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Disadvantage and Disordered Eating in Boys: Examining Phenotypic and Genotype x Environment Associations across Development

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Abstract

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. 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. 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. Results highlight disadvantage as a novel risk factor for disordered eating in boys, particularly those with genetic vulnerabilities.

General Scientific Summary

This was the first study to examine phenotypic and genotype x environment associations between disadvantage and disordered eating in boys during the critical developmental risk period. We

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found that both familial and neighborhood disadvantage were associated with significantly greater genetic influences on disordered eating in boys during early adrenarche, and significantly greater phenotypic disordered eating symptoms during late adrenarche. Results suggest that boys living in disadvantaged environments may be at elevated risk for disordered eating, particularly if they have underlying genetic vulnerabilities.

Keywords

disordered eating; eating disorders; poverty; neighborhood disadvantage; socioeconomic status; etiology; genotype x environment interaction; males; puberty; adrenarche

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 ($M_{\text{age}} = 12.27$, $SD = 2.96$) 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.

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: α = .77; ages 13-17; α = .81) and pubertal development (early adrenarche: α = .70; early gonadarche: α = .78; mid/late gonadarche: α = .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 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 (α = .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

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[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.

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. 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) 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), disadvantage (βxD, βyD, βzD in Figure S1), and their interaction (βxPD, βyPD, βzPD in Figure S1). 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). 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 ($\beta = .08$, $p = .001$, 95% CI [.03, .13]) but not in early adrenarche ($\beta = -.03$, $p = .640$, 95% CI [−.14, .09]). Similarly, when controlling for BMI, family SES was significantly associated with disordered eating for boys in late adrenarche ($\beta = -.08$, $p = .001$, 95% CI [−.12, −.03]), but not in early adrenarche ($\beta = .05$, $p = .360$, 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 S3–S4 and Figure S2), 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 S2). 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 and 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 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 2–3 and Figures 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 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 and 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.

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[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.

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.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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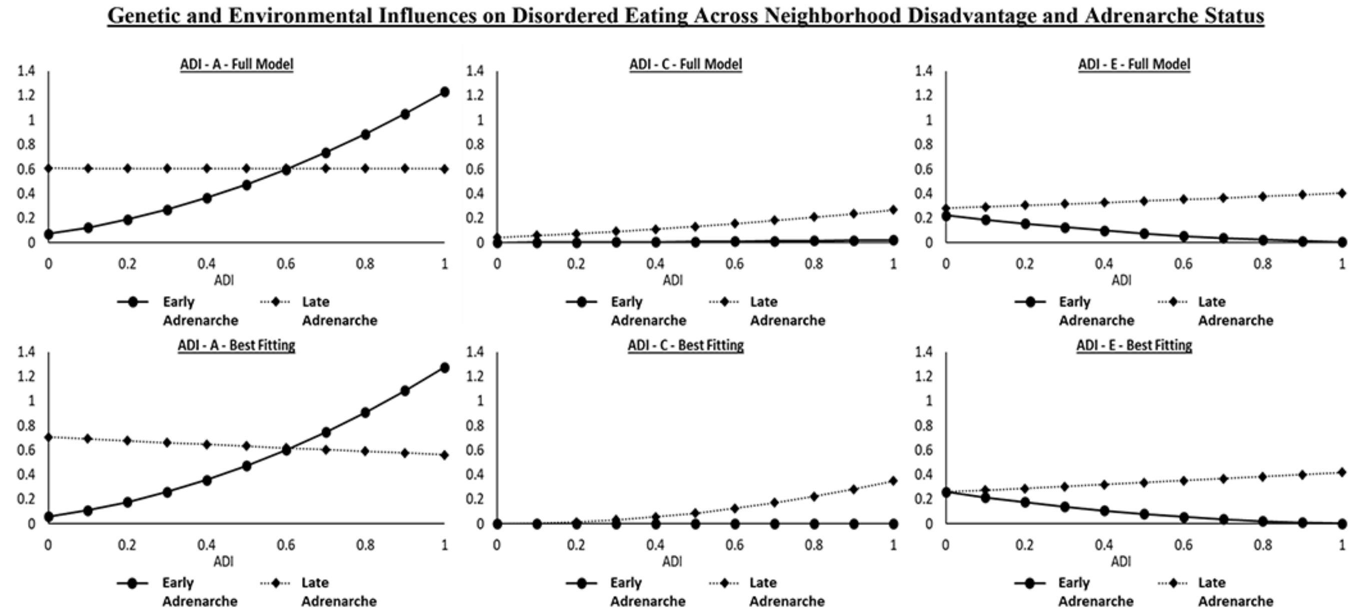

Figure 1.

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.

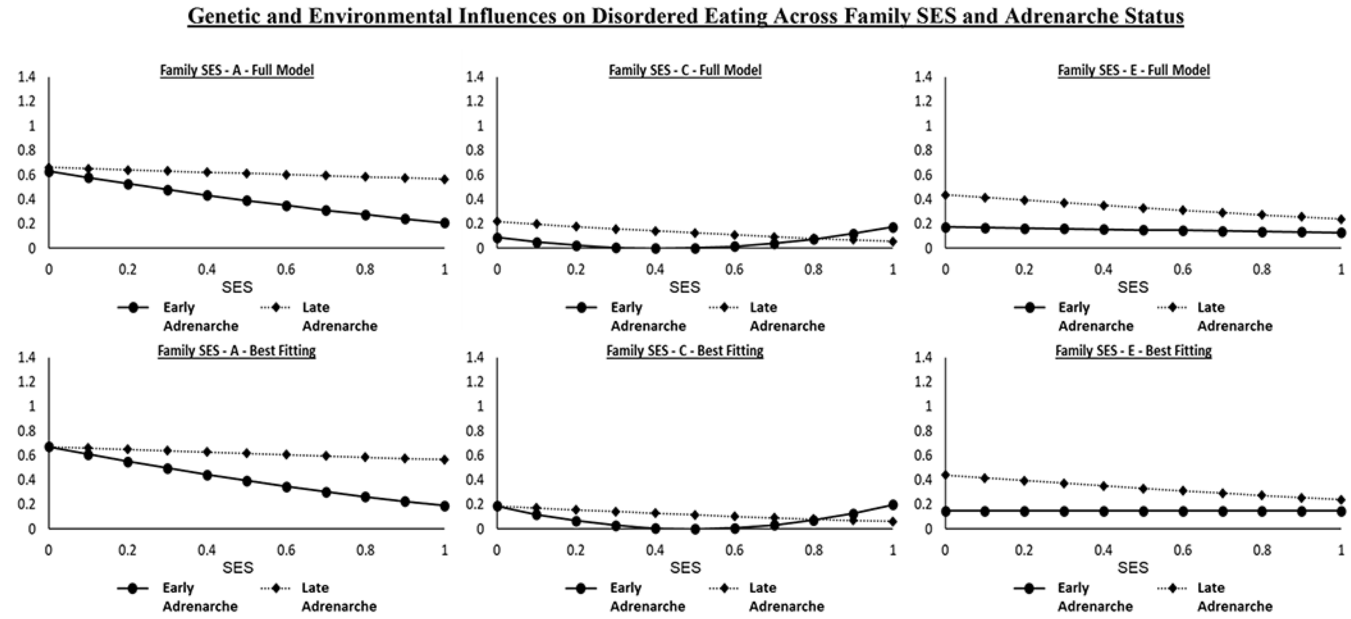

Figure 2.

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

Table 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

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**
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/Caucasian. Effects significant at $p < .05$ are bolded.

Table 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

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 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.