separate factor from the other disordered eating symptoms did not have appreciably better fit (RMSEA = .071; CFI = .943, TLI = .922, SRMR = .035), and so the single factor model was preferred due to parsimony. When comparing model fit across adrenarche, a model constraining all factor loadings to equality across early and late adrenarche had adequate fit (RMSEA = .072; CFI = .921, TLI = .919, SRMR = .067) that was similar to the fit of a model that allowed factor loadings to differ for boys in early and late adrenarche (RMSEA = .075; CFI = .925, TLI = .910, SRMR = .052) (AIC = 5492.107 for the constrained model and 5510.630 for the unconstrained model; BIC = 5782.110 for the constrained model and 5740.216 for the unconstrained model). The chi-square test comparing models with and without factor loadings constrained to equality across adrenarche was significant ($\chi 2$ = 38.52, p <.001), but this was likely due to the fact that the chi-square statistic is very sensitive to sample size and has a high likelihood of rejecting more parsimonious models when sample size is large

(Bentler & Bonett, 1980). Altogether, we concluded that the fit of a single latent factor model was adequate to enable analyses of disordered eating as a single composite scale across adrenarche in boys.

The MTP-ED showed expected correlations with age ($r = .13$, $p < .001$), pubertal status ($r = .13$, $p < .001$), BMI percentile ($r = .29$, $p < .001$), and internalizing symptoms (e.g., worry, depression; $r = .25$, $p < .001$) in boys that were similar to associations for other self-report measures of disordered eating (Mond et al., 2014; Neumark-Sztainer & Hannan, 2000; Thomas et al., 2021). The MTP-ED discriminated between boys with and without a lifetime parent-reported ED (AN, BN, or BED) on a checklist of physical and mental health conditions on the MTP intake questionnaire (*M*(*SD*) with no ED: .92 (1.92); *M*(*SD*) with lifetime ED: 3.32 (3.41); $p < .001$).

At the time the current study was conducted, 299 boys ages 7-18 and their primary caregiver from a separate, ongoing study (*Twin Study of Mood, Behavior, and Hormones in Males*) had completed the MTP-ED, with parents competing the MTP-ED in relation to their child and boys completing the MTP-ED in relation to themselves. Boys in this study also completed the Minnesota Eating Behavior Survey (MEBS; von Ranson et al., 2005), an established self-report measure of ED symptoms. Correlations were large between self-reported MTP-ED and self-reported MEBS total scores ($r = .66$, $p < .001$). As is typical in the ED literature, correlations between parent- and self-reported MTP-ED were significant but small-to-moderate in magnitude ($r = .26$, $p < .001$). While parent- and youth-reported symptoms represent somewhat distinct perspectives on a youth's disordered eating, prior research suggests parent-reported symptoms differentiate youth with and without clinical EDs (Accurso & Waller, 2021) and show similar or greater concordance with objective external measurements (e.g., BMI, clinician-reported symptoms) as adolescent-reported symptoms (Couturier et al., 2007; Steinberg et al., 2004;

Swanson et al., 2014). Parent report may be particularly useful for younger boys who may have difficulty understanding disordered eating items.

Tables

Table S1.1. *Descriptive statistics for participant demographics and symptoms (N =* 3,484*)*

Participant Characteristics	Mean (SD) or % of Sample (N)	Range
Age	12.27 (2.96)	8.05-17.99
Zygosity (N listed as number of pairs)		
Monozygotic	43.4% (756)	—
Dizygotic	56.5% (984)	—
Unknown zygosity	0.1% (2)	—
Race/ethnicity		
White (non-Latinx)	84.6% (2,948)	—
Black/African American (non-Latinx)	7.1% (248)	—
Latinx/Hispanic	1.4% (48)	—
Asian American	1.1% (38)	—
Native American/American Indian	0.3% (10)	—
More than one race	3.6% (124)	—
Other/Unknown	2.0% (68)	—
BMI percentile	55.28 (30.47)	0.5–99.5
Raw BMI	19.56 (4.16)	13.17–38.39
PDS score	2.01 (.88)	1–4
Categorical adrenarche status		
Early adrenarche	14.8% (495)	—
Late adrenarche	85.2% (2,847)	—
Combined parental income (in thousands of dollars)	$90.39 (54.41)	$0–$300+
Mother's education level		
Less than high school	2.9% (98)	—
High school graduate	15.8% (538)	—
Less than 4 years of college	33.6% (1,144)	—
College graduate (4-6 years of college)	34.9% (1,190)	—
Post-graduate education	12.9% (440)	—
Father's education level		
Less than high school	4.7% (148)	—
High school graduate	23.4% (746)	—
Less than 4 years of college	28.1% (896)	—
College graduate (4-6 years of college)	31.4% (1,000)	—
Post-graduate education	12.4% (394)	—
Area Deprivation Index (ADI) percentile rank relative to all census tracts in the United States	37.34 (26.39)	1–100

Table S1.1 (cont'd)

Symptom Measures	Mean (SD) or % of Sample (N)	Sample Range	Possible Range	Cronbach's alpha
MTP-ED total score	.94 (1.95)	0–15	0–18	.79
Reported having AN, BN, or BED	0.9% (31)	—	—	—
Reported being treated for AN, BN, or BED	0.4% (14)	—	—	—
Internalizing symptoms	1.47 (1.78)	0–10	0–10	.65

Note: PDS = Pubertal Development Scale; BMI = body mass index; MTP-ED = Michigan Twins Project Eating Disorder Survey; AN = anorexia nervosa; BN = bulimia nervosa; BED = binge-eating disorder; internalizing symptoms = score on the Emotional Symptoms subscale of the Strengths and Difficulties Questionnaire (Goodman et al., 1997). N's may not add up to the total N for all variables due to missing values. The lower percentage of participants with reported eating disorders likely reflects the young average age of the sample, as threshold eating disorders are very rare in boys prior to mid-adolescence (Smink et al., 2012).

Table S1.2. *MLMs examining associations between disadvantage and disordered eating, with neighborhood disadvantage and family SES included in the same model*

Controlling For Adrenarche									
BMI Not Included as a Covariate					***BMI Included as a Covariate***				
Variable	**β**	**SE**	***p***	**95% CI**	**Variable**	**β**	**SE**	***p***	**95% CI**
Intercept	-.30	.06	**<.001**	-.42, -.19	Intercept	-.29	.06	**<.001**	-.40, -.17
ADI	.07	.03	**.006**	.02, .13	ADI	.05	.03	**.048**	.0004, .10
Family SES	-.05	.03	**.048**	-.10, -.0005	Family SES	-.03	.03	.187	-.08, .02
Adrenarche status	.36	.06	**<.001**	.23, .48	Adrenarche status	.34	.06	**<.001**	.22, .47
Race/ethnicity					Race/ethnicity				
Black/African American (non-Latinx)	.02	.09	.802	-.16, .21	*Black/African American (non-Latinx)*	-.02	.10	.818	-.22, .17
Latinx/Hispanic	.22	.20	.267	-.17, .62	*Latinx/Hispanic*	.04	.22	.864	-.39, .46
Asian American	.02	.23	.922	-.44, .48	*Asian American*	.07	.24	.771	-.41, .55
Native American/ American Indian	-.17	.50	.735	-1.15, .81	*Native American/ American Indian*	-.23	.48	.630	-1.16, .70
More than one race	.006	.13	.960	-.24, .25	*More than one race*	.06	.13	.631	-.19, .31
Other/unknown	.34	.16	**.036**	.02, .66	*Other/unknown*	.48	.18	**.007**	.13, .83
					BMI Percentile	.28	.02	**<.001**	.24, .32
Adrenarche as a Moderator									
BMI Not Included as a Covariate					***BMI Included as a Covariate***				
Variable	**β**	**SE**	***p***	**95% CI**	**Variable**	**β**	**SE**	***p***	**95% CI**
Intercept	-.31	.06	**<.001**	-.44, -.19	Intercept	-.31	.06	**<.001**	-.44, -.19
ADI	-.03	.06	.663	-.15, .10	ADI	-.01	.06	.844	-.14, .11
Family SES	-.05	.07	.472	-.18, .08	Family SES	.03	.07	.608	-.10, .16
Adrenarche status	.37	.07	**<.001**	.24, .50	Adrenarche status	.37	.07	**<.001**	.24, .50
ADI x adrenarche	.12	.07	.080	-.01, .25	ADI x adrenarche	.08	.07	.262	-.06, .21
SES x adrenarche	-.005	.07	.946	-.14, .13	SES x adrenarche	-.08	.07	.264	-.22, .06
Race/ethnicity					Race/ethnicity				
Black/African American (non-Latinx)	.003	.10	.977	-.18, .19	*Black/African American (non-Latinx)*	-.04	.10	.702	-.23, .16

Table S1.2 (cont'd)

Latinx/Hispanic	.21	.20	.304	-.19, .60	*Latinx/Hispanic*	.02	.22	.942	-.41, .44
Asian American	.03	.23	.892	-.43, .49	*Asian American*	.07	.24	.759	-.40, .55
Native American/ American Indian	-.19	.50	.704	-1.17, .79	*Native American/ American Indian*	-.24	.48	.617	-1.17, .69
More than one race	-.003	.13	.984	-.25, .24	*More than one race*	.05	.13	.697	-.20, .30
Other/unknown	.35	.16	**.031**	.03, .67	*Other/unknown*	.49	.18	**.006**	.14, .83
					BMI Percentile	.29	.02	**<.001**	.25, .33

Note: MLM = multilevel model; ADI = Area Deprivation Index; family SES = family socioeconomic status; adrenarche = coded 0 = early adrenarche, 1 = late adrenarche; BMI = body mass index. The outcome for all models is standardized, log-transformed Michigan Twins Project Eating Disorder Survey (MTP-ED) total score. Reference group for race/ethnicity is White. Effects significant at $p < .05$ are bolded.

Table S1.3. *Model fit comparisons for genotype x environment models across adrenarche status with BMI percentile not regressed out*

Model	-2lnL	χ^2 Δ (df)	*p*	AIC	BIC	SABIC
Neighborhood Disadvantage – *No Convergence of Full Model*						
Family SES						
Full model	8046.966	—	—	8092.965	8217.611	8144.543
Nested submodels						
No moderation	8162.084	115.118 (9)	<.001	8190.084	8265.955	8221.479
Constrain E SES mod	8047.242	.276 (1)		8091.242	8210.468	8140.577
Constrain E SES and SES x adrenarche mods	8054.980	8.014 (2)	.018	8096.979	8210.786	8144.072
Constrain E SES mod, A and C adrenarche mods	8047.534	.568 (3)	.904	8087.535	8195.922	8132.385
Constrain E SES mod, A adrenarche and SES x adrenarche mods, C adrenarche mod	8055.912	8.946 (4)	.062	8093.912	8196.881	8136.520
Constrain E SES mod, A adrenarche mod, C SES and SES x adrenarche mods	8057.026	10.060 (4)	.039	8095.026	8197.994	8137.634

Note: ADI = Area Deprivation Index percentile; SES = family socioeconomic status; BMI = body mass index; adrenarche = adrenarche status (0 = early adrenarche, 1 = late adrenarche); mod(s) = moderator(s); -2lnL = minus twice the log-likelihood; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion; full model = model with paths and all moderators; A = additive genetic variance; C = shared environmental variance; E = nonshared environmental variance. Although the model examining the ADI did not converge, cotwin correlations were consistent with results from the analogous GxE model that controlled for BMI in suggesting earlier activation of genetic influences for boys in early adrenarche living in disadvantaged neighborhoods. Specifically, the difference in the cotwin correlation between MZ and DZ twins was much greater for boys in early adrenarche living in more disadvantaged neighborhoods (high ADI: MZ = .982, DZ = .115) than for boys in early adrenarche living in less disadvantaged neighborhoods (low ADI: MZ = .421, DZ = .408).

Table S1.4. *Unstandardized path and moderator estimates for full and best-fitting genotype x environment models across adrenarche, without BMI percentile regressed out*

Neighborhood Disadvantage (ADI) – *No Convergence of Full Model*												
	Family SES											
Model	**a**	**c**	**e**	**β_{xP}**	**β_{yP}**	**β_{zP}**	**β_{xD}**	**β_{yD}**	**β_{zD}**	**β_{xPD}**	**β_{yPD}**	**β_{zPD}**
Full model	**.797 (.566, 1.029)**	.392 (-.052, .836)	**.436 (.296, .575)**	.010 (-.274, .294)	.110 (-.359, .580)	**.209 (.053, .365)**	-.378 (-.785, .029)	**-.836 (-1.397, -.276)**	-.060 (-.284, .164)	.297 (-.225, .819)	.619 (-.041, 1.279)	-.117 (-.374, .141)
Best-fitting	**.821 (.676, .966)**	**.473 (.224, .723)**	**-.406 (-.473, -.340)**	—	—	**-.237 (-.329, -.145)**	**-.444 (-.707, -.180)**	**-.949 (-1.290, -.607)**	—	**.348 (.096, .601)**	**.764 (.465, 1.064)**	**.176 (.060, .291)**

Note: Outcome is standardized, log-transformed MTP-ED total score without BMI percentile regressed out. ADI = Area Deprivation Index percentile (higher values indicate greater neighborhood disadvantage); family SES = family socioeconomic status (lower values indicate greater familial disadvantage); a = additive genetic influences at the lowest levels of the moderators; c = shared environmental influences at the lowest levels of the moderators; e = non-shared environmental influences at the lowest levels of the moderators; β_{xP}, β_{yP}, β_{zP} = coefficients for moderation of genetic/environmental variance by adrenarche; β_{xD}, β_{yD}, β_{zD} = coefficients for moderation of genetic/environmental variance by neighborhood disadvantage/family SES; β_{xPD}, β_{yPD}, β_{zPD} = coefficients representing changes in the moderating effects of disadvantage across adrenarche (i.e., the disadvantage x development interaction). 95% confidence intervals of parameter estimates are included in parentheses. Effects significant at $p < .05$ are bolded.

Figures

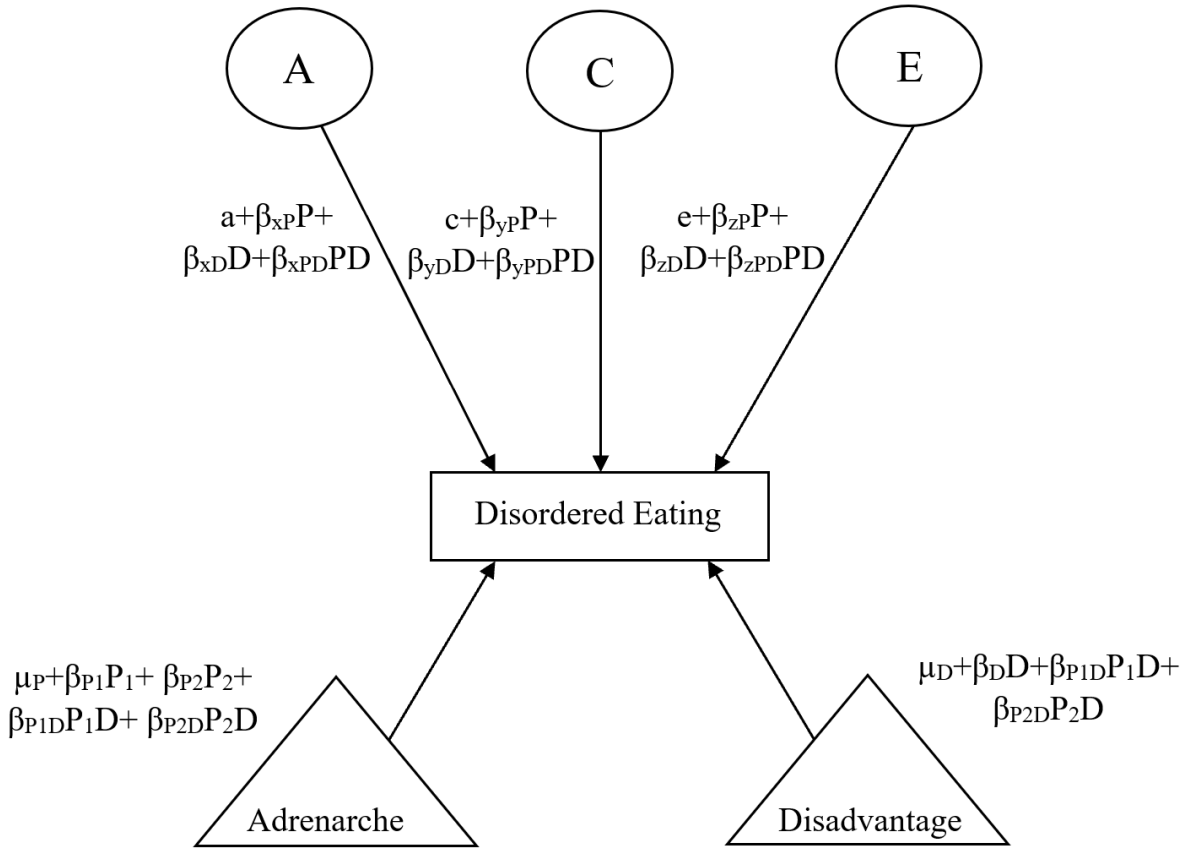


Figure S1.1. *Path diagram for the full twin moderation model*. Disadvantage = Area Deprivation Index percentile (neighborhood disadvantage), or a factor score comprised of mother's education level, father's education level, and combined parental income (family SES); Disordered Eating = standardized, log-transformed Michigan Twins Project Eating Disorder Survey (MTP-ED) total score with or without BMI percentile regressed out; Adrenarche = adrenarche status (0 = early adrenarche, 1 = late adrenarche); A = additive genetic influences; C = shared environmental influences; E = non-shared environmental influences; P_1 and P_2 = adrenarche status for twin 1 and twin 2; D = disadvantage for the twin pair; μ_P, μ_D, a, c, e = intercepts; β_{P1} = regression coefficient representing the phenotypic association between twin 1's adrenarche status and their own disordered eating; β_{P2} = regression coefficient representing the phenotypic association between

Figure S1.1 (cont'd)

twin 2's adrenarche status and twin 1's disordered eating; β_D = regression coefficient representing the phenotypic association between disadvantage and twin 1's disordered eating; β_{P1D} = regression coefficient representing moderation of the phenotypic association between disadvantage and twin 1's disordered eating by twin 1's adrenarche status; β_{P2D} = regression coefficient representing moderation of the phenotypic association between disadvantage and twin 1's disordered eating by twin 2's adrenarche status; β_{xP}, β_{yP}, β_{zP} = coefficients for moderation of genetic and environmental influences by adrenarche status; β_{xD}, β_{yD}, β_{zD} = coefficients for moderation of genetic and environmental influences by disadvantage; β_{xPD}, β_{yPD}, β_{zPD} = coefficients representing developmental differences in the moderating effects of disadvantage on genetic/environmental influences (i.e., the adrenarche x disadvantage interaction).

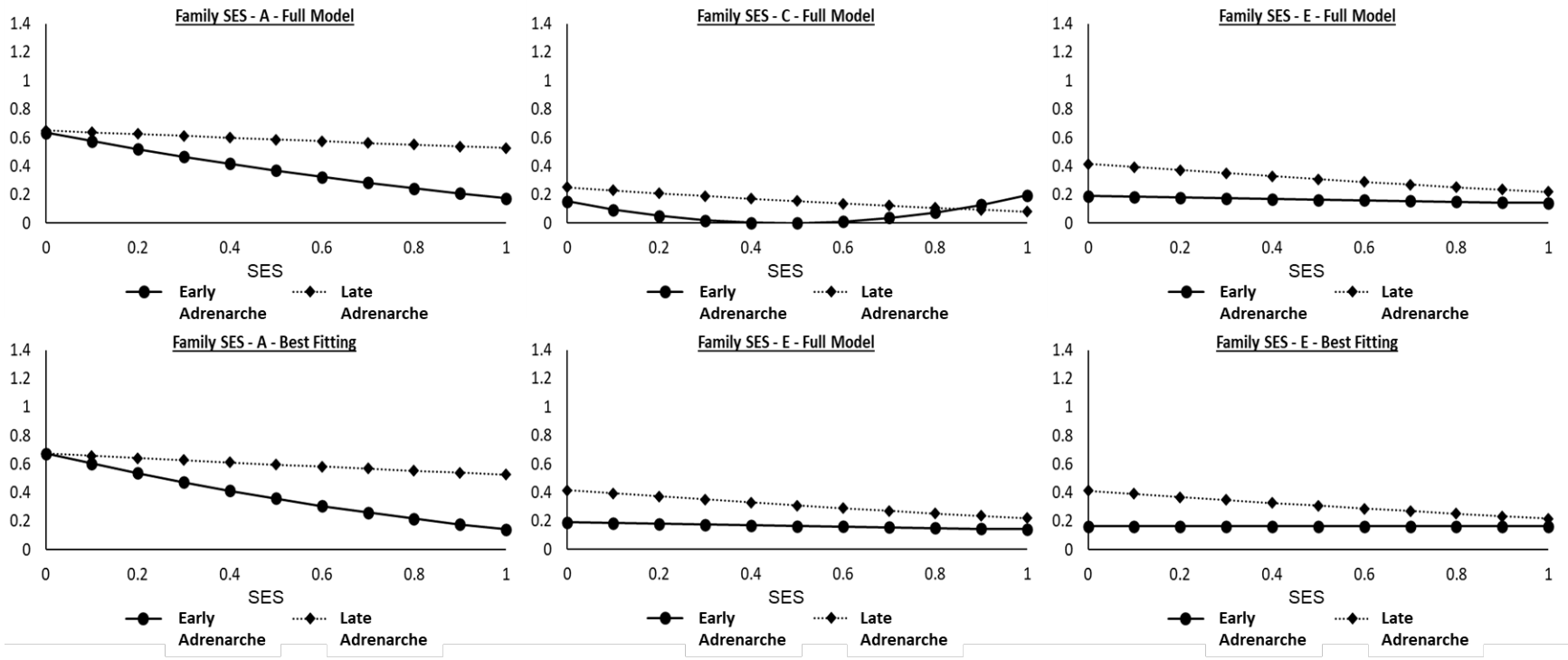


Figure S1.2. *Additive genetic (A), shared environmental (C), and non-shared environmental (E) influences on disordered eating across adrenarche status and family socioeconomic status (SES), without body mass index regressed out.*

APPENDIX B: SUPPLMENTAL MATERIAL FOR CHAPTER 2

Tables

Table S2.1. *Distribution of age, disordered eating, and neighborhood disadvantage by wave*

Full Sample						
Age in years	***N* (%) at time 1**	***N* (%) at time 2**	***N* (%) at time 3**	**Total *N***	**MEBS total score Mean (SD)**	**ADI Mean (SD)**
6	600 (29.1%)	0 (0.0%)	0 (0.0%)	600	5.59 (4.22)	60.62 (22.08)
7	408 (19.8%)	6 (0.8%)	0 (0.0%)	414	5.63 (4.14)	55.42 (22.11)
8	344 (16.7%)	4 (0.5%)	0 (0.0%)	348	5.54 (4.05)	59.13 (21.80)
9	338 (16.4%)	12 (1.6%)	0 (0.0%)	350	5.77 (4.29)	54.68 (22.54)
10	312 (15.1%)	38 (4.9%)	8 (2.1%)	358	5.02 (4.02)	55.63 (24.38)
11	58 (2.8%)	36 (4.7%)	30 (7.9%)	124	5.45 (4.50)	56.93 (25.13)
12	0 (0.0%)	58 (7.6%)	26 (6.8%)	84	7.12 (5.27)	59.48 (19.70)
13	0 (0.0%)	62 (8.1%)	32 (8.4%)	94	7.31 (4.24)	62.40 (23.45)
14	0 (0.0%)	154 (20.1%)	30 (7.9%)	184	8.32 (4.77)	60.49 (21.23)
15	0 (0.0%)	162 (21.1%)	74 (19.5%)	236	7.26 (4.27)	60.29 (20.84)
16	0 (0.0%)	110 (14.3%)	68 (17.9%)	178	7.66 (4.78)	62.91 (18.73)
17	0 (0.0%)	94 (12.2%)	42 (11.1%)	136	7.97 (5.30)	60.92 (19.25)
18	0 (0.0%)	24 (3.1%)	42 (11.1%)	66	8.22 (4.44)	51.43 (21.22)
19	0 (0.0%)	4 (0.5%)	26 (6.8%)	30	7.09 (5.61)	65.33 (27.58)
20	0 (0.0%)	2 (0.3%)	2 (0.5%)	4	11.17 (3.06)	87.00 (0.00)
21	0 (0.0%)	2 (0.3%)	0 (0.0%)	2	—	25.00 (0.00)
Mean (SD) age in years	8.02 (1.49)	14.77 (2.27)	15.63 (2.33)	—	—	—
Female Participants						
Age in years	***N* (%) at time 1**	***N* (%) at time 2**	***N* (%) at time 3**	**Total *N***	**MEBS total score Mean (SD)**	**ADI Mean (SD)**
6	286 (28.5%)	0 (0.0%)	0 (0.0%)	286	5.81 (4.45)	61.62 (22.17)

Table S2.1 (cont'd)

7	205 (20.4%)	0 (0.0%)	0 (0.0%)	205	5.38 (4.07)	55.32 (21.83)
8	153 (15.3%)	0 (0.0%)	0 (0.0%)	153	5.47 (4.06)	57.56 (21.50)
9	172 (17.1%)	0 (0.0%)	0 (0.0%)	172	5.44 (4.51)	50.08 (22.09)
10	150 (15.0%)	12 (3.3%)	0 (0.0%)	162	4.20 (3.64)	55.50 (25.41)
11	37 (3.7%)	15 (4.1%)	11 (6.2%)	63	5.23 (5.08)	53.29 (25.79)
12	0 (0.0%)	14 (3.9%)	8 (4.5%)	22	7.25 (3.84)	60.25 (21.22)
13	0 (0.0%)	34 (9.4%)	3 (1.7%)	37	7.88 (4.41)	61.94 (22.91)
14	0 (0.0%)	81 (22.4%)	13 (7.3%)	94	9.32 (5.00)	58.60 (21.92)
15	0 (0.0%)	81 (22.4%)	46 (26.0%)	127	7.74 (4.41)	60.27 (21.09)
16	0 (0.0%)	60 (16.6%)	39 (22.0%)	99	8.70 (4.90)	61.21 (19.19)
17	0 (0.0%)	46 (12.7%)	15 (8.5%)	61	10.04 (5.01)	61.87 (18.41)
18	0 (0.0%)	16 (4.4%)	24 (13.6%)	40	9.46 (4.60)	46.24 (20.11)
19	0 (0.0%)	1 (0.3%)	16 (9.0%)	17	6.67 (5.94)	71.79 (25.34)
20	0 (0.0%)	0 (0.0%)	2 (1.1%)	2	11.17 (3.06)	—
21	0 (0.0%)	2 (0.6%)	0 (0.0%)	2	—	25.00 (0.00)
Mean (SD) age in years	8.05 (1.51)	15.19 (1.94)	16.17 (2.09)	—	—	—
			Male Participants			
Age in years	***N* (%) at time 1**	***N* (%) at time 2**	***N* (%) at time 3**	**Total *N***	**MEBS total score Mean (SD)**	**ADI Mean (SD)**
6	314 (29.7%)	0 (0.0%)	0 (0.0%)	314	5.39 (4.00)	59.68 (21.99)
7	203 (19.2%)	6 (1.5%)	0 (0.0%)	209	5.88 (4.21)	55.53 (22.45)
8	191 (18.1%)	4 (1.0%)	0 (0.0%)	195	5.60 (4.05)	60.38 (22.02)
9	166 (15.7%)	12 (3.0%)	0 (0.0%)	178	6.10 (4.05)	59.07 (22.14)
10	162 (15.3%)	26 (6.4%)	8 (3.9%)	196	5.72 (4.20)	55.74 (23.55)
11	21 (2.0%)	21 (5.2%)	19 (9.4%)	61	5.65 (3.90)	60.83 (24.02)
12	0 (0.0%)	44 (10.8%)	18 (8.9%)	62	7.08 (5.62)	59.22 (19.35)
13	0 (0.0%)	28 (6.9%)	29 (14.3%)	57	6.89 (4.12)	62.70 (24.02)
14	0 (0.0%)	73 (18.0%)	17 (8.4%)	90	7.17 (4.24)	62.52 (20.40)

Table S2.1 (cont'd)

15	0 (0.0%)	81 (20.0%)	28 (13.8%)	109	6.77 (4.08)	60.31 (20.68)
16	0 (0.0%)	50 (12.3%)	29 (14.3%)	79	6.17 (4.21)	64.90 (18.12)
17	0 (0.0%)	48 (11.8%)	27 (13.3%)	75	6.62 (5.07)	60.18 (19.97)
18	0 (0.0%)	8 (2.0%)	18 (8.9%)	26	6.63 (3.73)	59.78 (20.67)
19	0 (0.0%)	3 (0.7%)	10 (4.9%)	13	7.64 (5.41)	56.30 (29.36)
20	0 (0.0%)	2 (0.5%)	0 (0.0%)	2	—	87.00 (0.00)
21	0 (0.0%)	0 (0.0%)	0 (0.0%)	0	—	—
Mean (SD) age in years	7.98 (1.47)	14.39 (2.47)	15.16 (2.42)	—	—	—

Note: MEBS = Minnesota Eating Behavior Survey; ADI = Area Deprivation Index; SD = standard deviation. Dashes indicate that a value is not applicable.

Table S2.2. *Items assessed by the Area Deprivation Index (Singh, 2003)*

1. Percent of population aged 25 or older with less than 9 years of education
2. Percent of population aged 25 or older with at least a high school diploma
3. Percent of employed persons aged 16 years or older in white-collar occupations
4. Median family income (in dollars)
5. Income disparity ($\log_{10}$(100*[number of households with less than $10,000 dollars in income/number of households with an income of $50,000 dollars or more])
6. Median home value (in dollars)
7. Median gross rent (in dollars)
8. Median monthly mortgage (in dollars)
9. Home ownership rate (percent owner-occupied housing units)
10. Unemployment rate (percent of civilian labor force aged ≥16 unemployed)
11. Percent of families below the poverty level
12. Percent of population below 150% of the poverty threshold
13. Percent single-parent households with children less than 18 years old
14. Percent of households without a motor vehicle
15. Percent of households without telephone service
16. Percent occupied housing units without complete plumbing (log transformed)
17. Crowding (percent of households with more than 1 person per room)

Table S2.3. *Model fit comparisons constraining parameters across sex for the neighborhood disadvantage and disordered eating multilevel growth curve model, with disordered eating not log transformed*

Model	AIC	BIC	SABIC	-2lnL	Scaling Correction Factor	$\chi^2\Delta$ (df)	*p*
Full model	20094.26	20786.63	20424.40	19866.26	6.40	—	—
Constraining ADI Parameters							
Constrain all ADI parameters	20091.07	20753.07	20406.73	19873.07	6.55	2.04 (5)	.844
Constraining DE Parameters							
Constrain all DE parameters	20098.85	20760.85	20414.51	19880.85	6.65	14.21 (5)	.014
Constrain DE intercept factor mean	20092.97	20779.27	20420.22	19866.97	6.45	0.73 (1)	.393
Constrain DE intercept factor variance	20096.30	20782.60	20423.55	19870.30	6.45	2.84 (1)	.092
Constrain DE slope factor mean	20097.31	20783.60	20424.55	19871.31	6.46	8.01 (1)	.005
Constrain DE slope factor variance	20094.52	20780.82	20421.77	19868.52	6.45	1.78 (1)	.182
Constrain DE intercept-slope covariance	20093.67	20779.97	20420.92	19867.67	6.45	1.07 (1)	.301
Constraining ADI → DE Association Parameters							
Constrain all ADI → DE parameters	20091.67	20765.82	20413.13	19869.67	6.55	3.88 (3)	.275
Constrain all parameters except DE slope factor mean	20089.98	20709.46	20385.37	19885.98	6.92	9.88 (12)	.626
Constrain all parameters except DE slope factor mean, DE intercept factor variance	**20086.17**	**20711.73**	**20384.45**	**19880.17**	**6.87**	**6.70 (11)**	**.823**

Note: ADI = Area Deprivation Index; DE = disordered eating; -2lnL = minus twice the log-likelihood; $\chi^2\Delta$ = chi square change; df = degrees of freedom; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted

Table S2.3 (cont'd)

Bayesian Information Criterion. Dashes indicate parameters are not applicable. The best-fitting model is bolded.

Table S2.4. *Estimates from the best-fitting neighborhood disadvantage and disordered eating multilevel growth curve model, with disordered eating not log transformed*

Parameter	Unstandardized Estimate	SE	*p*	95% CI
Best-Fitting Model in Girls				
Neighborhood disadvantage (ADI)				
Intercept factor mean	5.42	.07	**<.001**	5.29, 5.55
Intercept factor residual variance	4.44	.18	**<.001**	4.10, 4.79
Slope factor mean	-.02	.01	**.017**	-.03, -.003
Slope factor residual variance	.02	.004	**<.001**	.02, .03
Intercept-slope covariance	-.08	.02	**<.001**	-.12, -.04
Disordered eating (DE)				
Intercept factor mean	3.99	.49	**<.001**	3.03, 4.95
Intercept factor residual variance	10.69	1.83	**<.001**	7.10, 14.28
Slope factor mean	.20	.10	**.042**	.01, .40
Slope factor residual variance	.20	.04	**<.001**	.13, .27
Intercept-slope covariance	-.79	.20	**<.001**	-1.18, -.41
Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.14	.06	**.033**	.01, .26
ADI intercept factor → DE slope factor	.01	.01	.250	-.01, .04
ADI slope factor → DE slope factor	.24	.15	.098	-.04, .53
Best-Fitting Model in Boys				
Neighborhood disadvantage (ADI)				
Intercept factor mean	5.42	.07	**<.001**	5.29, 5.55
Intercept factor residual variance	4.44	.18	**<.001**	4.10, 4.79
Slope factor mean	-.02	.01	**.017**	-.03, -.003
Slope factor residual variance	.02	.004	**<.001**	.02, .03
Intercept-slope covariance	-.08	.02	**<.001**	-.12, -.04
Disordered eating (DE)				
Intercept factor mean	3.99	.49	**<.001**	3.03, 4.95
Intercept factor residual variance	7.67	1.74	**<.001**	4.25, 11.09
Slope factor mean	-.01	.13	.917	-.27, .25
Slope factor residual variance	.20	.04	**<.001**	.13, .27
Intercept-slope covariance	-.79	.20	**<.001**	-1.18, -.41

Table S2.4 (cont'd)

Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.14	.06	**.033**	.01, .26
ADI intercept factor → DE slope factor	.01	.01	.250	-.01, .04
ADI slope factor → DE slope factor	.24	.15	.098	-.04, .53

Note: ADI = Area Deprivation Index.

Table S2.5. *Model fit comparisons constraining parameters across sex for the neighborhood disadvantage and disordered eating multilevel growth curve models, controlling for time 1 family income*

Model	AIC	BIC	SABIC	-2lnL	Scaling Correction Factor	$\chi^2\Delta$ (df)	*p*
Full model	17200.88	18039.01	17600.53	16924.88	5.56	—	—
<u>Constraining ADI Parameters</u>							
Constrain all ADI parameters	17197.95	18005.71	17583.11	16931.95	5.64	2.09 (5)	.837
<u>Constraining INC Parameters</u>							
Constrain all T1 INC parameters (i.e., mean and variance)	17197.39	18023.37	17591.24	16925.39	5.62	0.26 (2)	.880
<u>Constraining DE Parameters</u>							
Constrain all DE parameters	17202.74	18010.51	17587.91	16936.74	5.73	11.14 (5)	.049
Constrain DE intercept factor mean	17199.03	18031.09	17595.78	16925.03	5.60	0.15 (1)	.702
Constrain DE intercept factor variance	17205.37	18037.43	17602.12	16931.37	5.60	6.22 (1)	.013
Constrain DE slope factor mean	17200.30	18032.35	17597.04	16926.29	5.60	1.34 (1)	.248
Constrain DE slope factor variance	17199.67	18031.73	17596.42	16925.67	5.59	0.64 (1)	.425
Constrain DE intercept-slope covariance	17202.04	18034.09	17598.78	16928.04	5.60	3.03 (1)	.082
<u>Constraining ADI → DE Association Parameters</u>							
Constrain all ADI → DE parameters	17197.54	18017.45	17588.50	16927.54	5.66	2.63 (3)	.453
<u>Constraining INC → DE Association Parameters</u>							
Constrain all T1 INC → DE parameters	17197.11	18023.09	17590.96	16925.11	5.63	0.22 (2)	.894

Table S2.5 (cont'd)

Constrain all parameters except DE intercept factor variance	17191.06	17932.02	17544.37	16947.06	6.04	11.75 (16)	.761
Constrain all parameters except DE intercept factor variance and DE intercept-slope covariance	**17186.07**	**17933.10**	**17542.28**	**16940.07**	**6.00**	**7.79 (15)**	**.932**

Note: T1 = time 1; ADI = Area Deprivation Index; INC = family income; DE = disordered eating; -2lnL = minus twice the log-likelihood; $\boldsymbol{\chi^2\Delta}$ = chi square change; df = degrees of freedom; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion. Dashes indicate parameters are not applicable. The best-fitting model is bolded.

Table S2.6. *Estimates from the best-fitting neighborhood disadvantage and disordered eating multilevel growth curve model, controlling for time 1 family income*

Parameter	Unstandardized Estimate	SE	*p*	95% CI
Best-Fitting Model in Girls				
Neighborhood disadvantage (ADI)				
Intercept factor mean	5.42	.07	**<.001**	5.29, 5.55
Intercept factor residual variance	4.45	.18	**<.001**	4.10, 4.80
Slope factor mean	-.02	.01	**.014**	-.03, -.003
Slope factor residual variance	.02	.004	**<.001**	.02, .03
Intercept-slope covariance	-.08	.02	**<.001**	-.12, -.04
Family income (INC)				
Time 1 mean	5.78	.05	**<.001**	5.68, 5.88
Time 1 variance	3.71	.19	**<.001**	3.33, 4.08
Disordered eating (DE)				
Intercept factor mean	1.49	.13	**<.001**	1.23, 1.74
Intercept factor residual variance	.47	.06	**<.001**	.35, .60
Slope factor mean	.05	.02	**.031**	.004, .09
Slope factor residual variance	.01	.001	**<.001**	.003, .01
Intercept-slope covariance	-.04	.01	**<.001**	-.05, -.03
Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.03	.01	**.027**	.003, .05
ADI intercept factor → DE slope factor	.00	.002	.806	-.004, .003
ADI slope factor → DE slope factor	.03	.02	.128	-.01, .07
Associations between T1 INC and DE parameters				
T1 INC → DE intercept factor	-.02	.01	.10	-.05, .004
T1 INC → DE slope factor	.00	.002	.84	-.01, .004
Best-Fitting Model in Boys				
Neighborhood disadvantage (ADI)				
Intercept factor mean	5.42	.07	**<.001**	5.29, 5.55
Intercept factor residual variance	4.45	.18	**<.001**	4.10, 4.80
Slope factor mean	-.02	.01	**.014**	-.03, -.003
Slope factor residual variance	.02	.004	**<.001**	.02, .03
Intercept-slope covariance	-.08	.02	**<.001**	-.12, -.04

Table S2.6 (cont'd)

Family income (INC)				
Time 1 mean	5.78	.05	**<.001**	5.68, 5.88
Time 1 variance	3.71	.19	**<.001**	3.33, 4.08
Disordered eating (DE)				
Intercept factor mean	1.49	.13	**<.001**	1.23, 1.74
Intercept factor residual variance	.32	.05	**<.001**	.23, .41
Slope factor mean	.05	.02	**.031**	.004, .09
Slope factor residual variance	.01	.001	**<.001**	.003, .01
Intercept-slope covariance	-.03	.01	**<.001**	-.04, -.02
Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.03	.01	**.027**	.003, .05
ADI intercept factor → DE slope factor	.00	.002	.806	-.004, .003
ADI slope factor → DE slope factor	.03	.02	.128	-.01, .07
Associations between T1 INC and DE parameters				
T1 INC → DE intercept factor	-.02	.01	.102	-.05, .004
T1 INC → DE slope factor	.00	.002	.835	-.01, .004

Note: T1 = time 1; ADI = Area Deprivation Index.

Table S2.7. *Model fit comparisons constraining parameters across sex for the neighborhood disadvantage and disordered eating multilevel growth curve models, controlling for time 1 BMI percentile*

Model	AIC	BIC	SABIC	-2lnL	Scaling Correction Factor	χ^2 Δ (df)	*p*
Full model	19216.84	20054.97	19616.48	18940.84	5.46	—	—
<u>Constraining ADI Parameters</u>							
Constrain all ADI parameters	19213.60	20021.36	19598.77	18947.60	5.54	2.00 (5)	.850
<u>Constraining BMI Parameters</u>							
Constrain all T1 BMI parameters (i.e., mean and variance)	19213.50	20039.48	19607.35	18941.50	5.53	0.76 (1)	.385
<u>Constraining DE Parameters</u>							
Constrain all DE parameters	19223.40	20031.16	19608.56	18957.40	5.63	17.53 (5)	.004
Constrain DE intercept factor mean	19215.83	20047.88	19612.57	18941.82	5.49	1.02 (1)	.313
Constrain DE intercept factor variance	19220.68	20052.74	19617.43	18946.68	5.49	6.62 (1)	.010
Constrain DE slope factor mean	19220.16	20052.22	19616.91	18946.16	5.49	6.88 (1)	.009
Constrain DE slope factor variance	19215.28	20047.34	19612.03	18941.28	5.49	0.35 (1)	.552
Constrain DE intercept-slope covariance	19217.40	20049.46	19614.15	18943.40	5.49	2.55 (1)	.111
<u>Constraining ADI → DE Association Parameters</u>							
Constrain all ADI → DE parameters	19213.91	20033.82	19604.87	18943.91	5.56	2.88 (3)	.411
<u>Constraining BMI → DE Association Parameters</u>							
Constrain all T1 BMI → DE parameters	19213.11	20039.09	19606.96	18941.11	5.52	0.26 (2)	.879

Table S2.7 (cont'd)

Constrain all parameters except DE intercept factor variance and DE slope factor mean	19205.78	19952.81	19561.99	18959.78	5.90	10.54 (15)	.785
Constrain all parameters except DE intercept factor variance, DE slope factor mean, and DE intercept-slope covariance	**19200.04**	**19953.14**	**19559.14**	**18952.04**	**5.87**	**6.04 (14)**	**.966**

Note: T1 = time 1; ADI = Area Deprivation Index; BMI = body mass index percentile; DE = disordered eating; -2lnL = minus twice the log-likelihood; $\chi^2\Delta$ = chi square change; df = degrees of freedom; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion. Dashes indicate parameters are not applicable. The best-fitting model is bolded.

Table S2.8. *Estimates from the best-fitting neighborhood disadvantage and disordered eating multilevel growth curve model, controlling for time 1 BMI percentile*

Parameter	Unstandardized Estimate	SE	*p*	95% CI
Best-Fitting Model in Girls				
Neighborhood disadvantage (ADI)				
Intercept factor mean	5.42	.07	**<.001**	5.29, 5.55
Intercept factor residual variance	4.44	.18	**<.001**	4.09, 4.79
Slope factor mean	-.02	.01	**.015**	-.03, -.003
Slope factor residual variance	.02	.004	**<.001**	.02, .03
Intercept-slope covariance	-.08	.02	**<.001**	-.12, -.04
Body mass index (BMI)				
Time 1 mean	5.92	.07	**<.001**	5.79, 6.06
Time 1 variance	8.46	.20	**<.001**	8.07, 8.84
Disordered eating (DE)				
Intercept factor mean	1.22	.09	**<.001**	1.04, 1.39
Intercept factor residual variance	.46	.06	**<.001**	.34, .58
Slope factor mean	.03	.02	**.042**	.001, .06
Slope factor residual variance	.004	.001	**<.001**	.003, .01
Intercept-slope covariance	-.04	.01	**<.001**	-.05, -.03
Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.03	.01	**.011**	.01, .05
ADI intercept factor → DE slope factor	.00	.002	.828	-.004, .003
ADI slope factor → DE slope factor	.03	.02	.164	-.01, .07
Associations between T1 BMI and DE parameters				
T1 BMI → DE intercept factor	.03	.01	**<.001**	.02, .05
T1 BMI → DE slope factor	.004	.001	**<.001**	.002, .01
Best-Fitting Model in Boys				
Neighborhood disadvantage (ADI)				
Intercept factor mean	5.42	.07	**<.001**	5.29, 5.55
Intercept factor residual variance	4.44	.18	**<.001**	4.09, 4.79
Slope factor mean	-.02	.01	**.015**	-.03, -.003
Slope factor residual variance	.02	.004	**<.001**	.02, .03
Intercept-slope covariance	-.08	.02	**<.001**	-.12, -.04

Table S2.8 (cont'd)

Body mass index (BMI)				
Time 1 mean	5.92	.07	**<.001**	5.79, 6.06
Time 1 variance	8.46	.20	**<.001**	8.07, 8.84
Disordered eating (DE)				
Intercept factor mean	1.22	.09	**<.001**	1.04, 1.39
Intercept factor residual variance	.31	.04	**<.001**	.22, .39
Slope factor mean	-.004	.02	.828	-.05, .04
Slope factor residual variance	.004	.001	**<.001**	.003, .01
Intercept-slope covariance	-.03	.01	**<.001**	-.04, -.02
Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.03	.01	**.011**	.01, .05
ADI intercept factor → DE slope factor	.00	.002	.828	-.004, .003
ADI slope factor → DE slope factor	.03	.02	.164	-.01, .07
Associations between T1 BMI and DE parameters				
T1 BMI → DE intercept factor	.03	.01	**<.001**	.02, .05
T1 BMI → DE slope factor	.004	.001	**<.001**	.002, .01

Note: T1 = time 1; ADI = Area Deprivation Index. BMI percentiles were divided by 10 to increase interpretability of model parameters (i.e., an increase of 1 on BMI in this model represents an increase of 10 BMI percentiles).

Table S2.9. *Model fit comparisons constraining parameters across sex for the neighborhood disadvantage and disordered eating multilevel growth curve model, including participants in the under-resourced arm only*

Model	AIC	BIC	SABIC	-2lnL	Scaling Correction Factor	$\chi^2\Delta$ (df)	*p*
Full model	11305.19	11697.09	11468.36	11161.19	2.01	—	—
Constraining ADI Parameters							
Constrain all ADI parameters	11300.55	11665.23	11452.38	11166.55	1.97	2.11 (5)	.834
Constraining DE Parameters							
Constrain all DE parameters	11297.51	11662.19	11449.34	11163.51	2.07	2.00 (5)	.849
Constraining ADI → DE Association Parameters							
Constrain all ADI → DE parameters	11300.62	11676.19	11456.98	11162.62	2.06	1.49 (3)	.685
Constrain all parameters	**11288.59**	**11609.73**	**11422.29**	**11170.58**	**2.08**	**5.56 (13)**	**.961**

Note: ADI = Area Deprivation Index; DE = disordered eating; -2lnL = minus twice the log-likelihood; $\chi^2\Delta$ = chi square change; df = degrees of freedom; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion. Note that in this model, racial identity was collapsed into White, Black, or other racial identity due to the small number of participants with racial/ethnic identities other than White or Black. Dashes indicate parameters are not applicable. The best-fitting model is bolded.

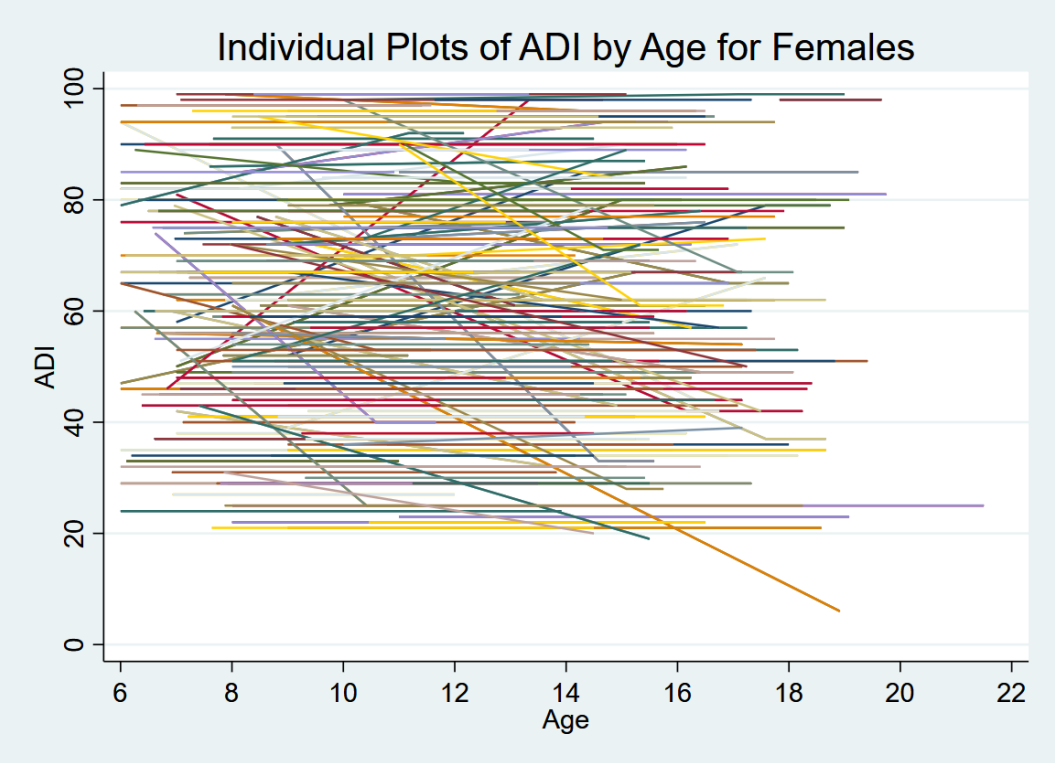
Table S2.10. *Estimates from the best-fitting neighborhood disadvantage and disordered eating multilevel growth curve model, including participants in the under-resourced arm only*

Parameter	Unstandardized Estimate	SE	*p*	95% CI
Best-Fitting Model in Girls				
Neighborhood disadvantage (ADI)				
Intercept factor mean	6.24	.08	**<.001**	6.08, 6.39
Intercept factor residual variance	3.31	.22	**<.001**	2.89, 3.73
Slope factor mean	-.03	.01	**<.001**	-.05, -.02
Slope factor residual variance	.02	.01	**<.001**	.01, .03
Intercept-slope covariance	-.06	.02	**.010**	-.11, -.02
Disordered eating (DE)				
Intercept factor mean	1.37	.14	**<.001**	1.09, 1.65
Intercept factor residual variance	.23	.07	**<.001**	.11, .36
Slope factor mean	.06	.02	**.005**	.02, .11
Slope factor residual variance	.003	.001	**.023**	<.001, .06
Intercept-slope covariance	-.02	.01	**.010**	-.04, -.01
Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.04	.02	**.028**	.004, .08
ADI intercept factor → DE slope factor	-.01	.003	.068	-.01, <.001
ADI slope factor → DE slope factor	.01	.03	.727	-.04, .06
Best-Fitting Model in Boys				
Neighborhood disadvantage (ADI)				
Intercept factor mean	6.24	.08	**<.001**	6.08, 6.39
Intercept factor residual variance	3.31	.22	**<.001**	2.89, 3.73
Slope factor mean	-.03	.01	**<.001**	-.05, -.02
Slope factor residual variance	.02	.01	**<.001**	.01, .03
Intercept-slope covariance	-.06	.02	**.010**	-.11, -.02
Disordered eating (DE)				
Intercept factor mean	1.37	.14	**<.001**	1.09, 1.65
Intercept factor residual variance	.23	.07	**<.001**	.11, .36
Slope factor mean	.06	.02	**.005**	.02, .11
Slope factor residual variance	.003	.001	**.023**	<.001, .06
Intercept-slope covariance	-.02	.01	**.010**	-.04, -.01

Table S2.10 (cont'd)

Associations between ADI and DE parameters				
ADI intercept factor → DE intercept factor	.04	.02	**.028**	.004, .08
ADI intercept factor → DE slope factor	-.01	.003	.068	-.01, <.001
ADI slope factor → DE slope factor	.01	.03	.727	-.04, .06

Note: ADI = Area Deprivation Index. Note that in this model, racial identity was collapsed into White, Black, or other racial identity due to the small number of participants with racial/ethnic identities other than White or Black.



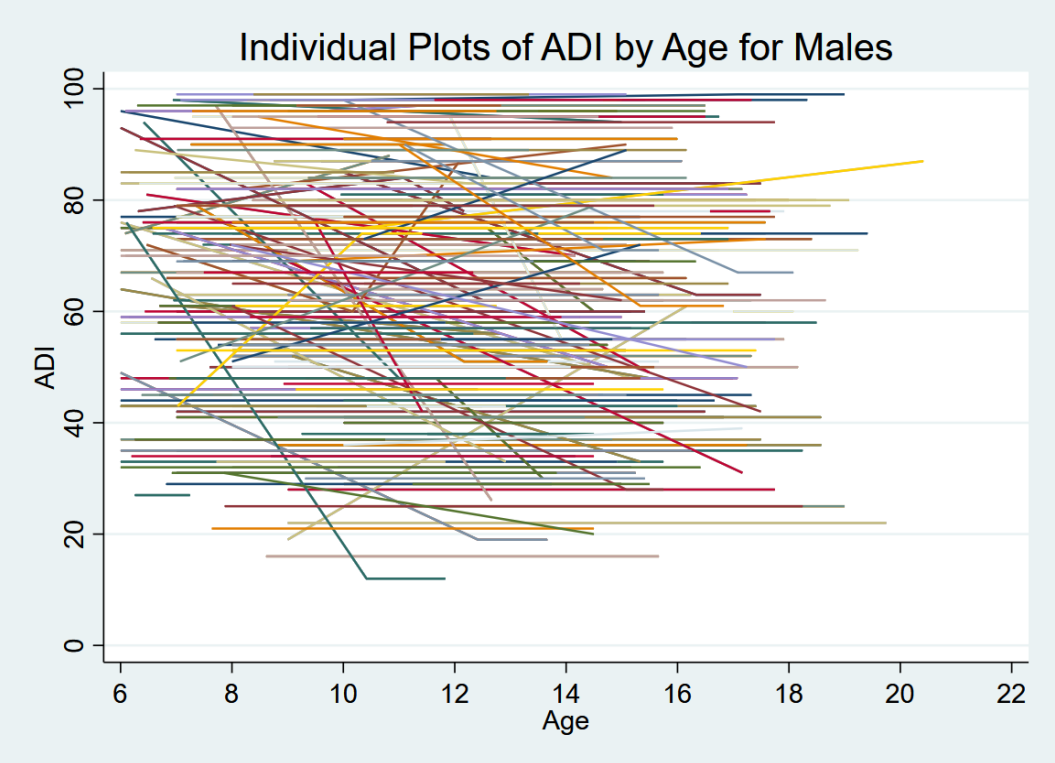
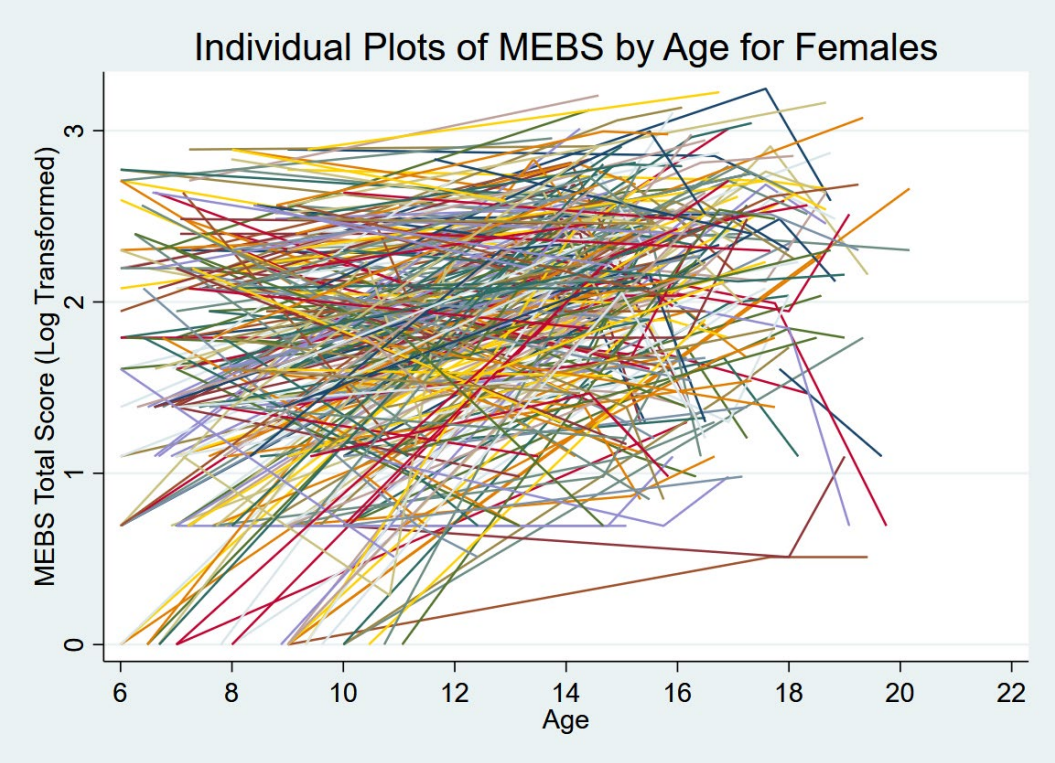


Figure S2.1. *Levels of neighborhood disadvantage by age for individual participants with at least two timepoints.* ADI = Area Deprivation Index.



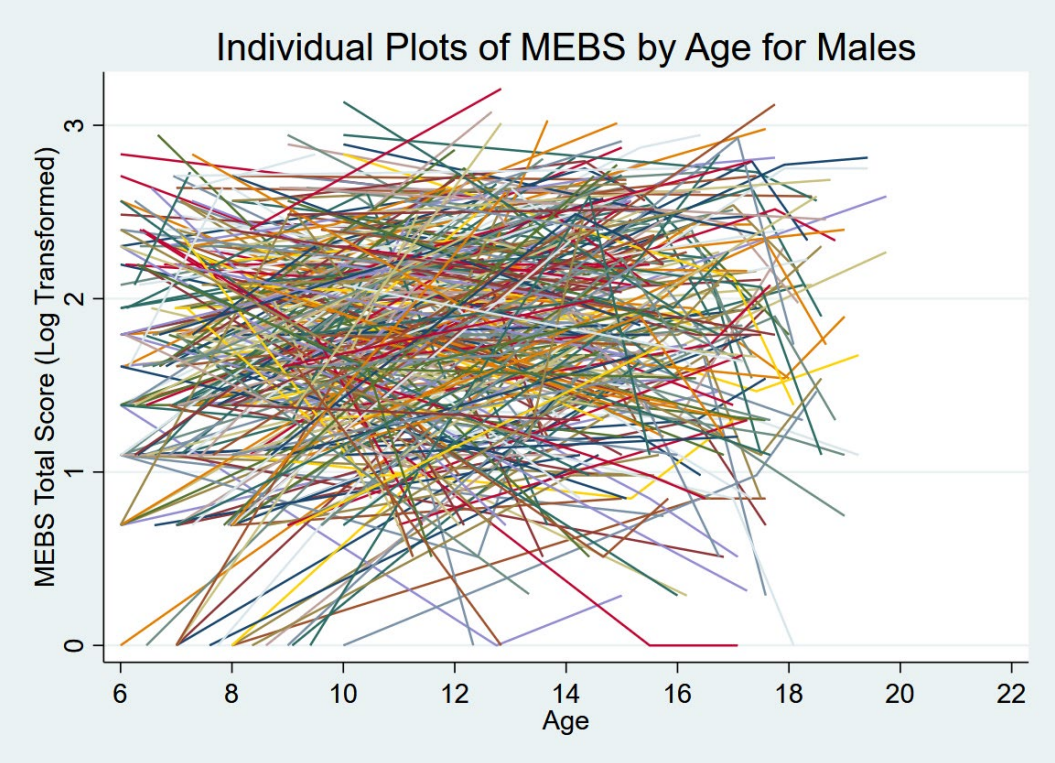


Figure S2.2. *Levels of disordered eating by age for individual participants with at least two timepoints.* MEBS = Minnesota Eating Behavior Survey.

APPENDIX C: SUPPLMENTAL MATERIAL FOR CHAPTER 3

Tables

Table S3.1. *Model fit comparisons constraining parameters across sex for cross-sectional and longitudinal effects, using impact rather than frequency of community violence exposure*

Model	AIC	BIC	SABIC	χ^2 (df)	Scaling Correction Factor	$\chi^2\Delta$ (df)	*p*
Full model	58002.58	58948.66	58414.91	11.33 (12)	1.0470	—	—
Constrain all parameters	57998.74	58860.35	58374.26	34.37 (27)	1.1064	22.68 (15)	.091
T1 associations							
Constrain T1 ECV → T1 DE	58000.71	58941.16	58410.58	11.08 (13)	1.0825	0.09 (1)	.764
Constrain T1 NR → T1 DE	58001.86	58942.31	58411.74	12.53 (13)	1.0491	1.20 (1)	.273
Constrain T1 age → T1 DE	58008.82	58949.27	58418.69	19.00 (13)	1.0582	6.91 (1)	.009
Constrain Black racial identity → T1 DE	58001.66	58942.11	58411.53	11.97 (13)	1.0818	0.72 (1)	.396
Constrain other POC identity → T1 DE	58005.93	58946.38	58415.81	16.40 (13)	1.0497	4.95 (1)	.026
Constrain T1 INC → T1 DE	58000.58	58941.02	58410.45	11.09 (13)	1.0702	0.00 (1)	1.000
FU associations							
Constrain FU ECV → FU DE	58002.54	58942.99	58412.41	12.88 (13)	1.0735	1.41 (1)	.235
Constrain FU NR → FU DE	58001.41	58941.86	58411.29	12.38 (13)	1.0260	1.08 (1)	.299
Constrain FU age → FU DE	58002.10	58942.55	58411.98	12.69 (13)	1.0551	1.32 (1)	.235
Constrain Black racial identity → FU DE	58001.26	58941.71	58411.13	12.25 (13)	1.0242	0.91 (1)	.340
Constrain other POC identity → FU DE	58000.89	58941.34	58410.77	11.54 (13)	1.0553	0.27 (1)	.603
Constrain FU INC → FU DE	58001.10	58941.55	58410.98	12.07 (13)	1.0271	0.67 (1)	.413
T1 → FU associations							
Constraint T1 DE → FU DE	58003.00	58943.45	58412.87	13.47 (13)	1.0608	1.98 (1)	.159

Table S3.1 (cont'd)

Constrain T1 ECV → FU DE	58001.09	58941.53	58410.96	11.71 (13)	1.0572	0.43 (1)	.512
Constrain T1 NR → FU DE	58000.92	58941.37	58410.79	11.85 (13)	1.0306	0.41 (1)	.522
Constrain all parameters except T1 age → T1 DE, other POC identity → T1 DE, T1 DE	**57989.34**	**58862.21**	**58369.77**	**22.35 (25)**	**1.1021**	**11.07 (13)**	**.605**

Note. T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; ECV = reported impact of exposure to community violence; NR = neighborhood resources; POC = person of color; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion; χ^2 **Δ** = change in chi-square; df = degrees of freedom. The best-fitting model is bolded.

Table S3.2. *Estimates for all parameters from the best-fitting model for cross-sectional and longitudinal effects of neighborhood factors on disordered eating, using impact rather than frequency of community violence exposure*

Parameter	Standardized Estimate	Unstandardized Estimate	SE	95% CI
Best Fitting Model in Girls				
Direct effects of T1 variables on T1 outcomes				
T1 ECV → T1 DE	**.17**	**.23**	**.03**	**.16, .30**
T1 NR → T1 DE	.01	.02	.09	-.15, .20
T1 age → T1 DE	**-.11**	**-.30**	**.10**	**-.50, -.10**
Black racial identity → T1 DE	**.10**	**1.49**	**.45**	**.65, 2.44**
Other POC racial identity → T1 DE	**.09**	**1.35**	**.55**	**.25, 2.43**
T1 family income → T1 DE	-.05	-.10	.06	-.22, .02
Direct effects of FU variables on FU outcomes				
FU ECV → FU DE	**.13**	**.29**	**.09**	**.14, .47**
FU NR → FU DE	.04	.11	.13	-.16, .36
FU age → FU DE	.07	.16	.11	-.06, .37
Black racial identity → FU DE	-.05	-.83	.67	-2.11, .45
Other POC racial identity → FU DE	-.07	-1.19	.64	-2.41, .12
FU family income → FU DE	-.03	-.11	.16	-.44, .18
Direct effects of T1 variables on FU outcomes				
T1 ECV → FU DE	-.03	-.05	.06	-.17, .08
T1 NR → FU DE	**-.12**	**-.39**	**.16**	**-.71, -.09**
T1 DE → FU DE	**.13**	**.14**	**.06**	**.03, .24**
Indirect effects of T1 variables on FU outcomes				
T1 ECV → T1 DE → FU DE	**.02**	**.03**	**—**	**.01, .06**
T1 NR → T1 DE → FU DE	.001	.002	—	-.02, .03

Table S3.2 (cont'd)

Best Fitting Model in Boys

Direct effects of T1 variables on T1 outcomes				
T1 ECV → T1 DE	**.21**	**.23**	**.03**	**.16, .30**
T1 NR → T1 DE	.01	.02	.09	-.15, .20
T1 age → T1 DE	.02	.05	.09	-.14, .24
Black racial identity → T1 DE	**.11**	**1.49**	**.45**	**.65, 2.44**
Other POC racial identity → T1 DE	-.003	-.05	.43	-.90, .79
T1 family income → T1 DE	-.05	-.10	.06	-.22, .02
Direct effects of FU variables on FU outcomes				
FU ECV → FU DE	**.18**	**.29**	**.09**	**.14, .47**
FU NR → FU DE	.05	.11	.13	-.16, .36
FU age → FU DE	.08	.16	.11	-.06, .37
Black racial identity → FU DE	-.06	-.83	.67	-2.11, .45
Other POC racial identity → FU DE	-.08	-1.19	.64	-2.41, .12
FU family income → FU DE	-.04	-.11	.16	-.44, .18
Direct effects of T1 variables on FU outcomes				
T1 ECV → FU DE	-.04	-.05	.06	-.17, .08
T1 NR → FU DE	**-.13**	**-.39**	**.16**	**-.71, -.09**
T1 DE → FU DE	**.13**	**.14**	**.06**	**.03, .24**
Indirect effects of T1 variables on FU outcomes				
T1 ECV → T1 DE → FU DE	**.03**	**.03**	**—**	**.01, .06**
T1 NR → T1 DE → FU DE	.001	.002	—	-.02, .03

Note. T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; NR = neighborhood resources; ECV = reported impact of exposure to community violence; POC = person of color. Note that standardized effects were computed separately for girls and boys (i.e., using sex-specific standard deviations), and thus could differ slightly even when unstandardized effects were

Table S3.2 (cont'd)

constrained to equality across sex. Dashes indicate that a parameter is not applicable. Statistically significant paths are bolded.

Table S3.3. *Estimates for all parameters from the best-fitting model for cross-sectional and longitudinal effects of neighborhood factors on disordered eating in the full sample*

Parameter	Standardized Estimate	Unstandardized Estimate	SE	95% CI
Best Fitting Model in Girls				
Direct effects of T1 variables on T1 outcomes				
T1 ECV → T1 DE	**.15**	**.21**	**.03**	**.15, .27**
T1 NR → T1 DE	.01	.02	.09	-.15, .21
T1 age → T1 DE	**-.11**	**-.32**	**.10**	**-.51, -.12**
Black racial identity → T1 DE	**.10**	**1.47**	**.45**	**.66, 2.39**
Other POC racial identity → T1 DE	**.09**	**1.36**	**.55**	**.29, 2.41**
T1 family income → T1 DE	-.05	-.11	.06	-.23, .02
Direct effects of FU variables on FU outcomes				
FU ECV → FU DE	**.14**	**.23**	**.06**	**.11, .36**
FU NR → FU DE	.04	.11	.13	-.16, .37
FU age → FU DE	.07	.15	.11	-.06, .37
Black racial identity → FU DE	-.06	-.96	.66	-2.19, .27
Other POC racial identity → FU DE	-.07	-1.21	.65	-2.41, .10
FU family income → FU DE	-.02	-.07	.17	-.42, .25
Direct effects of T1 variables on FU outcomes				
T1 ECV → FU DE	-.01	-.01	.05	-.12, .09
T1 NR → FU DE	**-.13**	**-.41**	**.16**	**-.72, -.11**
T1 DE → FU DE	**.12**	**.14**	**.05**	**.03, .24**
Indirect effects of T1 variables on FU outcomes				
T1 ECV → T1 DE → FU DE	**.02**	**.03**	**—**	**.01, .05**
T1 NR → T1 DE → FU DE	.001	.003	—	-.02, .03

Table S3.3 (cont'd)

Best Fitting Model in Boys

Direct effects of T1 variables on T1 outcomes				
T1 ECV → T1 DE	**.19**	**.21**	**.03**	**.15, .27**
T1 NR → T1 DE	.01	.02	.09	-.15, .21
T1 age → T1 DE	.01	.03	.10	-.16, .22
Black racial identity → T1 DE	**.11**	**1.47**	**.45**	**.66, 2.39**
Other POC racial identity → T1 DE	.003	.05	.44	-.79, .91
T1 family income → T1 DE	-.05	-.11	.06	-.23, .02
Direct effects of FU variables on FU outcomes				
FU ECV → FU DE	**.21**	**.23**	**.06**	**.11, .36**
FU NR → FU DE	.05	.11	.13	-.16, .37
FU age → FU DE	.07	.15	.11	-.06, .37
Black racial identity → FU DE	-.07	-.96	.66	-2.19, .27
Other POC racial identity → FU DE	-.08	-1.21	.65	-2.41, .10
FU family income → FU DE	-.02	-.07	.17	-.42, .25
Direct effects of T1 variables on FU outcomes				
T1 ECV → FU DE	-.01	-.01	.05	-.12, .09
T1 NR → FU DE	**-.13**	**-.41**	**.16**	**-.72, -.11**
T1 DE → FU DE	**.13**	**.14**	**.05**	**.03, .24**
Indirect effects of T1 variables on FU outcomes				
T1 ECV → T1 DE → FU DE	**.02**	**.03**	**—**	**.01, .05**
T1 NR → T1 DE → FU DE	.001	.003	—	-.02, .03

Note. T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; NR = neighborhood resources; ECV = exposure to community violence; POC = person of color. Note that standardized effects were computed separately for girls and boys (i.e., using sex-specific standard deviations), and thus could differ slightly even when unstandardized effects were constrained to

Table S3.3 (cont'd)

equality across sex. Dashes indicate that a parameter is not applicable. Statistically significant paths are bolded.

Table S3.4. *Model fit comparisons constraining parameters across sex for cross-sectional and longitudinal effects, including participants in the under-resourced arm at intake only*

Model	AIC	BIC	SABIC	χ^2 (df)	Scaling Correction Factor	$\chi^2\Delta$ (df)	*p*
Full model	31111.59	31936.77	31403.19	19.35 (12)	0.9768	—	—
Constrain all parameters	31107.36	31858.85	31372.92	43.37 (27)	1.0298	24.03 (15)	.065
T1 associations							
Constrain T1 ECV → T1 DE	31109.89	31930.15	31399.75	19.00 (13)	1.0103	0.21 (1)	.647
Constrain T1 NR → T1 DE	31112.02	31932.29	31401.88	21.94 (13)	0.9724	2.64 (1)	.104
Constrain T1 age → T1 DE	31112.03	31932.30	31401.89	21.67 (13)	0.9849	2.26 (1)	.133
Constrain Black racial identity → T1 DE	31109.60	31929.86	31399.46	19.56 (13)	0.9673	0.01 (1)	.920
Constrain other POC identity → T1 DE	31113.38	31933.64	31403.24	23.17 (13)	0.9793	3.75 (1)	.053
Constrain T1 INC → T1 DE	31109.65	31929.92	31399.51	19.19 (13)	0.9882	0.05 (1)	.823
FU associations							
Constrain FU ECV → FU DE	31111.40	31931.66	31401.26	20.59 (13)	1.0059	1.34 (1)	.247
Constrain FU NR → FU DE	31110.70	31930.96	31400.55	20.61 (13)	0.9707	1.23 (1)	.267
Constrain FU age → FU DE	31113.22	31933.48	31403.08	22.38 (13)	1.0068	2.65 (1)	.104
Constrain Black racial identity → FU DE	31110.49	31930.75	31400.35	20.66 (13)	0.9583	1.21 (1)	.271
Constrain other POC identity → FU DE	31109.79	31930.06	31399.65	18.98 (13)	1.0066	0.15 (1)	.699
Constrain FU INC → FU DE	31110.38	31930.64	31400.24	20.15 (13)	0.9774	0.80 (1)	.371
T1 → FU associations							
Constraint T1 DE → FU DE	31115.03	31935.29	31404.88	25.00 (13)	0.9734	5.82 (1)	.016
Constrain T1 ECV → FU DE	31109.62	31929.88	31399.48	19.08 (13)	0.9924	0.03 (1)	.862
Constrain T1 NR → FU DE	31110.03	31930.30	31399.89	19.96 (13)	0.9693	0.50 (1)	.480

Table S3.4 (cont'd)

Constrain all parameters except T1 other POC identity → T1 DE, T1 DE → FU DE	31102.65	31863.97	31371.68	34.71 (25)	1.0360	15.64 (13)	.269
Constrain all parameters except T1 other POC identity → T1 DE, T1 DE → FU DE, FU age → FU DE	**31099.77**	**31866.01**	**31370.54**	**30.06 (24)**	**1.0343**	**11.16 (12)**	**.515**

Note. T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; ECV = exposure to community violence; NR = neighborhood resources; POC = person of color; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion; χ^2 **Δ** = change in chi-square; df = degrees of freedom. The best-fitting model is bolded.

Table S3.5. *Estimates for all parameters from the best-fitting model for cross-sectional and longitudinal effects of neighborhood factors on disordered eating, including participants in the under-resourced arm at intake only*

Parameter	Standardized Estimate	Unstandardized Estimate	SE	95% CI
Best Fitting Model in Girls				
Direct effects of T1 variables on T1 outcomes				
T1 ECV → T1 DE	**.15**	**.16**	**.04**	**.08, .25**
T1 NR → T1 DE	.03	.09	.11	-.12, .29
T1 age → T1 DE	**-.08**	**-.21**	**.10**	**-.41, -.01**
Black racial identity → T1 DE	**.11**	**1.36**	**.43**	**.51, 2.20**
Other POC racial identity → T1 DE	**.13**	**1.72**	**.64**	**.39, 2.97**
T1 family income → T1 DE	-.06	-.12	.07	-.27, .03
Direct effects of FU variables on FU outcomes				
FU ECV → FU DE	**.11**	**.17**	**.09**	**.02, .36**
FU NR → FU DE	.05	.11	.17	-.23, .46
FU age → FU DE	**.25**	**.60**	**.18**	**.22, .93**
Black racial identity → FU DE	-.05	-.76	.84	-2.53, .80
Other POC racial identity → FU DE	-.09	-1.35	.99	-3.04, .77
FU family income → FU DE	.003	.01	.21	-.41, .42
Direct effects of T1 variables on FU outcomes				
T1 ECV → FU DE	-.04	-.05	.08	-.19, .11
T1 NR → FU DE	**-.14**	**-.44**	**.20**	**-.82, -.04**
T1 DE → FU DE	**.24**	**.28**	**.11**	**.06, .49**
Indirect effects of T1 variables on FU outcomes				
T1 ECV → T1 DE → FU DE	**.04**	**.05**	**—**	**.01, .10**
T1 NR → T1 DE → FU DE	.01	.03	—	-.03, .10

Table S3.5 (cont'd)

Best Fitting Model in Boys

Direct effects of T1 variables on T1 outcomes				
T1 ECV → T1 DE	**.16**	**.16**	**.04**	**.08, .25**
T1 NR → T1 DE	.03	.09	.11	-.12, .29
T1 age → T1 DE	**-.08**	**-.21**	**.10**	**-.41, -.01**
Black racial identity → T1 DE	**.12**	**1.36**	**.43**	**.51, 2.20**
Other POC racial identity → T1 DE	-.01	-.09	.66	-1.39, 1.19
T1 family income → T1 DE	-.06	-.12	.07	-.27, .03
Direct effects of FU variables on FU outcomes				
FU ECV → FU DE	**.18**	**.17**	**.09**	**.02, .36**
FU NR → FU DE	.06	.11	.17	-.23, .46
FU age → FU DE	.03	.07	.20	-.30, .43
Black racial identity → FU DE	-.06	-.76	.84	-2.53, .80
Other POC racial identity → FU DE	-.09	-1.35	.99	-3.04, .77
FU family income → FU DE	.004	.01	.21	-.41, .42
Direct effects of T1 variables on FU outcomes				
T1 ECV → FU DE	-.05	-.05	.08	-.19, .11
T1 NR → FU DE	**-.14**	**-.44**	**.20**	**-.82, -.04**
T1 DE → FU DE	.03	.03	.09	-.14, .22
Indirect effects of T1 variables on FU outcomes				
T1 ECV → T1 DE → FU DE	.01	.01	—	-.02, .04
T1 NR → T1 DE → FU DE	.001	.003	—	-.02, .04

Note. T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; NR = neighborhood resources; ECV = exposure to community violence; POC = person of color. Note that standardized effects were computed separately for girls and boys (i.e., using sex-specific standard deviations), and thus could differ slightly even when unstandardized effects were constrained to

Table S3.5 (cont'd)

equality across sex. Dashes indicate that a parameter is not applicable. Statistically significant paths are bolded.

Table S3.6. *Model fit comparisons constraining parameters across sex for supplemental model examining associations between global neighborhood disadvantage and disordered eating through neighborhood factors*

Model	AIC	BIC	SABIC	χ^2 (df)	Scaling Correction Factor	$\chi^2\Delta$ (df)	*p*
Full model	69199.37	70460.81	69749.14	11.81 (14)	1.0170	—	—
Constrain all parameters	69197.50	70346.31	69698.19	45.82 (34)	1.0944	33.21 (20)	.032
T1 associations							
Constrain T1 ECV → T1 DE	69200.21	70456.02	69747.53	13.99 (15)	1.0620	1.68 (1)	.195
Constrain T1 NR → T1 DE	69198.16	70453.97	69745.48	12.51 (15)	1.0233	0.72 (1)	.396
Constrain T1 ADI → T1 DE	69197.69	70453.50	69745.01	12.03 (15)	1.0255	0.29 (1)	.590
Constrain T1 ADI → T1 ECV	69197.41	70453.22	69744.73	11.50 (15)	1.0474	0.03 (1)	.862
Constrain T1 ADI → T1 NR	69198.00	70453.81	69745.32	11.93 (15)	1.0597	0.38 (1)	.538
Constrain T1 age → T1 DE	69205.58	70461.39	69752.90	19.74 (15)	1.0244	7.28 (1)	.007
Constrain Black racial identity → T1 DE	69198.24	70454.05	69745.56	12.32 (15)	1.0453	0.61 (1)	.435
Constrain other POC identity → T1 DE	69201.80	70457.61	69749.12	16.09 (15)	1.0214	4.09 (1)	.043
Constrain T1 INC → T1 DE	69197.38	70453.19	69744.70	11.55 (15)	1.0405	0.01 (1)	.920
FU associations							
Constrain FU ECV → FU DE	69201.63	70457.44	69748.95	15.71 (15)	1.0357	3.28 (1)	.070
Constrain FU NR → FU DE	69198.54	70454.35	69745.86	13.24 (15)	0.9957	1.69 (1)	.194
Constrain FU ADI → FU DE	69197.89	70453.70	69745.21	12.61 (15)	0.9937	0.78 (1)	.377
Constrain FU age → FU DE	69199.00	70454.81	69746.32	13.36 (15)	1.0215	1.51 (1)	.219
Constrain Black racial identity → FU DE	69197.37	70453.18	69744.69	12.00 (15)	1.0016	0.01 (1)	.920
Constrain other POC identity → FU DE	69198.30	70454.11	69745.62	12.51 (15)	1.0346	0.73 (1)	.393
Constrain FU INC → FU DE	69197.38	70453.19	69744.70	11.98 (15)	1.0035	0.02 (1)	.888
T1 → FU associations							
Constraint T1 DE → FU DE	69200.03	70455.84	69747.35	14.29 (15)	1.0271	2.28 (1)	.131

Table S3.6 (cont'd)

Constrain T1 ECV → FU DE	69197.56	70453.37	69744.88	11.96 (15)	1.0198	0.18 (1)	.671
Constrain T1 NR → FU DE	69197.84	70453.65	69745.16	12.42 (15)	1.0050	0.57 (1)	.450
Constrain T1 ADI → FU DE	69203.10	70458.91	69750.42	18.47 (15)	0.9605	33.85 (1)	<.001
Constrain all parameters except T1 age → T1 DE, other POC identity → T1 DE, FU ECV → FU DE, T1 ADI → FU DE	**69181.75**	**70353.09**	**69692.25**	**24.16 (30)**	**1.0921**	**12.42 (16)**	**.715**
Constrain all parameters except T1 age → T1 DE, other POC identity → T1 DE, T1 ADI → FU DE	69184.40	70350.10	69692.45	28.26 (31)	1.0982	16.34 (1)	.429

Note. T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; ADI = Area Deprivation Index; ECV = exposure to community violence; NR = neighborhood resources; POC = person of color; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; SABIC = sample size adjusted Bayesian Information Criterion; χ^2 **Δ** = change in chi-square; df = degrees of freedom. The best-fitting model is bolded.

Table S3.7. *Estimates for all parameters from the best-fitting supplemental model examining associations between global neighborhood disadvantage and disordered eating through neighborhood factors*

Parameter	Standardized Estimate	Unstandardized Estimate	SE	95% CI
Best Fitting Model in Girls				
Direct effects of T1 variables on T1 outcomes				
T1 ADI→ T1 DE	.05	.08	.05	-.02, .18
T1 ECV → T1 DE	**.15**	**.20**	**.03**	**.14, .27**
T1 NR → T1 DE	.01	.03	.09	-.14, .22
T1 age → T1 DE	**-.11**	**-.31**	**.10**	**-.50, -.10**
Black racial identity → T1 DE	**.09**	**1.34**	**.45**	**.51, 2.25**
Other POC racial identity → T1 DE	**.09**	**1.30**	**.55**	**.19, 2.36**
T1 INC → T1 DE	-.03	-.08	.07	-.21, .05
T1 ADI→ T1 ECV	**.23**	**.31**	**.04**	**.23, .39**
T1 ADI→ T1 NR	**-.09**	**-.06**	**.02**	**-.11, -.01**
Indirect effects of T1 variables on T1 outcomes				
T1 ADI→ T1 ECV → T1 DE	**.03**	**.06**	**—**	**.04, .09**
T1 ADI→ T1 NR → T1 DE	-.001	-.002	—	-.02, .01
Direct effects of FU variables on FU outcomes				
FU ADI→ FU DE	-.08	-.15	.16	-.47, .19
FU ECV → FU DE	**.25**	**.42**	**.12**	**.19, .65**
FU NR → FU DE	.06	.17	.13	-.11, .44
FU age → FU DE	.07	.16	.11	-.05, .37
Black racial identity → FU DE	-.05	-.85	.65	-2.06, .45
Other POC racial identity → FU DE	-.07	-1.13	.65	-2.32, .15
FU INC → FU DE	-.02	-.06	.17	-.39, .24
Direct effects of T1 variables on FU outcomes				
T1 ADI→ FU DE	-.11	-.23	.20	-.63, .14

Table S3.7 (cont'd)

T1 ECV → FU DE	.001	.001	.06	-.11, .12
T1 NR → FU DE	**-.14**	**-.45**	**.16**	**-.77, -.15**
T1 DE → FU DE	**.11**	**.13**	**.05**	**.02, .23**
Indirect effects of T1 variables on FU outcomes				
T1 ADI→ T1 DE → FU DE	.01	.01	—	-.002, .03
T1 ADI→ T1 ECV → FU DE	<.001	<.001	—	-.04, .03
T1 ADI→ T1 NR → FU DE	**.01**	**.03**	**—**	**.003, .06**
T1 ADI→ T1 ECV → T1 DE → FU DE	**.004**	**.01**	**—**	**.001, .02**
T1 ADI→ T1 NR → T1 DE → FU DE	<.001	<.001	—	-.002, .001
<u>Best Fitting Model in Boys</u>				
Direct effects of T1 variables on T1 outcomes				
T1 ADI→ T1 DE	.05	.08	.05	-.02, .18
T1 ECV → T1 DE	**.18**	**.20**	**.03**	**.14, .27**
T1 NR → T1 DE	.01	.03	.09	-.14, .22
T1 age → T1 DE	.01	.03	.10	-.16, .22
Black racial identity → T1 DE	**.10**	**1.34**	**.45**	**.51, 2.25**
Other POC racial identity → T1 DE	.002	.03	.44	-.80, .91
T1 INC → T1 DE	-.04	-.08	.07	-.21, .05
T1 ADI→ T1 ECV	**.18**	**.31**	**.04**	**.23, .39**
T1 ADI→ T1 NR	**-.10**	**-.06**	**.02**	**-.11, -.01**
Indirect effects of T1 variables on T1 outcomes				
T1 ADI→ T1 ECV → T1 DE	**.03**	**.06**	**—**	**.04, .09**
T1 ADI→ T1 NR → T1 DE	-.001	-.002	—	-.02, .01
Direct effects of FU variables on FU outcomes				
FU ADI→ FU DE	-.08	-.15	.16	-.47, .19
FU ECV → FU DE	**.15**	**.17**	**.07**	**.02, .31**

Table S3.7 (cont'd)

FU NR → FU DE	.08	.17	.13	-.11, .44
FU age → FU DE	.08	.16	.11	-.05, .37
Black racial identity → FU DE	-.06	-.85	.65	-2.06, .45
Other POC racial identity → FU DE	-.07	-1.13	.65	-2.32, .15
FU INC → FU DE	-.02	-.06	.17	-.39, .24
Direct effects of T1 variables on FU outcomes				
T1 ADI→ FU DE	**.19**	**.37**	**.17**	**.02, .67**
T1 ECV → FU DE	.001	.001	.06	-.11, .12
T1 NR → FU DE	**-.15**	**-.45**	**.16**	**-.77, -.15**
T1 DE → FU DE	**.12**	**.13**	**.05**	**.02, .23**
Indirect effects of T1 variables on FU outcomes				
T1 ADI→ T1 DE → FU DE	.01	.01	—	-.002, .03
T1 ADI→ T1 ECV → FU DE	<.001	<.001	—	-.04, .03
T1 ADI→ T1 NR → FU DE	**.01**	**.03**	**—**	**.003, .06**
T1 ADI→ T1 ECV → T1 DE → FU DE	**.004**	**.01**	**—**	**.001, .02**
T1 ADI→ T1 NR → T1 DE → FU DE	<.001	<.001	—	-.002, .001

Note. T1 = intake; FU = follow-up; ADI = Area Deprivation Index; DE = Minnesota Eating Behavior Survey total score; NR = neighborhood resources; ECV = exposure to community violence; POC = person of color. Note that standardized effects were computed separately for girls and boys (i.e., using sex-specific standard deviations), and thus could differ slightly even when unstandardized effects were constrained to equality across sex. Dashes indicate that a parameter is not applicable. Statistically significant paths are bolded.

Figures

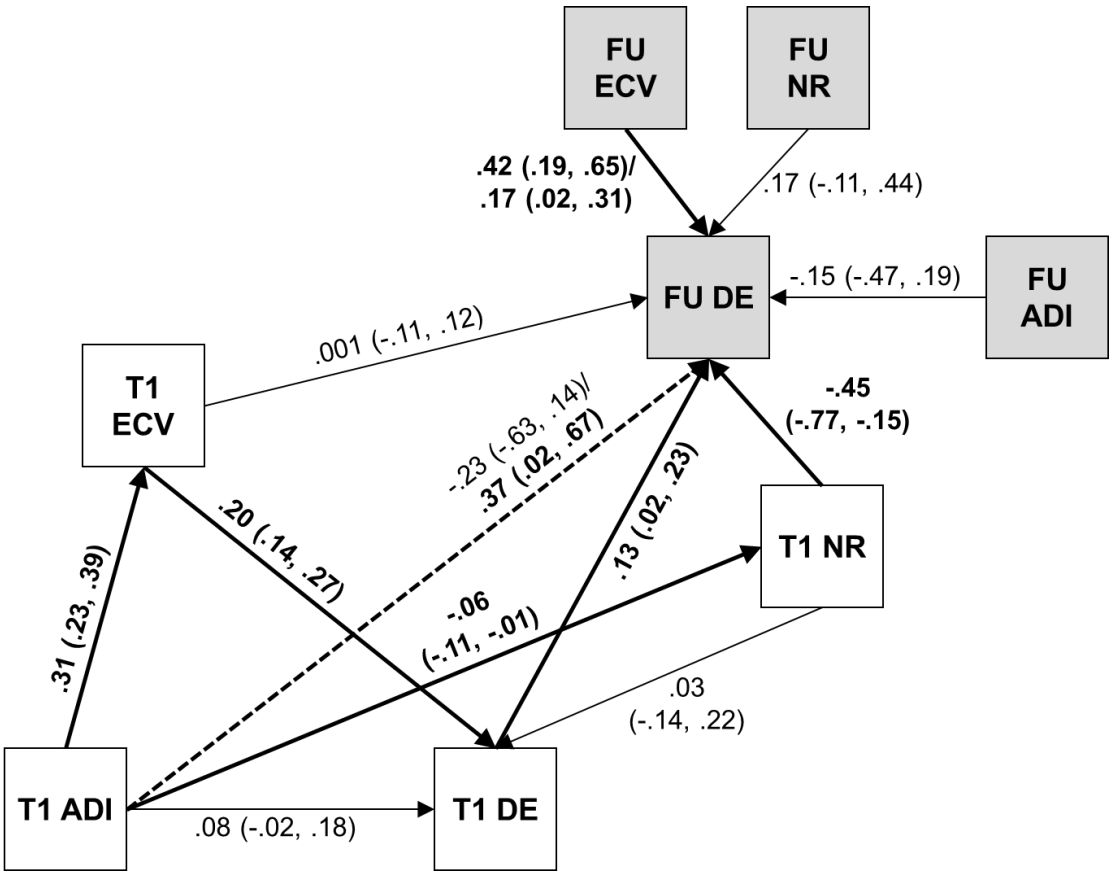


Figure S3.1. *Best-fitting supplemental model examining associations between global neighborhood disadvantage and disordered eating through neighborhood factors*. T1 = intake; FU = follow-up; DE = Minnesota Eating Behavior Survey total score; ADI = Area Deprivation Index; ECV = exposure to community violence; NR = neighborhood resources. White boxes represent variables at T1 and grey boxes represent variables at FU. Bolded lines represent paths that are significant in participants of both sexes and dashed lines represent paths that are significant in participants of one sex only. For estimates that differed by sex, the estimate for girls is listed first, followed by a slash and the estimate for boys. 95% confidence intervals are included in parentheses. Covariates are not depicted for legibility.