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The associations of polygenic scores for risky behaviors and parenting behaviors with adolescent externalizing problems

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Abstract

The current study focused on longitudinal effects of genetics and parental behaviors and their interplay on externalizing behaviors in a panel study following individuals from adolescence to young adulthood. The nationally representative sample of Add Health participants of European ancestry included $N = 4{,}142$ individuals, measured on three occasions. Parenting was operationalized as experiences with child maltreatment and maternal closeness. Externalizing problems were operationalized as alcohol use, cannabis use, and antisocial behaviors. Genetic effects were operationalized as a polygenic score (PGS) of risky behaviors. The results showed significant effects for child maltreatment, maternal closeness, and PGS, above and beyond other factors and previous levels of externalizing behaviors. Furthermore, maternal closeness was found to negatively correlate with PGS. No significant interaction effects of parenting and PGS were found. The results underscore the joint independent effects of parenting and genetics on the change in externalizing behaviors from adolescence to young adulthood.

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Declarations

Ethics approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The IRB approval was obtained at the site of the original data collection. IRB approval for the analysis of the secondary data was given to all authors of this study.

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Adolescence is a period traditionally marked by increases in externalizing behaviors, which encompasses a broad set of behaviors related to behavioral disinhibition, including substance use, risky sex, and antisocial behaviors (Krueger et al. 2002; McKee et al. 2008; Samek et al. 2015; Luk et al. 2016; Harden et al. 2017). These behaviors incur high individual and societal costs (Cicchetti and Toth 2005; Foster and Jones 2005; Carter 2019). Both parenting behaviors (Fletcher et al. 2004; McKee et al. 2008) and genetic influences (Krueger et al. 2002; Kendler et al. 2011; Hicks et al. 2013; Samek et al. 2015; Dick et al. 2016) contribute to variation in these externalizing problems. Importantly, parenting and genetic influences do not act in isolation. Parents also shape the environment in which children grow up, which can then affect the extent to which children's genetic liability is expressed. The current study focused on analyzing the interplay of genetic influences and parenting behaviors during adolescence, and the longitudinal associations with the development of externalizing behaviors in young adulthood.

Externalizing Behaviors

Parenting and Adolescent Externalizing Behaviors

Substantial research has found that that parenting behaviors can positively and negatively affect adolescent adjustment (Cicchetti and Toth 2005; McKee et al. 2008; Vieno et al. 2009; Kim and Cicchetti 2009; Lowe and Dotterer 2013). Specifically, two dimensions of parenting have emerged as strong correlates of adolescent externalizing behaviors, closeness and hostility (Steinberg and Morris 2001; McKee et al. 2008; Pinquart 2017; Tung et al. 2019). Parental closeness is characterized as a warm and supportive parent-child relationship (Baumrind 1991). Behavioral indicators of warm, close parenting include involvement, acceptance, positive affect, and praise (McKee et al., 2008). In contrast, hostility is characterized by harsh parenting behaviors, which manifest as aggression, anger, disapproval, negative affect, and may also include child maltreatment (i.e., physical, sexual, or emotional abuse and/or neglect; McKee et al. 2008). Together, parental closeness and child maltreatment can be conceptualized as manifestations of two parenting behaviors with widely differing effects on children, and thus, different developmental outcomes, one beneficial and one detrimental. These parenting behaviors create a climate that shapes adolescents' emotional, behavioral, and social regulation and competence (Darling and Steinberg 1993; Steinberg 2001).

Developmental research has shown that parental closeness is protective against adolescent externalizing behavior (Vieno et al. 2009; MacKenzie et al. 2012; Berkien et al. 2012). As mentioned previously, higher levels of parental closeness are associated with higher levels of warmth, acceptance, involvement, monitoring, and praise (McKee et al. 2008). In their review of the associations between parenting behaviors and children's internalizing and externalizing behaviors, McKee et al. (2008) posit that one mechanism by which higher parental closeness influences externalizing behaviors is through emotional and behavioral

regulation. Research provides evidence that low levels of parental warmth can impede children's ability to regulate their emotions and behaviors (Morris et al. 2007, 2017). Along those same lines, high levels of parental monitoring can protect against adolescent externalizing behaviors through limit-setting and behavioral control, which promotes self-regulation (Hoeve et al. 2009) This suggests that parental closeness improves adolescent adjustment by creating an environmental climate that fosters adolescents' emotional and behavioral regulation (Deutsch et al. 2012).

In contrast, hostile and harsh parenting is often associated with greater externalizing problems in adolescence (Dodge et al. 1994; Steinberg et al. 1994, 2006; McKee et al. 2008). One extreme form of hostile parenting behavior is child maltreatment. Prior research provides evidence that child maltreatment is consistently found to be associated with poorer adjustment in children, including higher levels of risk-taking, delinquency, and antisocial behaviors (e.g., (Keiley et al. 2001; Manly et al. 2001; Kim and Cicchetti 2009). Child maltreatment creates a pathogenic relational environment in which parent-child interactions create a cycle of negative reinforcement whereby parents are more likely to engage in harsh, coercive behavior, and adolescents are more likely to engage in externalizing behaviors (McKee et al. 2008). Adolescents socialized to use these harsh, coercive behaviors are then more likely to engage in delinquent and antisocial behaviors outside of the home (McKee et al. 2008; Pinquart 2017). Taken together, this implies that adolescent externalizing behaviors may be modeled and reinforced by harsh parenting behaviors.

The associations between parenting behaviors and adolescent externalizing behaviors are particularly concerning because they are associated with increased externalizing behaviors in young adulthood, a critical period for the development of risky substance use (Schulenberg et al. 1996; Sher and Rutledge 2007; Brown et al. 2008; Foster et al. 2018; Johnson et al. 2019). Externalizing behaviors do not arise de novo during young adulthood; rather, such behaviors are associated with trajectories of risk-related or risk-taking behaviors that begin earlier in development (Powell et al. 2010; Korhonen et al. 2010; Chen & Jacobson 2012). This underscores the need to employ a developmental and lifespan approach when considering risk and protective factors.

Genetics of Externalizing Behaviors

Evidence from twin studies (Krueger et al. 2002; Kendler et al. 2011; Hicks et al. 2013; Samek et al. 2015) and molecular genetic studies (Salvatore et al. 2015; Linnér et al. 2019; Su et al. 2018; Dick et al. 2019) indicate that individual externalizing behaviors are moderately heritable, with h^2 ranging from 40–60%. When we consider these traits jointly, we find evidence of a shared, highly heritable underlying factor (~80%; Krueger et al. 2002), and the majority of genetic variance of these traits is accounted for by this shared liability towards externalizing behaviors. For example, ~75–80% of the heritability for alcohol use disorders is shared in common with other externalizing disorders (Kendler and Myers 2014). Additionally, these externalizing traits are also associated with personality characteristics, including impulsivity and sensation seeking (Krueger et al. 2002).

There is also growing recognition that genetic influences on behavioral traits, such as adolescent externalizing behaviors, may interface with the environment in complex ways.

First, gene-environment correlations (*r*GE), or the degree to which environmental factors are influenced by genetic factors (Shanahan and Boardman 2009; Dick et al. 2018), may account for a proportion of genetic variance in adolescent externalizing behaviors. Of relevance to the present study, parenting behaviors (via *r*GE) serve as one mechanism through which genetic predispositions influence externalizing problems (Horwitz and Neiderhiser 2011; Avinun and Knafo 2014; Su et al. 2018). Parents and their offspring demonstrate overlap in shared genetic liability for externalizing behaviors, and these parents create an environmental climate that encourages or reinforces adolescent delinquent and antisocial behaviors (i.e., passive and evocative *r*GE (Scarr and McCartney 1983). In other words, adolescent externalizing behaviors are partially explained through common genetic predispositions with their parents, which may also be correlated with the environment that their parents cultivate. Thus, extant research suggests that parenting behaviors are an important mechanism to consider in the study of genetic influences on adolescent externalizing (Horwitz and Neiderhiser 2011; Avinun and Knafo 2014; Su et al. 2018).

Second, gene-environment interactions (GxE), or the degree to which genetic influences are moderated or conditioned by environmental factors (Shanahan and Boardman 2009; Dick et al. 2018), may also affect adolescent externalizing behaviors. Previous studies indicate that the influence of genetic predispositions on behavior decrease in environments with high social control (e.g., parental closeness, involvement, or monitoring; Shanahan & Boardman, 2009). On the other hand, genetic influences on substance misuse were found to be higher in environments characterized by high parental negativity, low parental warmth and monitoring, affiliation with delinquent peers, among others (Dick et al. 2007; Kendler et al. 2011; Samek et al. 2015). Taken together, this indicates that the influence of genetic predispositions on adolescent externalizing behaviors may vary as a function of parenting behaviors.

In the present study, we evaluated the longitudinal associations between both parenting behaviors and genetic liability in adolescence with externalizing behaviors in early adulthood. Employing three waves of data spanning seven years, the study tested whether there exist discernible genetic effects on a variety of externalizing behaviors in young adulthood, even when controlling for their antecedent levels. In this way, we modeled the genetic and environmental effects on the developmental changes in the externalizing behaviors. Further, we assessed whether parental behaviors in adolescence or childhood had persistent and long-lasting effects on developmental outcomes in young adulthood and characterized whether these parental behaviors were associated with genetic predispositions. We measured genetic predispositions using polygenic risk scores derived from a discovery genome-wide association study (GWAS) of risky behaviors (Linnér et al. 2019), which is composed of four behaviors that reflect some key dimensions of the externalizing spectrum (substance use, risky sex, and sensation seeking). We limited our analytic sample to individuals of primarily European ancestries because the discovery GWAS used to create the polygenic scores was conducted in individuals of European ancestries. Polygenic scores perform poorly when there is a mismatch between discovery and target samples (Martin et al. 2017; Duncan et al. 2019). We measured externalizing behaviors using three different behaviors: alcohol consumption, cannabis use, and antisocial behavior (Krueger et al. 2002) and tested the following research questions:

RQ1: Is parenting environment in adolescence (indicated by closeness and child maltreatment) associated with externalizing behaviors in young adulthood?

RQ2: Is there an association between genetic liability and externalizing behaviors after controlling for the potential effects of parenting?

RQ3: Is there an association between genetic liability and parenting (i.e., support for *r*GE)?

RQ4: Does parenting moderate the association between genetic liability and externalizing behaviors (i.e., support for a GxE)?

All these research questions were tested by estimating a structural model, shown in Figure 1.

Methods

Sample

The National Longitudinal Study of Adolescent to Adult Health (Add Health) is a nationally representative sample comprising 20,745 students in grades 7–12 from a stratified sample of 132 schools. Of those who completed the first in-home interview (1994–1995), 14,738 completed the second interview (1996), 15,197 completed the third interview (2001–2002), and 15,701 completed the fourth interview (2007–2008). The study period covered roughly 14 years between Waves I and IV, ranging from adolescence (12–18 years old) into young adulthood (24–32 years old). During the Wave IV interviews, 15,159 individuals provided samples for genotyping (12,254 agreed to genetic data archiving). Genotyping was conducted using the Illumina Omni1 and Omni2.5 arrays. Genotypic data are available for 9,974 individuals after quality control. For the current analyses, measures from three waves of data were used: Wave 1, Wave 2 (1 year from Wave 1), and Wave 3 (7 years from Wave 1). In this way, the current analyses were limited to unrelated individuals of European ancestry with longitudinal weights from Wave 1 to Wave 3 (N = 4,142), because the discovery sample used to compute polygenic scores was of primarily European ancestries. Imputation was conducted on single nucleotide polymorphisms (SNPs) with a call rate of 98% and a minor allele frequency (MAF) >1% using the Michigan Imputation Server. Individuals of European ancestry were imputed to the Haplotype Reference Consortium (McCarthy et al. 2016).

Measures

Age.—The age of respondents. The average age at Wave 1 was 15.8 years (SD = 1.77).

Sex.—The sex of the participants, coded as 0 = *male*, 1 = *female*.

Family structure.—This was coded as 0 = *not a two-parent family*, 1 = *two-parent family*.

Parental education.—Average of the highest attained education of each parent (when available), which we recoded to a scale ranging from 1 = *never went to school*, to 9 = *professional training beyond a four-year college or university*.

Maternal closeness.—Assessed at Wave 1 with four questions asking adolescents about the relationships with their mothers. This was rated on a 1–5 Likert-type scale, ranging from 1 = *strongly agree* to 5 = *strongly disagree*. For the purposes of the current study, this scale was reverse-coded so that higher values indicated higher closeness. Sample item: "[Do you agree or disagree with the following statement?] Most of the time, your mother is warm and loving toward you". The full list of items is listed in Appendix 1. Reliability of closeness was ω = .85.

Child maltreatment.—Assessed at Wave 3 asking participants to answer 4 retrospective items: two items related to neglect (frequency of times parents left the respondent alone home and Social Security involvement with the family), physical abuse (1 item; rated as the frequency of how many times parents slapped, hit, or kicked the respondent), and sexual abuse (1 item; rated as the frequency of how many times the respondent was sexually touched or forced to engage in sexual behavior or relation) that happened to them before the sixth grade (age of 12). The full list of items is listed in Appendix 1. The reliability of child maltreatment was ω = .55.

Polygenic Score (PGS).—Created using summary statistics from a GWAS of risky behaviors (the first principal component of smoking initiation, alcoholic drinks per week, propensity for automobile speeding, and reported the number of sexual partners) in individuals of European ancestry in the UK Biobank (Linnér et al., 2019). We created PGS using a Bayesian regression and continuous shrinkage method (PRS-CS; Ge et al., 2019). PRS-CS uses information on the correlation among variants that are physically close along the genome (referred to as linkage disequilibrium, or LD) from an external reference panel (1000 Genomes Phase III European subsample; Auton et al., 2015) to estimate the posterior effect sizes for each variant in a given set of GWAS summary statistics. We limited the variants to those in the HapMap3 reference panel (McCarthy et al. 2016) that overlapped between the original GWAS summary statistics, the LD reference panel, and the target sample. Finally, we converted PGS to a Z-score ($M = 0$, $SD = 1$) for interpretation.

Principal components.—To account for potential confounding from population stratification, we included the first 10 ancestry-specific principal components (PCs). Ancestry-specific PCs were estimated using unrelated individuals within each ancestry group (see Braudt & Harris, 2020) for a full description of how ancestry-specific PCs were calculated in the Add Health data).

Alcohol use.—Assessed at Wave 2 and Wave 3. This measure was operationalized using two items asking how many days respondents had drunk in the past 12 months (Never; 1 or 2 days in past 12 months; Once a month or less; 2 or 3 days/month; 1 or 2 days/week; 3–5 days/week; Every day/almost every day) and the number of drinks they had each time they drank (answered as an open-ended item). Days drinking were recoded to reflect drinking days in the past month: Never = 0; 1 or 2 days in the past 12 months = 0.125; once a month or less = 1; 2 or 3 days/month = 2.5; 1 or 2 days/week = 6; 3–5 days/week = 15; everyday/almost every day = 30. The observed variable alcohol use (for descriptive statistics) was then

created by multiplying the number days of drinking in the past month by the number of drinks per occasion and then divided by 30 to obtain average alcohol use per day.

Cannabis use.—Assessed at Wave 2 and Wave 3. Cannabis use was measured by answering the question "During the past 30 days, how many times did you use marijuana?", with an open-ended response, ranging from 0 to 900.

Antisocial behavior.—Assessed at Wave 2 and Wave 3 was assessed using a 10-item self-report measure asking about the frequency of certain delinquent behaviors in the past 12 months. It ranges from 0 (never) to 2 (more than once) and includes items asking about the frequency of using a weapon on someone, using hard drugs, stealing, damaging property, or hurting someone. Sample item: "In the past 12 months, how often did you deliberately damage property that didn't belong to you?" The 10-item measure was tested in a confirmatory factor analysis (CFA) and further changes were made (see Results), resulting in a final 7-item measure. See Appendix 1 for list of items used. The reliability of antisocial behavior at Wave 2 was ω = .69, and ω = .69 at Wave 3.

Plan of Analysis

In the first step, all scales in the current study were tested in a confirmatory factor analysis to determine their psychometric validity. The reliability of scales was assessed using McDonald's omega (ω). Then, descriptive statistics and correlations were computed to assess the relationships between the variables of interest.

In the next step, the effects of the two types of parenting, PGS, and control variables were used as independent variables for the outcomes of interest, namely alcohol use, cannabis use, and antisocial behavior. Closeness and child maltreatment were modeled as unidimensional latent factors. Alcohol use was modeled as a latent factor with the frequency of use and number of drinks as indicators. Antisocial behavior was modeled as a unidimensional latent factor. The residual variances of indicators for alcohol use and antisocial behavior were allowed to covary for the same items from Wave 2 and Wave 3. This main effect model is referred to as Model 1.

To assess the presence of *r*GE, the significance of the correlations between closeness and PGS and child maltreatment and PGS was estimated. To assess the presence of GxE, interaction terms of PGS and parenting variables were created and entered into the model, along with interaction terms of PGS and all covariates and parenting variables with all covariates (Model 2) All models used Taylor series linearization to adjust standard errors for clustering in schools and stratification by regions in the Add Health data collection. All models used longitudinal sample weights to provide nationally representative estimates. All models were assessed in Mplus 8 (Muthén & Muthén, 1998–2017).

Sensitivity analyses.—Substance use and initiation were modeled separately given that 1) there are etiological differences in the initiation and use (Kendler et al. 1999; Verweij et al. 2010), and the genetic liability for risky behaviors could be relevant to both; 2) the outcome variables included a substantial number of non-users, i.e., individuals who never initiated. A dichotomous initiation variable was developed which reflects whether

individuals have used the substance by Wave 3. Instead of controlling for substance use in the previous wave, the initiation variable was entered in the model and allowed to covary with substance use at Wave 3, and then was regressed on all the independent variables just like the substance use. This enabled us to determine whether the genetic propensity, indexed by PGS, was associated with both initiation and substance use or whether the effect from main analyses was driven by either one.

Results

All the variables indicated by latent factors were, with some modifications, found to adequately fit the data (see Appendix 2 for details on these analyses). Descriptive statistics and bivariate correlations of the newly developed scales were assessed (Table 1 and Table 2, respectively). The correlations reported are polychoric correlations that accounted for both nesting and weighting. Closeness at Wave 1 was negatively associated with maltreatment (r = −.22, 95%CI [−.16, −.27]), and negatively associated with all outcome variables at both Wave 2 and Wave 3 (with the exception of antisocial behavior at Wave 3). Closeness was also negatively associated with RISK PGS (r = −.07 [−.02, −.11]), suggesting the existence of rGE effect at the bivariate level. Maltreatment was positively associated with all three outcome variables at both waves. Maltreatment was not significantly associated with RISK PGS. RISK PGS was also significantly associated with alcohol use at Wave 2 and 3 (r = .07 [.03, .12] and r = .07 [.03, .12] respectively), cannabis use at Wave 3 (r = .06 [.04, .07]) and antisocial behavior at Wave 2 (r = .11, [.05, .17]), providing some evidence of a genetic effect on these outcomes. Boys were significantly more likely to report higher levels of alcohol use, cannabis use, and antisocial behavior across all waves. All scales showed good reliability (closeness: ω = .85, antisocial behavior Wave 2: ω = .69, antisocial behavior Wave 3: ω = .69) with the exception of maltreatment, showing an adequate reliability (ω = .55).

In the next step, these variables were used in structural models. Each outcome, measured at Wave 3, was regressed on the same outcome measured at Wave 2, thus testing for developmental changes in the construct over time. Both Wave 2 and Wave 3 outcomes were regressed on closeness, maltreatment, RISK PGS, and control variables (Model 1). Then, interaction terms of closeness * RISK and maltreatment * RISK (and RISK * covariates and parenting * covariates) were added (Model 2).

All the standardized effects are shown in Table 3. The results showed that previous levels of externalizing behaviors showed the relatively strongest associations of all covariates with the externalizing behavior at Wave 3. Among parenting variables, parental maltreatment in childhood was a significantly positively associated with alcohol use at Wave 3 (β = .09 [.01, .17]), antisocial behavior (β = .16 [.09, .22]), and cannabis use (β = .11 [.06, .15]). Higher closeness was found to be significantly associated with lower cannabis use at Wave 3, (β = −.06 [−.01, −.11]), as well as with lower alcohol use (β = −.07 [−.01, −.14]). RISK PGS was found to be positively associated with alcohol use (β = .07 [.02, .11]), cannabis use (β = .05 [.01, .08], but not with antisocial behavior (β = .03 [−.04, .07]). Furthermore, some evidence for rGE was found, as closeness was significantly and negatively associated with RISK PGS, r = .07 [.02, .12]. On the other hand, maltreatment was unrelated to PGS, r = .01, [−.05,

.07]. Finally, when testing for GxE, we found no significant interactions between the showed polygenic score for risky behaviors and either parenting behavior, closeness or maltreatment, on any of the three externalizing measures.

Sensitivity analyses.

For sensitivity analyses, initiation of alcohol and cannabis were tested to understand whether they would affect the findings of the significant positive effect of PGS. The same structural model was tested but used a dichotomous variable of "initiation by Wave 3" instead of Wave 2 measure. The results showed that RISK PGS, in all cases, was significantly positively associated with both the initiation by Wave 3 as well as with use for alcohol and cannabis (controlling for initiation; see Table A1 in Appendix 3 for results).

Discussion

The current study used three waves of Add Health data to test the longitudinal associations of maternal closeness and child maltreatment with externalizing behaviors, measured in young adulthood. Furthermore, the model tested the direct effect of genetic propensity, indicated by a polygenic score for risky behavior, and assessed the shared genetic basis of parenting and risky behavior, as well as their interactive effect. The results provided evidence of important developmental effects of both parental behaviors as well as genetic predispositions for risky behaviors, net the effects of previous levels of externalizing behaviors. We also found evidence for *r*GE of risky behaviors PGS and maternal closeness, measured in adolescence. Finally, we found no evidence of GxE for the PGS and childhood maltreatment or PGS and maternal closeness.

First, the results supported the importance of parenting behaviors affecting later externalizing behavior. In the current study, we focused on two poles of a supposed parenting spectrum – child maltreatment and closeness, hypothesizing that child maltreatment would serve as a risk factor for higher subsequent externalizing behaviors (Dodge et al. 1994; Steinberg et al. 1994, 2006; McKee et al. 2008), while being close to parents would serve as a protective factor (Vieno et al. 2009; MacKenzie et al. 2012; Berkien et al. 2012). The current results largely confirmed these presuppositions, as child maltreatment was found to be associated with higher levels of alcohol use, cannabis use, and antisocial behavior. Importantly, these main effects remained after controlling for previous levels of externalizing behaviors. In this way, child maltreatment was related to changes in antisocial behavior from Wave 2 to Wave 3. These results support the importance of experience with childhood maltreatment leading to later problem behaviors (Manly et al. 2001; Kim and Cicchetti 2009), confirming previous findings of a long-term effect of victimization on later delinquent behavior (Smith and Thornberry 1995; Lansford et al. 2007). Childhood maltreatment creates a pathogenic environment for the child, which oftentimes results in behavioral disinhibition/poor self-regulation, which is one of the most salient risk factors in the developmental cascade to externalizing problems (Smith et al. 2008; Cicchetti and Handley 2019).

Similarly, adolescents who felt closer to their mother reported lower levels of alcohol use and cannabis use in early adulthood. The current study results partially confirm existing

research that showed that children who are close to their parents show lower levels of externalizing behaviors (Gault-Sherman 2012). Higher levels of parental closeness are associated with higher levels of warmth, acceptance, involvement, monitoring, and praise (McKee et al. 2008). Parental closeness promotes emotional and behavioral self-regulation (Hoeve et al. 2009), which may curb children's delinquent behavior. Moreover, children who are close to their parents may be more able to imagine their parental reaction to potential misconduct (Gottfredson and Hirschi 1990; Hoeve et al. 2009; Deutsch et al. 2012) and may therefore avoid engaging in externalizing behaviors to prevent jeopardizing the parent-child relationship.

Furthermore, closeness was found to be negatively correlated with the genetic propensity for risky behavior. This suggests that participants with higher levels of a genetic propensity for risky behaviors experienced lower levels of maternal closeness in adolescence. This might be because a genetic basis for risky behavior, shared between children and their parents, also manifests itself as lower levels of parental closeness (Burt et al. 2005; Horwitz and Neiderhiser 2011). Alternatively, it might mean that children with genetically higher levels of a propensity for risky behaviors elicit less warmth from their parents, maybe due to higher levels of conflict (Kendler and Baker 2007; Horwitz and Neiderhiser 2011; Avinun and Knafo 2014). However, there is a third possibility which might explain this association where individuals with higher genetic propensity for risky behaviors tended to rate closeness to their mothers lower.

The genetic effect, indexed by PGS for risky behaviors, was found to be associated with higher levels of alcohol use as well as cannabis use. This provides further evidence for the shared genetic basis for different types of substance use (Krueger et al. 2002; Dick and Agrawal 2008; Dick et al. 2008). However, no significant main effect was found for antisocial behavior. This was not surprising given that the PGS of risky behavior was originally constructed as a first principal component of four types of risky behaviors, from which two reflect substance use (alcohol and cigarette use; Linnér et al., 2019). It is possible that the antisocial behavior, as measured in the current study, might be a distinctive enough facet of externalizing behaviors that might simply not be captured by the polygenic score, given its still modest explanatory power. Importantly, a genetic effect, indexed by PGS of risky behavior, showed a positive association with alcohol and cannabis use, net the effects of previous levels of use in adolescence, suggesting that genetic effects are equally important not only for use during adolescence but also for developmental changes over time, similar to prior work using twin designs (Long et al. 2017).

Finally, although we found evidence for the main effects of parenting and genetic propensity, the experience with parenting did not condition the effect of genetic risk on externalizing behavior (GxE). Previous studies that demonstrated a GxE effect on adolescent problem behaviors found that this effect was limited to the developmental context of adolescence (Dick et al., 2007; Samek et al., 2015), with the *r*GE accounting for the largest proportion of variance in the parent-child relationship and externalizing behaviors in young adulthood (Samek et al. 2015). Further, many of the previous GxE studies that found significant interaction effects were based on smaller convenience samples using a candidate gene approach, thus increasing the chance that the observed interaction was in fact due to a type

I error (Keller 2014; Dick et al. 2015). A possible explanation for the current null findings might be the fact that there are certain areas of the genome that might be more susceptible to environmental conditioning than others; however, such effect becomes evened out as the creation of the polygenic score averages the genetic effects across the whole genome.

Limitations and Future Directions

These analyses have several important limitations. First, the analyses were limited to individuals of primarily European ancestries for methodological reasons (Martin et al. 2017; Duncan et al. 2019). GWAS with sizable samples of subjects with non-European ancestries are needed for creating more powerful PGS in non-European samples. Ensuring equal representation in genetic studies will be especially important so that everyone can benefit from this type of research and existing health disparities are not exacerbated (Martin et al. 2019). Second, we did not account for *r*GE as a function of the non-transmitted allele (i.e., “genetic nurture”; Kong et al. 2018), which can result in upwardly biased SNP estimates (Bulik-Sullivan et al. 2015; Lee et al. 2018; Balbona et al. 2021). Future research should consider this approach because it 1) reduces the potential for confounding and measurement error as the non-transmitted parental alleles are more likely to be independent of the offspring’s polygenic score, and 2) allows for greater disentanglement of the variance in the parent-child relationship and externalizing behaviors among young adults.

Next, the current study used a retrospective measure of childhood maltreatment, where respondents at Wave 3 were asked to report on experiences that happened before they were 12. Although using a retrospective measure of childhood maltreatment or abuse is a common method (MacMillan et al. 2001; Higgins and McCabe 2001), it might introduce a potential bias as it relies on participants remembering experiences from more than 10 years ago. Relatedly, childhood maltreatment has been found to be multifactorial, with unique genetic and environmental effects depending on the type of maltreatment (e.g., emotional abuse vs sexual abuse; Pezzoli et al. 2019). Modeling it as a unidimensional measure (as we did in the current study due to limited number of indicators) averages across potentially unique associations of different types of maltreatment with the risky behaviors analyzed in this study.

Furthermore, all of the parenting and outcome measures used in the study were based on self-reports. Although using self-reports when reporting antisocial behavior might be more precise than using other sources (Thornberry and Krohn 2000), employing parental reports of parenting in addition to child-reported ones would provide a more rich and complex picture of the child-parent relationship. Relatedly, it is possible that individuals with higher externalizing behaviors would have been less likely to participate in the initial data collection as well as in the follow-ups, thus creating a potential for selection bias in the available sample. We employed maternal closeness as a proxy for positive parenting, as data on the relationship with the father were not available. It is unclear whether we would observe the same pattern of effect if paternal closeness was available.

Lastly, the estimates might be affected by measurement error in the self-reported variables, potentially attenuating the observed associations, as well as possible confounding in the

observed association between the polygenic score and the outcome due to population stratification (Lawson et al. 2020).

Conclusions

The current study used a nationally-representative sample of adolescents to test the longitudinal effect of parenting and genetic predisposition for risky behaviors on externalizing behaviors in young adulthood. We found evidence that childhood maltreatment was positively associated with alcohol use, cannabis use, and antisocial behavior, while lower levels of maternal closeness were associated with higher levels of cannabis and alcohol use. Furthermore, a PGS indexing risky behavior led to higher levels of alcohol and cannabis use, above and beyond other variables. Moreover, we found a significant correlation was parental maternal closeness and PGS, suggesting a shared genetic component. There were no significant interactions between parenting and PGS. These results indicate that parental behaviors in adolescence can have a long-lasting effect on externalizing behaviors, particularly experiences with maltreatment in the family. Furthermore, the current study findings also provide further evidence that these externalizing behaviors have a genetic component, and that they share a genetic basis with the parenting itself, in case of maternal closeness.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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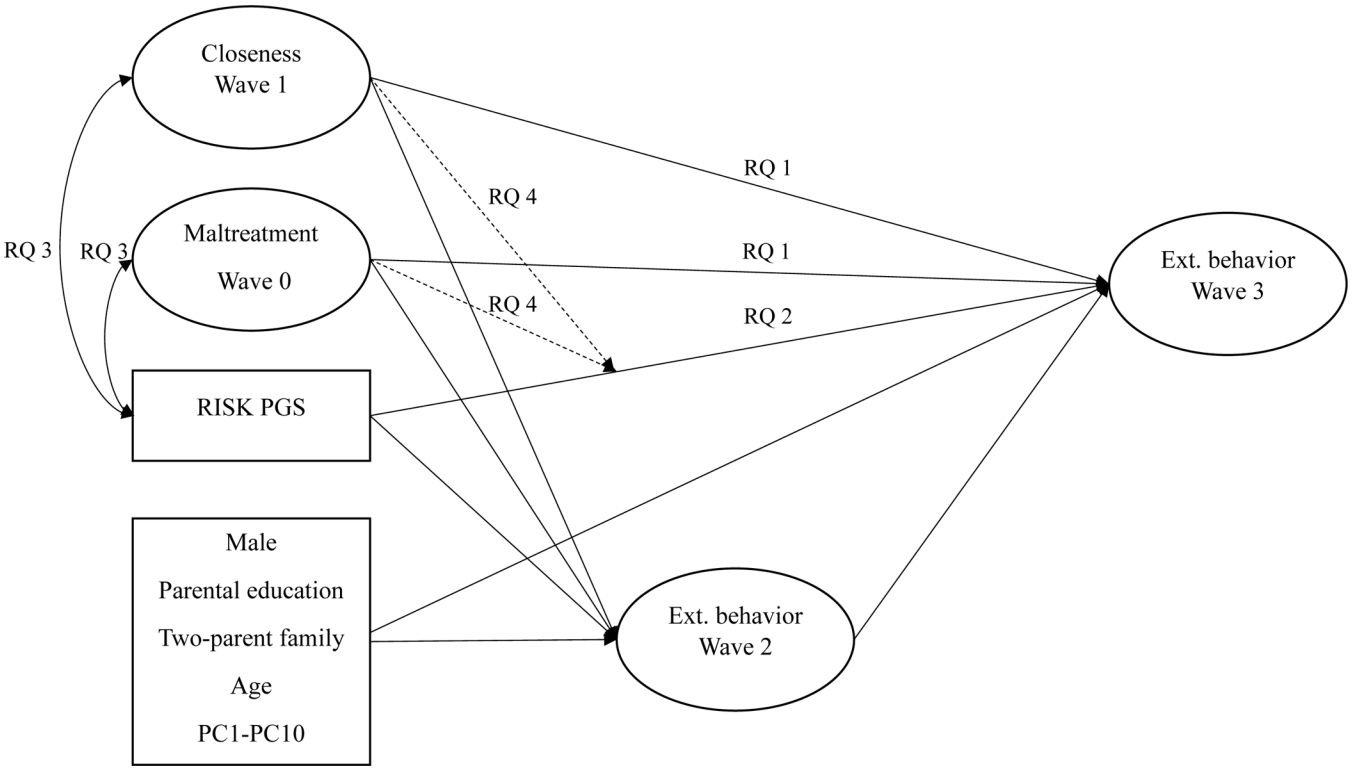

Figure 1.
The overview of the structural model and the research questions tested. Ext. = externalizing. Wave 0 = retrospectively asking about the experience before the age of 12

Table 1

Descriptive Statistics of Study Variables

	Mean/%	*SD*	Min	Max
Closeness W1	4.38	0.62	1.33	5
Child maltreatment W0	0.33	0.41	0	2
Alcohol use W2	0.29	0.99	0	10
Alcohol use W3	0.78	1.40	0	10
Cannabis use W2	2.84	26.62	0	900
Cannabis use W3	4.61	14.66	0	500
Antisocial behavior W2	0.11	0.24	0	2.86
Antisocial behavior W3	0.08	0.20	0	1.86
Male	48.6%	0.50	0	1
RISK PGS	0.00	1.01	−3.51	4.05
Parental education	6.03	1.68	2	9
Two-parent family	63.3%	0.48	0	1
Age W1	15.84	1.77	11.96	21.34

Note. W1 = measured at Wave 1; W2 = measured at Wave 2, W3 = measured at W3, W0 = retrospectively asking about the experience before the age of 12

Table 2

Polychoric Correlation Matrix of Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Closeness W1													
2. Maltreatment W0	−.22***												
3. Alcohol use W2	−.19***	.08**											
4. Alcohol use W3	−.08**	.09*	.34***										
5. Cannabis use W2	−.08*	.06***	.25***	.08***									
6. Cannabis use W3	−.09**	.15***	.14***	.25***	.15*								
7. Antisocial behavior W2	−.22**	.23**	.39***	.28***	.12***	.16***							
8. Antisocial behavior W3	−.07	.34***	.13**	.49***	.05	.20***	.48***						
9. Male	.10***	.05	.09**	.31***	.05***	.11***	.21***	.34***					
10. RISK PGS	−.07**	.03	.07**	.07**	.03	.05**	.11***	.03	−.04*				
11. Parental education	.02	−.12***	−.02	.19***	.01	.01	.01	.14***	.04	−.03			
12. Two-parent family	.09***	−.20***	−.07**	.05	−.07*	−.05*	−.13**	.02	.02	−.08***	.09***		
13. Age W1	−.16***	−.02	.30***	−.03	.09**	−.03	−.03	−.17***	.05*	.00	−.04	−.04	
ω	.85	.55					.69	.69					

Note.

* $p < .05$

** $p < .01$

*** $p < .001$.

ω = internal reliability assessed by McDonald's omega.

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Table 3

Standardized Effects from Structural Models for Each Externalizing Behavior

	Alcohol use				Cannabis use		
		beta	*95% CI*			*beta*	*95% CI*
Model 1	Male	0.31	[.24, .34]	Model 1	Male	0.11	[.07, .15]
	Parental education	0.19	[.15, .26]		Parental education	0.02	[−.02, .06]
	Two-parent family	0.06	[.01, .15]		Two-parent family	−0.02	[−.05, .02]
	Age	−0.11	[−.18, −.03]		Age	−0.05	[−.08, −.01]
	Alcohol use W2	0.26	[.17, .36]		Cannabis use W2	0.13	[.03, .23]
	RISK PGS	0.07	[.02, .11]		RISK PGS	0.05	[.01, .08]
	Closeness	−0.07	[−.01, −.14]		Closeness	−0.06	[−.01, −.11]
	Maltreatment	0.09	[.01, .17]		Maltreatment	0.11	[.06, .15]
Model 2	Closeness*PGS interaction	−0.04	[−.06, .02]	Model 2	Closeness*PGS interaction	−0.02	[−.06, .03]
	Maltreatment*PGS interaction	−0.05	[−.11, .04]		Maltreatment*PGS interaction	0.04	[−.02, .08]
	Antisocial behavior						
		beta	*95% CI*				
Model 1	Male	0.18	[.15, .22]				
	Parental education	0.12	[.04, .15]				
	Two-parent family	0.07	[−.01, .09]				
	Age	−0.10	[−.16, −.07]				
	Antisocial behavior Wave 2	0.22	[.10, .34]				
	RISK PGS	0.03	[−.04, .07]				
	Closeness	−0.04	[−.11,.03]				
	Maltreatment	0.16	[.09, .22]				
Model 2	Closeness*PGS interaction	−0.02	[−.06, .02]				
	Maltreatment*PGS interaction	−0.04	[−.11, .04]				

Note. Model 1 included main effects. Model 2 = Model 1 + interactions terms of PGS and closeness/maltreatment, as well as interaction terms of all PGS with all covariates, and closeness/maltreatment with all covariates (not shown). Model 1 and Model 2 included principal components to control for population stratification (effects not shown).