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Regular Article

Childhood maltreatment affects adolescent sensitivity to parenting and close friendships in predicting growth in externalizing behavior

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Abstract

Childhood maltreatment robustly predicts adolescent externalizing behaviors (EB; e.g., violence, delinquency, substance use) and may crystalize patterns of EB by influencing sensitivity to the social environment (e.g., parenting, friendships). In a nationally representative sample of 9,421 adolescents, we modeled latent growth curves of EB from age 13 to 32 years. Next, we explored whether maltreated youth differed from nonmaltreated youth in their sensitivity to parental closeness, friendship involvement, and polymorphisms from dopamine genes linked to EB (dopamine receptors D2 and D4, dopamine transporter). Overall, maltreated youth had significantly higher levels of EB across adolescence and adulthood; however, maltreated and nonmaltreated youth showed similar patterns of EB change over time: violent behavior decreased in adolescence before stabilizing in adulthood, whereas nonviolent delinquency and substance use increased in adolescence before decreasing in the transition to adulthood. Maltreatment reduced sensitivity to parental closeness and friendship involvement, although patterns varied based on type of EB outcome. Finally, none of the environmental effects on EB were significantly moderated by the dopamine polygenic risk score after accounting for multiple testing. These findings underline the enduring effects of early maltreatment and implicate that maltreatment may contribute to long-term risk for EB by influencing children's sensitivity to social relationship factors in adolescence.

Key words: adolescent development, childhood maltreatment, environmental sensitivity, externalizing behavior

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Externalizing behavior (EB), including violence, nonviolent delinquency (e.g., stealing), and substance abuse, is one of the costliest public health problems in North America (Foster & Jones, 2005; Welsh et al., 2008). Individual differences in EB are highly sensitive to development, including a precipitous increase during adolescence (Moffitt, 1993). Ranging from daily interactions with parents and friends (Sentse & Laird, 2010) to severe stressors such as maltreatment (Jaffee, Caspi, Moffitt, & Taylor, 2004; Oshri, Rogosch, Burnette, & Cicchetti, 2011), social experiences affect EB trajectories, which are further affected by genetic variation (Bakermans-Kranenburg & van IJzendoorn, 2011). Transient and mild EB may be normative in adolescence, reflecting identity formation and pursuit of social status (Brezina & Piquero, 2007; Englund et al., 2013; Roisman, Monahan, Campbell, Steinberg, & Cauffman, 2010); however, EB persists for a sizable minority of youth (Evans, Simons, & Simons, 2016; Moffitt, 1993), predicting antisocial personality disorder, alcohol/substance use disorders, and economic instability (Brown et al., 2008; Maughan et al., 2004; Moffitt & Caspi, 2001). Given its clinical and public health significance, identifying modifiable predictors of EB growth and desistance from adolescence to adulthood is necessary to design effective prevention programs.

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Maltreatment and EB

One of the most consistent and robust predictors of EB is childhood maltreatment (Jaffee et al., 2004; Kerig & Becker, 2015), including physical abuse, sexual abuse, and neglect. Youth with maltreatment histories are at elevated risk for adolescent violence, delinquency, and substance use (Oshri et al., 2011), particularly in combination with other family-level stressors such as domestic violence (Moylan et al., 2010). Additionally, early maltreatment has long-term sequelae: among 574 youth followed prospectively, youth with abuse histories were almost twice as likely than nonabused youth to be arrested 17 years later (Lansford et al., 2007).

Maltreatment likely has enduring effects on EB by influencing the biological processes involved in responding to the social environment (Bender, 2010; Egeland, Yates, Appleyard, & Dulmen, 2002; Rogosch, Oshri, & Cicchetti, 2010). Drawing from evolutionary theories, exposure to stressful and chaotic early environments may shift organisms toward heightened biological sensitivity to the environment, which may calibrate their biological systems (e.g., activation thresholds, stress reactivity) to better match their ecological environment (Boyce & Ellis, 2005; Ellis & Boyce, 2008). For example, early exposure to a dangerous/unpredictable environment (e.g., physical abuse) may heighten vigilance to threat and increase aggressive behaviors to thwart

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salient threats from the environment (Boyce, 2007; Lee & Hoaken, 2007). In line with this theoretical model, some studies of infants and children found that maltreatment and other early life stressors heightened sensitivity to environmental stimuli, including amplified neural activity in response to angry faces (Curtis & Cicchetti, 2011, 2013) and cortisol hyperreactivity (Essex, Klein, Cho, & Kalin, 2002; Evans & Kim, 2007). In contrast, other studies have reported an opposite pattern in which high levels of early life stress predicted hyporeactivity (e.g., lower cortisol responses) to stress (Gustafsson, Anckarsäter, Lichtenstein, Nelson, & Gustafsson, 2010). Emerging theories (e.g., adaptive calibration model; Del Giudice, Ellis, & Shirtcliff, 2011) propose that although exposure to moderately threatening environments may heighten sensitivity to stress, exposure to severe and traumatic stress (e.g., chronic maltreatment) may ultimately "blunt" the stress response system over time (Del Giudice et al., 2011). Developmental timing may also play a key role, given evidence from longitudinal studies that maltreatment heightens physiological responses to stress in childhood, but may ultimately lead to blunted profiles of stress responsivity in adolescence and adulthood (Bosch et al., 2012; Ellis, Oldehinkel, & Nederhof, 2017; Tarullo & Gunnar, 2006).

In the context of adolescent EB, a blunted physiological stress response may critically inhibit developmentally normative patterns of social learning and responsivity. Indeed, maltreatment interferes with adaptive processes such as attachment and responsiveness to social relationships (Cicchetti & Banny, 2014; Rogosch et al., 2010) and thus may blunt sensitivity to positive social relationships or effective socially based interventions for EB. For example, a longitudinal study of high-risk families found that maltreated children developed less close relationships with their parents, which subsequently predicted EB in elementary school and later adolescent conduct problems (Egeland et al., 2002); thus, maltreatment may contribute to adolescent EB by compromising children's interpersonal relationships. Investigating this process has important implications from a treatment perspective, given that many interventions for EB assume that changes in the social environment, including at home (e.g., parent training) and school (e.g., bullying), are sufficient to reduce EB. Clarifying which maltreated youth may be differentially influenced by their environments in adolescence is necessary to design appropriate interventions for maltreated adolescents at risk for persistent EB.

Sensitivity to parental closeness and peer relationships

A parent-child relationship characterized by emotional closeness, warmth/support, and communication is a potent protective factor in the development of severe EB (Chassin et al., 2005; Shaw, Hyde, & Brennan, 2012), including promoting resilient outcomes among maltreated youth (Afifi & Macmillan, 2011). Especially in early adolescence, parental closeness predicts lower adolescent EB, above and beyond parental control (e.g., discipline, monitoring) (Chassin et al., 2005; Hill, Hawkins, Catalano, Abbott, & Guo, 2005; Tilson, McBride, Lipkus, & Catalano, 2004). Parent training is therefore commonly integrated into EB prevention and intervention programs, particularly in childhood; however, not all youth benefit equally from parenting interventions. Compared with nonmaltreated youth, adolescents with maltreatment history have substantially higher risk for exhibiting severe EB, which is linked with treatment resistance (Masi et al., 2011). In particular, during the sensitive period of early childhood, maltreatment can disrupt important socioemotional developmental milestones, such as attachment formation with caregivers (Lowell, Renk, & Adgate, 2014). Childhood maltreatment may thus influence patterns of EB development by affecting later sensitivity to protective interpersonal factors such as parental closeness in adolescence.

Beyond parent-child relationships, peer relationships (e.g., friendships) become increasingly relevant during the transition into adolescence (Laible, Carlo, & Raffaelli, 2000). Multiple peer factors are linked to EB, including deviant peer affiliation (Hou et al., 2013; Wang & Dishion, 2012), peer acceptance and social status (Menting, van Lier, & Koot, 2011), and dyadic friendship support and conflict (Sentse & Laird, 2010). Peers play a critical socialization role during the malleable adolescence period, particularly during early adolescence (Steinberg & Monahan, 2007), when youth experience increased autonomy and opportunities to socialize with peers, paralleled by rapid neurobiological changes that shape the development of emotion and self-regulatory behaviors (Steinberg, 2008). During this sensitive period of learning, friendships characterized by frequent hostility or unresolved conflict can limit learning of prosocial skills and promote heightened sensitivity to rejection (Patterson, Reid, & Dishion, 1992; Zimmer-Gembeck, 2016) that lead to increased EB, whereas high friendship closeness and warmth predicts lower EB (Sentse & Laird, 2010; You & Bellmore, 2012). Few studies have tested whether friendship effects on EB differ based on maltreatment history, although maltreatment contributes to rejection sensitivity (Luterek, Harb, Heimberg, & Marx, 2004) and hostile attribution bias (Kay & Green, 2016), which positively predict reactively aggressive behaviors (Ayduk, Gyurak, & Luerssen, 2008; Dodge et al., 2015; Romero-Canyas, Downey, Berenson, Ayduk, & Kang, 2010). In contrast, one study reported that maltreated children exhibited decreased neural responses to social rejection cues (Puetz et al., 2016) and thus may show a blunted sensitivity to peer socialization effects. Given the central role of peers in shaping behavioral development, it is a priority to understand how these processes influencing adolescent EB may differ between maltreated and nonmaltreated youth.

Genetic influences on EB

Finally, genetic variation can also influence sensitivity to parenting and peer environments, although few studies have explored whether these patterns differ between maltreated and nonmaltreated youth. Whereas early Gene × Environment interaction (G×E) studies assumed a dual-risk model whereby genotypes increased vulnerability to adversity (e.g., abuse, neglect, harsh parenting) (Bakermans-Kranenburg & van IJzendoorn, 2006; Caspi et al., 2002), the differential susceptibility or biological sensitivity to context theory proposed that genetic "risk" may actually confer heightened susceptibility to the social environment, for better and for worse (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). That is, genotypes once thought to singularly increase vulnerability to early adversity may also increase sensitivity to environmental enrichment (Belsky, Bakermans-Kranenburg, & IJzendoorn, 2007).

Candidate genes that influence dopaminergic efficiency and transmission have been linked to EB because of their role in reward sensitivity (Janssens et al., 2015) and behavioral inhibition/impulsivity (Cornish et al., 2005; Eisenberg et al., 2007). The present study focused on three polymorphisms that have demonstrated previous links with functional changes in dopamine transmission linked to EB and were identified in a meta-analysis of differential susceptibility for EB (Bakermans-Kranenburg & van IJzendoorn, 2011): the dopamine receptor D4 (DRD4), the

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dopamine receptor D2 (DRD2), and the dopamine transporter (DAT1) gene (Belsky & Beaver, 2011; Brody, Yu, & Beach, 2015; Chester et al., 2015; Yu et al., 2014). Together, these dopamine variants cumulatively affect motivational sensitivity to environmental stimuli by affecting neural structures mediating reward, motivation, and learning (Feder, Nestler, & Charney, 2009; Wise, 2004). Youth with genotypes associated with lower dopaminergic efficiency, including the 7R allele of DRD4, the 10R allele of DAT1, and the A1 allele of DRD2, showed heightened vulnerability to early stress (e.g., maltreatment) compared with youth without these "plasticity alleles," but they also showed heightened sensitivity to social enrichment (Boardman et al., 2014; Brody et al., 2015), such as positive parenting or peer relationships. Meta-analyses of cross-sectional and intervention studies suggest that differential sensitivity to the environment based on genetic variation is not only plausible, but also may improve the precision of interventions (Bakermans-Kranenburg & van IJzendoorn, 2011, 2015).

Although theorized to lead to enduring developmental changes (Ellis et al., 2011), most studies have examined differential susceptibility in young children; thus, it is unclear how these effects may change over time in adolescence. Studies of adolescent differential susceptibility are more inconsistent compared with studies of early development: some evidence suggests that differential susceptibility to positive and negative parenting extends to adolescence and even young adulthood (Chhangur et al., 2015; Nikitopoulos et al., 2014), whereas other studies have observed differential susceptibility in childhood and preadolescence, but not in later adolescence (Zhang et al., 2015). Examining differential susceptibility in adolescence is complicated because models must consider salient environmental experiences over time, including the potential impact of previous adversity. Given that maltreatment itself may already influence sensitivity to parenting and peer influences through Early Stress × Later Environment interactions, it is unclear if patterns of genetic sensitivity to parenting and peer factors would be similar for maltreated versus nonmaltreated youth. One study of young children found that genetic variation in DRD4 and the serotonin transporter, 5-HTTLPR, predicted attachment disorganization for nonmaltreated children, whereas it had minimal effect on attachment organization for maltreated children (Cicchetti, Rogosch, & Toth, 2011). Although this study did not examine whether these effects influenced later outcomes in childhood or adolescence, it suggests that the impact of genetic variation on relational processes may differ based on early exposure to maltreatment.

Study aims

To investigate how childhood maltreatment (i.e., physical/sexual abuse, neglect) influences adolescent sensitivity to peer and parenting factors, the present study tested the following questions in a nationally representative longitudinal sample of adolescents: (1) Compared with nonmaltreated youth, do youth with a history of maltreatment have more persistent developmental trajectories of violence, delinquency, and substance use from adolescence to adulthood? (2) Does childhood maltreatment influence later sensitivity to parenting (ranging from low to high closeness) and friendship (ranging from low to high involvement with a close friend) on adolescent EB? If so, does early maltreatment blunt or heighten later sensitivity to the social environment? (3) Finally, if maltreatment affects sensitivity to later parenting and peer factors, are these patterns of sensitivity particularly pronounced for youth carrying a greater number of dopaminergic "plasticity genotypes" identified in previous studies (Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Beaver, 2011; Brody et al., 2015)?

Method

Participants

This study used data from the National Longitudinal Study of Adolescent Health (Add Health), an ongoing nationally representative study of US adolescents (Harris et al., 2008). Details of the study design are available at http://www.cpc.unc.edu/projects/addhealth. In 1994, 80 high schools and 52 middle schools were selected using a stratified cluster design. A subsample of individuals participated in in-home interviews at Wave 1 in 1994–1995 (n = 20,745, grades 7–12, ages 11–19 years). These participants were interviewed again a year later at Wave 2 in 1996 (n = 14,738, ages 13–20), another 6–7 years later at Wave 3 in 2001–2002 (n = 15,197, ages 18–28), and another 7 years later at Wave 4 in 2008 (n = 15,701, ages 25–34). Saliva samples were obtained at Wave 4 for genotyping. Previous attrition analyses reported by the developers of Add Health indicated differential attrition by gender and race (higher response rate for white and female respondents) (Harris, 2013); however, nonresponse bias analyses indicated that the Wave IV sample adequately represents the original Wave 1 sample after accounting for sampling weights (Brownstein et al., 2011). The present study included participants with available sampling weight data in all four waves (n = 9,421). Approximately 54.6% of adolescents included in analyses were female, and participants were racially and ethnically diverse (64.9% identifying as white, 21.5% black, 3.6% Native American, 7.1% Asian, 8.5% "other" race, and 15.4% Hispanic ethnicity). Participants in the present study did not differ in their rates of childhood maltreatment compared with the original Add Health participants not included in our models (χ^2 = .06, p = .80). Compared with nonparticipants, participants in this study had higher perceived parental closeness (t = –3.10, p = .002) and lower friendship closeness (t = 5.16, p < .001). Participants showed comparable levels of nonviolent delinquency at Wave 1 (t = 1.392, p = .16), but lower violence (t = 3.38, p = .001) and substance use (t = 7.161, p < .001) compared with nonparticipants.

Measures

Violence and delinquency

Violent behavior and nonviolent delinquency were assessed during a structured home interview with the youth at Waves 1, 2, 3, and 4. Violence items asked about the frequency or presence of physical violence in the past 12 months (e.g., In the past 12 months, how often did you hurt someone badly enough to need bandages or care from a doctor or nurse?, During the past 12 months, how often did this happen: you shot or stabbed someone?). Delinquency items asked about the frequency of nonviolent delinquent behaviors (e.g., In the past 12 months, how often did you steal something worth more than $50?, In the past 12 months, how often did you go into a house or building to steal something?). Given inconsistent scaling (e.g., dichotomous vs. frequency counts) and inclusion of some items (but not others) across time, we focused on the five violent and five nonviolent delinquency items that were administered at all four waves and dichotomized them (0 = absence of behavior, 1 = presence of behavior) for eventual summing for separate scales for violence

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(α = .60 at Wave 1) and nonviolent delinquency scales (α = .64 at Wave 1). Previous studies of Add Health have confirmed independence of similar violent and nonviolent delinquency factors (Barnes, Beaver, & Miller, 2010) and demonstrated predictive validity in their associations with expected constructs such as gang membership, substance use, and neighborhood disadvantage (Barnes et al., 2010; Barnes & Jacobs, 2013; Marcus & Jamison, 2013). The present study used the sum of items at each wave to model separate trajectories of violence and nonviolent delinquency.

Substance use

The structured adolescent home interview at Waves 1, 2, 3, and 4 also included questions about the frequency of using various substances in the past 30 days, including tobacco, marijuana, and other illicit drugs (e.g., cocaine, LSD, PCP, ecstasy, mushrooms, inhalants, heroin). In addition, participants were asked about binge drinking in the past year (i.e., Over the past 12 months, on how many days did you drink 5 or more drinks in a row?), and responded based on the following options: Never, 1–2 days, once a month or less, 2–3 days a month, 1–2 days a month, 3–5 days/month, or every day/almost every day. Tobacco, marijuana, and other illicit drugs were recoded dichotomously (0 = not used in past month, 1 = used once or more in past month). To be consistent with the 30-day scale, binge drinking was recoded dichotomously such that 0 = once a month or less (3–12 times) and 1 = 2–3 days/month or more. These four dichotomous substance variables (i.e., binge drinking, tobacco, marijuana, other illicit drugs) were then summed at each wave to model change in the number of substances used in the past month across adolescence and adulthood. Similar polysubstance variables in Add Health demonstrated significant and directionally consistent associations with related constructs (e.g., deviant peer affiliation, alcohol problems) (Vaughn, Beaver, DeLisi, Perron, & Schelbe, 2009).

Maltreatment

Childhood maltreatment was retrospectively assessed at Wave 3. During the in-home interview, participants reported the frequency of exposure to maltreatment from a parent or adult caregiver before age 12, including physical abuse (i.e., How often had your parents or other adult caregivers slapped, hit, or kicked you?), sexual abuse (i.e., How often had one of your parents or other adult caregivers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?), and neglect (i.e., How often had your parents or other adult caregivers not taken care of your basic needs, such as keeping you clean or providing food or clothing?). These items were designed to be consistent with the standard definition of maltreatment used by the US Centers for Disease Control and Prevention (Leeb, Paulozzi, Melanson, Simon, & Arias, 2008). Items were recoded to create an overall maltreatment variable in which participants were scored positive in history of childhood maltreatment if physical abuse, sexual abuse, or neglect were endorsed as occurring more than once (Haberstick et al., 2005), given that repeated maltreatment exposure is associated with increased risk of severity and enduring psychosocial consequences (Gilbert et al., 2009; Jonson-Reid, Khol, & Drake, 2012). Among participants included the present study, 26.2% reported more than one episode of maltreatment. Previous studies showed the maltreatment variable from Add Health to demonstrate predictive validity with a range of expected outcomes such as youth violence, young adult intimate partner violence, poor health, depression, binge drinking, and substance use (Fang & Corso, 2007; Hussey, Chang, & Kotch, 2006).

Parental closeness

An in-home structured interview at Wave 1 asked youth to report on various dimensions of parenting behavior. For youth living in a two-parent household, responses regarding maternal closeness were prioritized to facilitate comparisons with the majority of previous studies on parenting and differential susceptibility. The parental closeness index (seven items; α = .85) measures perceived emotional warmth, closeness, and communication between parent and child (e.g., How close do you feel to your parent?, How much do you think your parent cares about you?, Most of the time, your mother is warm and loving toward you). Items were measured on a 5-point Likert scale (1 = not at all, 5 = very much) and summed to form a total parental closeness score. This scale demonstrated predictive validity with multiple offspring outcomes including self-regulation, self-esteem, depression, and juvenile delinquency (Belsky & Beaver, 2011; Bynum & Kotchick, 2006; Li, Berk, & Lee, 2013).

Friend involvement

The structured home interview at Wave 1 asked participants to name their closest female and male friend. Participants then answered whether they did (1 = yes) or did not (0 = no) engage in the following activities with their friend in the past seven days: went to friend's house, met the friend after school to hang out or go somewhere, spent time with the friend during the past weekend, talked with the friend about a problem, and talked to the friend on the telephone. Each friendship activity was scored as positively endorsed if the participant reported engaging in the activity with either their closest male or female friend. Items were then summed to form a total friend involvement scale (five items; α = .64), with higher scores indicating more interaction with close friends.

Dopamine gene index

Saliva samples were collected from participants in Wave 4, and genomic DNA was isolated from buccal cells using standard methods to genotype for DRD4, DAT1, and DRD2. First, the 48 bp VNTR polymorphism located on chromosome 11p15.5 in exon 3 of the DRD4 was genotyped, which yields loci of 2 to 11 repeats. DRD4 genotypes including the most common polymorphisms (4- and 7-repeat) include 7/7, 7/4, and 4/4. Second, the 40-bp VNTR polymorphism in the 3′ untranslated region of exon 15 of DAT1 was genotyped, yielding 9-repeat (440 bp) and 10-repeat (480 bp) polymorphisms to form the following genotypes: 9/9, 9/10, and 10/10. Third, the TaqIA (rs1800497) polymorphism located on chromosome 11q22.3 of DRD2 was genotyped, yielding A1and A2 alleles with the following genotypes: A1/A1, A1/A2, and A2/A2. Hardy–Weinberg equilibrium was tested for each allele genotype in race/ethnicity specific strata; deviations from Hardy–Weinberg equilibrium (at α = ∼.05) were identified among black participants for DRD4, but no deviations were observed for DRD2 or DAT1 (Smolen et al., 2013); thus, race-ethnicity was controlled as a covariate in all models. Following previous strategies (Belsky & Beaver, 2011), we recoded each polymorphism such that the 7R/7R and 7R/4R (vs. 4R/4R) genotypes of DRD4, the 10/10R (vs. 9/10 or 9/9) genotype of DAT1, and the A1/A1 or A1/A2 (vs. A2/A2) genotypes of DRD2 were identified as "plasticity genotypes." Each polymorphism was assigned a point if the participant had a plasticity

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genotype. These values were summed to form a cumulative index of dopamine genotypes ranging from 0 to 3, with higher scores representing more genetic plasticity.

Data analytic plan

All analyses were conducted using Mplus, version 6.12. Appropriate survey weights and design effects were included to account for potential sample and population differences, selection probabilities, and differential rates of nonresponse and attrition. This also helps compensate for potential chance fluctuations of the sample from the broader population, which increases generalizability of the results to the general US population. Given the heightened susceptibility of candidate G×E studies to Type 1 error (Duncan & Keller, 2011) and inclusion of three separate outcomes (violence, nonviolent delinquency, substance use), a false discovery rate (FDR) adjustment (Benjamini & Hochberg, 1995) was applied to all model parameters ($Q = .10$) to evaluate significance after correcting for multiple testing (McDonald, 2014; Cribbie, 2007).

Unconditional latent growth model

Latent growth modeling (LGM) (also called latent growth curve analysis) was used to model change in violence, nonviolent delinquency, and substance use across ages 13–32. LGM is a longitudinal estimate of growth over time based on a structural equation modeling framework. LGM estimates the mean parameter level at a given point in time (i.e., intercept), the rate of increase/decrease over time (i.e., linear slope), and the rate of change of the increase/decrease (i.e., latent quadratic trend). Given that each wave of assessment in Add Health contained significant variability in chronological age (e.g., ranging from 11 to 19 in Wave 1), an accelerated longitudinal design was used, in which age (vs. wave) represented the unit of time (Bollen & Curran, 2006; Duncan, Duncan, & Hops, 1996). Thus, LGM modeled continuous change in violence and nonviolent delinquency and substance use from age 13 (the youngest participant at Wave 1 with sufficient data and representing the beginning of adolescence) to age 32 (oldest participant at Wave 3 with sufficient data). As shown in Table 1, this approach results in substantial missing data that are "missing by design" (Muthén & Muthén, 2007), which is considered "missing completely at random" (Little & Rubin, 1987). Mplus uses a maximum likelihood approach to appropriately account for this pattern of missingness (Duncan, Duncan, Stycker, & Chaumeton, 2007), which is significantly advantageous to older methods such as listwise deletion or imputation.

First, to determine if changes in violence, delinquency, and substance use are best captured by linear or quadratic growth, a series of unconditional models (i.e., without predictors) modeled each outcome from age 13 to 32. Zero-inflated Poisson modeling was used given the skewed count data represented by many youth not engaging in any EB (Liu, 2007). The Satorra–Bentler χ^2 difference test (Satorra & Bentler, 2001) evaluated whether a model including both a linear and quadratic function fit better than the model including only linear slope. Model fit was further evaluated by comparing the Akaike information criterion (AIC), Bayesian information criterion (BIC), and the sample-adjusted BIC (lower values indicate better fit for all three indices).

Conditional LGMs

After establishing the latent growth components (intercept, linear slope, quadratic function) appropriate for modeling change in

Table 1. Accelerated longitudinal design sample size (N) recoded by age and maltreatment status

	Age (years)																				
	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32
Total (*N*)	864	2,125	2,886	3,492	3,815	2,910	1,910	1,409	1,561	1,805	1,895	1,175	445	864	1,203	1,500	1,697	1,739	878	233
Nonmaltreated	661	1,599	2,146	2,580	2,794	2,129	1,413	1,057	1,141	1,347	1,373	872	326	654	897	1,125	1,255	1,272	643	169
Maltreated	203	526	740	912	1021	781	497	352	420	458	522	303	119	210	306	375	442	467	235	64

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each type of EB, the outcomes were regressed on parental closeness and friendship, controlling for sex and race-ethnicity (dummy-coded with white as the comparison group). Next, the main effect of dopaminergic gene index and its two-way interaction terms with parental closeness and friendship were added to the model. Separate models were conducted to predict violence, nonviolent delinquency, and substance use outcomes.

Multiple-group comparisons

For all unconditional and conditional models, a multigroup framework was used to compare adolescents with versus without a history of maltreatment. Multiple-group LGMs simultaneously evaluate developmental hypotheses in multiple groups (Duncan, Duncan, & Strycker, 2013). Specifically, maltreatment group differences were tested on (1) the intercept, slope, and quadratic function of each EB outcome; (2) independent associations of parental closeness and friendship involvement; and (3) interactive associations between dopaminergic genes and the environmental variables (parental closeness, friendship) on each outcome. Significance of group differences was determined by testing for equality of parameters between maltreated and nonmaltreated youth using the Wald χ^2 test, correcting for multiple testing as noted previously.

Results

Preliminary analyses

Table 2 shows descriptive statistics and bivariate correlations among the primary study variables. As expected, maltreatment history was significantly positively correlated with violence, nonviolent delinquency, and substance use at all waves. Parental closeness was inversely correlated with maltreatment and showed some weak negative correlations with EB outcomes. Parental closeness was inversely associated with friendship involvement, which was positively correlated with violence and nonviolent delinquency at the first two waves and with substance use across all waves, but negatively correlated with nonviolent delinquency at Wave 3. These different patterns of correlations between parental closeness and friendship involvement highlight the need to consider critical differences between parenting and friendship effects during this developmental period. Finally, polygenic dopaminergic risk was not correlated with the environmental variables (i.e., maltreatment, parental closeness, friendship involvement; all $p > .05$), reducing concerns about G×E correlations.

Latent growth of violence

Modeling growth in violence from ages 13–32, all fit indices (AIC, BIC, and sample-adjusted BIC) and the Satorra–Bentler χ^2 test indicated that including a nonlinear (quadratic) term in the unconditional model significantly improved model fit compared with the linear (slope only) model ($\Delta\chi^2$ (2) = 71.83, $p < .01$). Overall, violence significantly decreased linearly across adolescence (slope $b = -.071$, $SE = .018$, $p < .001$), although it showed a marginally significant quadratic trend such that the decrease in violent behavior slowed down to stabilize in adulthood (quadratic $b = .002$, $SE = .001$, $p = .068$). The multigroup model comparing maltreated with nonmaltreated youth found that maltreated youth engaged in significantly more violent behavior at age 13 (intercept) compared with nonmaltreated youth, but

Table 2. Descriptive statistics and bivariate correlations among primary study variables

Variable	*M*	*SD*	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Maltreatment (*n* and %)	2,319	26.20	—															
2. Parental closeness	30.41	4.24	-.12**	—														
3. Friendship involvement	3.49	1.46	-.01	-.06**	—													
4. Polygenic risk	1.50	.94	<.01	-.01	-.01	—												
5. Wave 1 violence	.47	.88	.10**	-.09**	.06**	.02*	—											
6. Wave 2 violence	.33	.76	.09**	-.06**	.04**	.04**	.50**	—										
7. Wave 3 violence	.18	.54	.10**	<.01	.01	.01	.24**	.27**	—									
8. Wave 4 violence	.10	.43	.05**	.01	<.01	−.01	.16**	.16**	.23**	—								
9. Wave 1 nonviolent delinquency	.55	.98	.11**	-.17**	.07**	<.01	.45**	.29**	.15**	.08**	—							
10. Wave 2 nonviolent delinquency	.44	.89	.09**	-.11**	.05**	-.01	.28**	.43**	.17**	.09**	.48**	—						
11. Wave 3 nonviolent delinquency	.30	.74	.12**	-.02	-.02*	-.01	.11**	.11**	.33**	.11**	.22**	.24**	—					
12. Wave 4 nonviolent delinquency	.15	.50	.09**	-.02	<.01	-.02	.10**	.13**	.18**	.29**	.17**	.18**	.28**	—				
13. Wave 1 substance use	.55	.93	.08**	-.19**	.19**	-.01	.31**	.24**	.11**	.08**	.41**	.26**	.07**	.08**	—			
14. Wave 2 substance use	.69	.97	.06**	-.16**	.19**	-.02*	.27**	.31**	.14**	.09**	.35**	.37**	.12**	.12**	.66**	—		
15. Wave 3 substance use	.87	1.06	.09**	-.07**	.09**	-.03*	.15**	.16**	.25**	.10**	.24**	.25**	.34**	.20**	.35**	.42**	—	
16. Wave 4 substance use	.78	.96	.07**	-.05**	.07**	-.02	.18**	.16**	.19**	.14**	.22**	.22**	.24**	.28**	.30**	.38**	.59**	—

Note: *$p < .05$; **$p < .01$.

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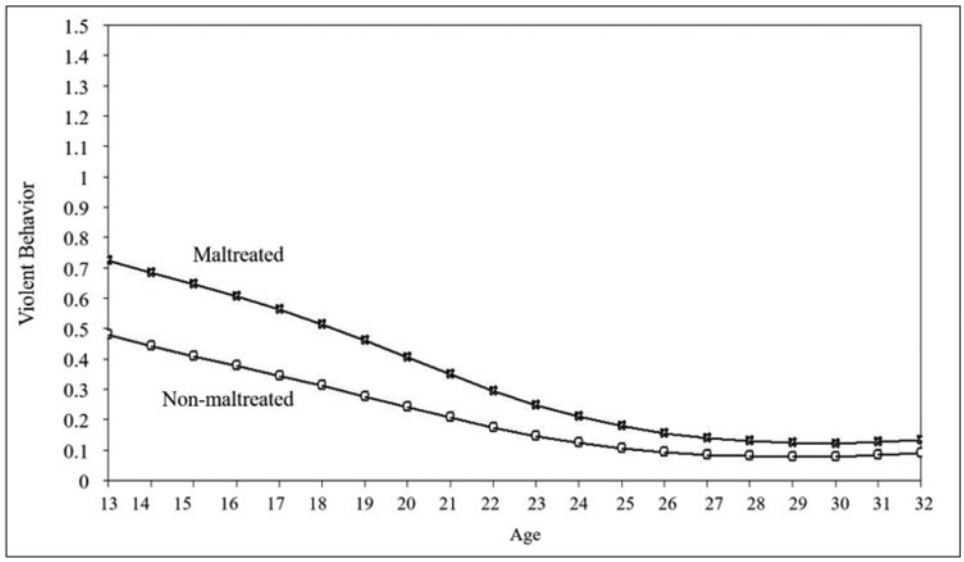

	Non-Maltreated		Maltreated		Wald Test	
	B (SE)	*p*	B (SE)	*p*	Value	*p*
Intercept	-1.413 (.035)	<.001*	-1.004 (.029)	<.001*	89.448	<.001*
Slope	-.078 (.013)	<.001*	-.052 (.011)	<.001*	2.837	.092
Quadratic	.003 (.001)	.002*	.001 (.001)	<.136	2.020	.155

Note. * = significant effect after applying false discovery rate correction for multiple testing.

Figure 1. Latent growth curves of violent behavior from age 13 to 32 for maltreated and nonmaltreated youth.

their patterns of linear and quadratic change across time did not significantly differ (Figure 1).

Parenting and friendship

Controlling for sex and race-ethnicity, parental closeness predicted lower age 13 violence for both maltreated and nonmaltreated youth, but this effect was significantly smaller for maltreated youth (Table 3). The association between friendship involvement and violence also differed significantly based on maltreatment: for nonmaltreated youth, greater friendship involvement predicted more violent behavior at age 13 followed by a significantly greater decrease in violence across adolescence. In contrast, friendship involvement had a significantly smaller effect on initial age 13 violence for maltreated youth, and friendship involvement was unrelated to violence change for maltreated youth.

Genetic susceptibility

To explore if group differences (maltreated vs. nonmaltreated) in sensitivity to parental closeness and friendship involvement were additionally heightened by genetic susceptibility, the main effect of dopaminergic risk and its interaction with parental closeness and friendship involvement were added to the model (Table 3). Models also included appropriate control for all gene–covariate and environment–covariate interactions to reduce the potential for spurious G×E (Keller, 2014). Overall, the direct effect of dopaminergic risk and its two-way interactions with parental closeness and friendship involvement did not significantly predict initial age 13 violence or its change from age 13 through 32, regardless of maltreatment history. Dopaminergic risk marginally interacted with friendship involvement to predict initial violence and change in violence over time for nonmaltreated youth (but not maltreated youth), although these interactions did not survive FDR correction to account for multiple testing.

Latent growth of nonviolent delinquency

When modeling growth in nonviolent delinquency, all fit indices (AIC, BIC, and sample-adjusted BIC) and the Satorra–Bentler χ^2 test indicated that the nonlinear (quadratic) unconditional model fit better than the linear (slope) unconditional model ($\Delta\chi^2$ (2) = 56.50, $p < .01$). Overall, nonviolent delinquency started low at age 13 (intercept $b = -1.357$, $SE = .078$, $p < .001$), and a quadratic growth pattern best characterized change in delinquency from age 13 through 32, such that delinquency increased in early-mid adolescence before decreasing in late adolescence and adulthood (slope $b = .075$, $SE = .019$, $p < .001$; quadratic $b = -.006$, $SE = .001$, $p < .001$). Multigroup models found that maltreated youth engaged in significantly more nonviolent delinquency behaviors at age 13 (intercept) compared with nonmaltreated youth, although their patterns of linear and quadratic change across time did not significantly differ (Figure 2).

Parenting and friendship

Controlling for sex and race-ethnicity, the effect of parental closeness and friendship involvement on nonviolent delinquency intercept significantly differed for maltreated vs. nonmaltreated youth (Table 4). Parental closeness predicted lower age 13 nonviolent delinquency for both maltreated and nonmaltreated youth, but this effect was significantly smaller for maltreated youth. Similar to results for violence, friendship involvement effects on nonviolent delinquency differed based on maltreatment history: for nonmaltreated youth, friendship involvement predicted significantly higher age 13 nonviolent delinquency at age 13 followed

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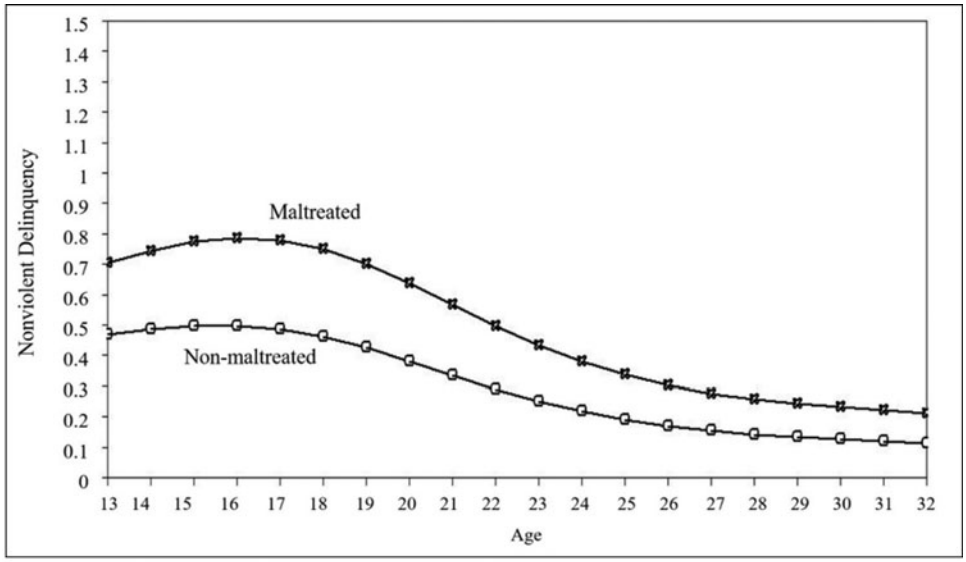
Table 3. Multigroup LGM parameter estimates of parental closeness, friendship involvement, and dopaminergic risk on violent behavior from age 13 to 32 for maltreated and nonmaltreated youth

	Full sample (n = 9,421)			Nonmaltreated (74%)			Maltreated (26%)			Wald test	
Predicting violence	*B* (*SE*)	ß	p^*	*B* (*SE*)	ß	p^*	*B* (*SE*)	ß	p^*	χ^2	p^*
Main environment effects[a]											
Intercept (age 13)											
Intercept	.172 (.208)	.155	.408	.155 (.288)	.138	.590	.053 (.361)	.052	.882		
Parental closeness	−.068 (.010)	−.259	<.001*	−.078 (.012)	−.275	<.001*	−.032 (.016)	−.149	.043	5.414	.020*
Friendship involvement	2.07 (.032)	.275	<.001*	.275 (.038)	.36	<.001*	.119 (.051)	.169	.019*	5.887	.015*
Linear slope (ages 13–32)											
Intercept	−.418 (.079)	−2.852	<.001*	−.402 (.102)	−2.663	<.001*	−.406 (.125)	−2.382	.001*		
Parental closeness	.006 (.003)	.184	.034	.011 (.004)	.276	.014*	−.002 (.005)	−.045	.744	2.943	.086
Friendship involvement	−.031 (.010)	−.311	<.001*	−.051 (.013)	−.494	<.001*	−.002 (.016)	.011	.936	5.542	.019*
Quadratic (ages 13–32)											
Intercept	.017 (<.001)	2.665	<.001*	.016 (.006)	2.265	.007*	.018 (.006)	2.229	.006*		
Parental closeness	<.001 (<.001)	−.148	.214	−.001 (<.001)	−.297	.073	<.001 (<.001)	.214	.184	4.099	.043
Friendship involvement	.002 (.001)	.412	.003*	.003 (.001)	.575	.001*	<.001 (.001)	.072	.679	2.923	.087
Adding G×E effects[b]											
Intercept (age 13)											
Intercept	.115 (.279)	.103	.681	.356 (.364)	.314	.328	−.370 (.595)	−.349	.534		
DA risk	.131 (.222)	.112	.554	.094 (.289)	.079	.746	.187 (.543)	.168	.730	.020	.888
DA Risk × Parenting	.013 (.012)	.049	.268	.022 (.017)	.076	.190	.014 (.018)	.063	.423	.099	.753
DA Risk × Friendship	.063 (.032)	.079	.049	.077 (.042)	.095	.065	.014 (.062)	.018	.821	.639	.424
Linear slope (ages 13–32)											
Intercept	−.463 (.105)	−2.911	<.001*	−.529 (.128)	−3.254	<.001*	−.363 (.155)	−1.765	.019*		
DA risk	.044 (.065)	.264	.496	.041 (.083)	.237	.623	.055 (.151)	.254	.715	.006	.939
DA Risk × Parenting	−.001 (.004)	−.029	.787	−.002 (.005)	−.059	.630	−.005 (.006)	−.062	.467	.062	.803
DA Risk × Friendship	−.024 (.011)	−.210	.033	−.027 (.014)	.231	.059	−.010 (.021)	−.068	.616	.419	.517
Quadratic (ages 13–32)											
Intercept	.019 (.006)	2.567	.002*	.023 (.008)	2.932	.003*	.011 (.009)	1.166	.203		
DA risk	−.002 (.003)	−.305	.497	−.004 (.005)	−.547	.383	−.002 (.007)	−.243	.744	.043	.835
DA Risk × Parenting	<.001 (<.001)	.030	.823	<.001 (<.001)	.072	.695	<.001 (<.001)	.073	.674	<.001	.994
DA Risk × Friendship	.001 (.001)	.233	.092	.001 (.001)	.221	.200	<.001 (.001)	.059	.733	.266	.606

Note: B = unstandardized parameter estimate; ß = standardized parameter estimate; DA = dopaminergic; G×E = Gene × Environment interaction; LGM = latent growth modeling; *significant effect after applying false discovery rate correction for multiple testing across all latent growth models; [a]models adjusted for sex and race-ethnicity; [b]models adjusted for sex, race-ethnicity, the main effects of parental closeness and friendship involvement, and all covariate–gene and covariate–environment interactions.



	Non-Maltreated		Maltreated		Wald Test	
	B (SE)	*p*	B (SE)	*p*	Value	*p*
Intercept	-1.386 (.051)	<.001*	-.980 (.028)	<.001*	88.036	<.001*
Slope	.051 (.016)	.001*	.069 (.009)	<.001*	1.437	.231
Quadratic	-.006 (.001)	<.001*	-.006 (.001)	<.001*	.153	.696

Note. * = significant effect after applying false discovery rate correction for multiple testing.

Figure 2. Latent growth curves of nonviolent delinquency from age 13 to 32 for maltreated and nonmaltreated youth.

and a greater decrease in nonviolent delinquency across time. In contrast, friendship involvement was unrelated to initial nonviolent delinquency nor its change over time for youth with maltreatment history.

Genetic susceptibility

Similar to results for violence, the direct effect of dopaminergic risk and its two-way interactions with parental closeness and friendship involvement did not significantly predict nonviolent delinquency at age 13 (intercept) nor its linear or quadratic change from age 13 to 32 (Table 4). These associations did not significantly differ based on maltreatment history.

Latent growth of substance use

All fit indices and the Satorra–Bentler χ^2 test indicated that the nonlinear (quadratic) model of substance use fit better than the linear (slope) unconditional model ($\Delta\chi^2$ (2) = 372.16, $p < .01$). Similar to the pattern of change for nonviolent delinquency, substance use started low at age 13 (intercept $b = -1.035$, $SE = .035$, $p < .001$), increased during adolescence (slope $b = .118$, $SE = .008$, $p < .001$), and then significantly decreased in rate of change in adulthood (quadratic $b = -.006$, $SE < .001$, $p < .001$). The multigroup model revealed that compared with nonmaltreated youth, maltreated youth used significantly more substances at age 13 (intercept), but did not differ in their pattern of substance use change across time (Figure 3).

Parenting and friendship

Controlling for sex and race-ethnicity, higher parental closeness in early adolescence was associated with lower initial substance use at age 13, but a greater increase in substance use across development (Table 5). In contrast, greater friendship involvement predicted more substance use at age 13 and a slower decrease in substance use across time. These differential effects of parental closeness and friendship involvement on substance use did not differ between maltreated and nonmaltreated youth.

Genetic susceptibility

The direct effect of dopaminergic risk and its two-way interactions with parental closeness did not significantly predict substance use at age 13 (intercept), and it also did not predict linear or quadratic change in substance use from age 13 through 32 (Table 5). G×E effects did not differ based on maltreatment history. Similarly, dopaminergic risk did not moderate friendship involvement in predicting age 13 substance use or its change over time, regardless of maltreatment history.

Discussion

In a nationally representative sample of adolescents followed into adulthood, we modeled separate latent trajectories of violence, nonviolent delinquency, and substance use from age 13 to 32 years and explored the effects of childhood maltreatment on these developmental trajectories. We additionally tested whether maltreatment history affected sensitivity to perceived parental closeness and friendship involvement in adolescence, including whether these effects were differentially affected by dopamine genes associated with environmental sensitivity. Overall, youth with childhood maltreatment histories had consistently higher levels of all three EB outcomes from adolescence to young adulthood; however, their patterns of EB change did not differ from nonmaltreated youth. Violence significantly decreased across adolescence before stabilizing at a low level in adulthood, whereas nonviolent delinquency and substance use followed negative quadratic patterns of increasing in adolescence and then decreasing in

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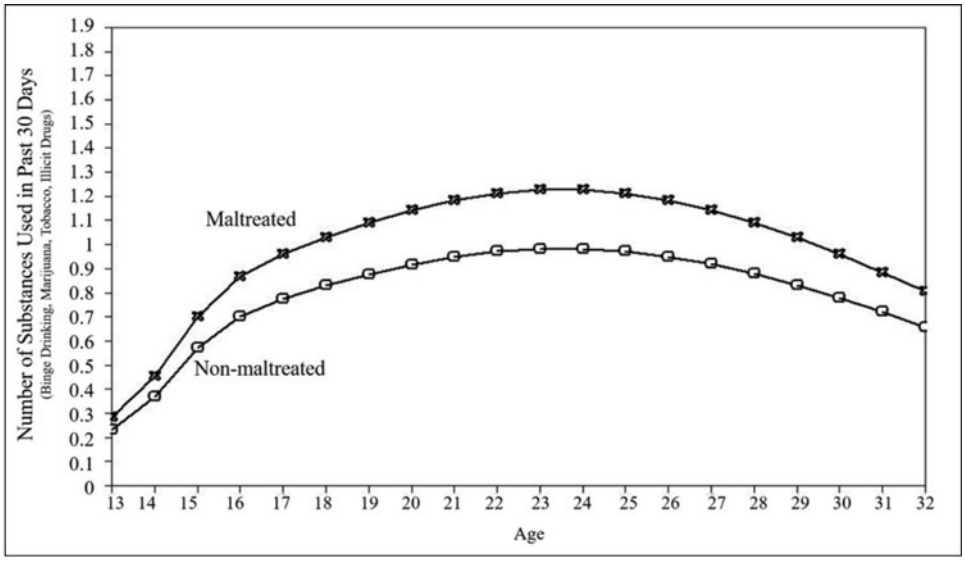
Table 4. Multigroup LGM parameter estimates of parental closeness, friendship involvement, and dopaminergic risk on nonviolent delinquency from ages 13 to 32 for maltreated and nonmaltreated youth

	Full sample (n = 9,421)			Nonmaltreated (74%)			Maltreated (26%)			Wald test	
Predicting nonviolent delinquency	*B* (*SE*)	ß	p^*	*B* (*SE*)	ß	p^*	*B* (*SE*)	ß	p^*	χ^2	p^*
Main environment effects[a]											
Intercept (age 13)											
Intercept	−.467 (.2060)	−.408	.023*	−.367 (.265)	−.313	.165	−.637 (.366)	−.595	.082		
Parental closeness	−.099 (.010)	−.363	<.001*	−.112 (.011)	−.377	<.001*	−.071 (.014)	−.317	<.001*	6.372	.012*
Friendship involvement	.168 (.029)	.216	<.001*	.239 (.037)	.299	<.001*	.067 (.046)	.090	.147	7.458	.006*
Linear slope (age 13–32)											
Intercept	−.215 (.056)	−2.049	<.001*	−.269 (.074)	−2.170	<.001*	−.118 (.091)	−1.282	.196		
Parental closeness	.010 (.003)	.405	.003*	.015 (.004)	.478	<.001*	.005 (.005)	.273	.286	2.678	.102
Friendship involvement	−.021 (.009)	−.295	.014*	−.040 (.010)	−.471	<.001*	.002 (.015)	.031	.891	5.505	.019*
Quadratic (ages 13–32)											
Intercept	.007 (.003)	1.64	.030	.008 (.004)	1.464	.073	.003 (.005)	.856	.557		
Parental closeness	<.001 (<.001)	−.374	.029	−.001 (<.001)	−.490	.001*	<.001 (<.001)	−.066	.842	3.973	.046
Friendship involvement	.001 (<.001)	.314	.060	.002 (.001)	.542	.001*	<.001 (.001)	−.083	.802	4.548	.033
Adding G×E effects[b]											
Intercept (age 13)											
Intercept	−.532 (.241)	−.465	.027*	−.621 (.325)	−.546	.056	−.663 (.461)	−.588	.150		
DA risk	.444 (.227)	.368	.051	.488 (.299)	.400	.102	.415 (.367)	.351	.258	.022	.883
DA Risk × Parenting	.015 (.008)	.054	.068	.021 (.012)	.070	.066	.008 (.016)	.033	.620	.408	.523
DA Risk × Friendship	.005 (.036)	.006	.898	.001 (.039)	.001	.983	.039 (.058)	.047	.493	.314	.576
Linear slope (ages 13–32)											
Intercept	−.244 (.075)	−2.069	.001*	−.184 (.100)	−1.400	.066	−.218 (.129)	−1.543	.092		
DA risk	−.032 (.071)	−.255	.654	−.101 (.081)	−.727	.215	.008 (.105)	.052	.942	.644	.422
DA Risk × Parenting	−.001 (.002)	−.027	.753	−.002 (.003	−.048	.607	.001 (.004)	.039	.780	.238	.626
DA Risk × Friendship	<.001 (.011)	.003	.981	.001 (.012)	.011	.934	−.015 (.018)	−.143	.397	.557	.456
Quadratic (age 13–32)											
Intercept	.009 (.004)	1.682	.045	.001 (.006)	.201	.817	.008 (.007)	1.178	.266		
DA risk	<.001 (.004)	−.069	.921	.003 (.005)	.415	.517	−.004 (.005)	−.516	.487	.973	.324
DA Risk × Parenting	<.001 (<.001)	−.041	.695	<.001 (<.001)	−.017	.866	<.001 (<.001)	−.092	.540	.115	.734
DA Risk × Friendship	<.001 (.001)	−.043	.792	<.001 (.001)	−.064	.649	.001 (.001)	.120	.540	.590	.443

Note: B = unstandardized parameter estimate; ß = standardized parameter estimate; DA = dopaminergic; G×E = Gene × Environment interaction; LGM = latent growth modeling; *significant effect after applying false discovery rate correction for multiple testing across all latent growth models; [a]models adjusted for sex and race-ethnicity; [b]models adjusted for sex, race-ethnicity, the main effects of parental closeness and friendship involvement, and all covariate–gene and covariate–environment interactions.



	Non-Maltreated		Maltreated		Wald Test	
	B (SE)	*p*	B (SE)	*p*	Value	*p*
Intercept	-.035 (.035)	<.001*	-.836 (.029)	<.001*	34.856	<.001*
Slope	.118 (.008)	<.001*	.123 (.006)	<.001*	.309	.578
Quadratic	-.006 (<.001)	<.001*	-.006 (<.001)	<.001*	.398	.585

Note. * = significant effect after applying false discovery rate correction for multiple testing.

Figure 3. Latent growth curves of substance use from age 13 to 32 for maltreated and nonmaltreated youth.

adulthood. Maltreatment appeared to reduce sensitivity to parental closeness and friendship involvement on violence and nonviolent delinquency in early adolescence and change in these outcomes over time. In contrast, predictions of substance use from parental closeness and friendship involvement were comparable for maltreated versus nonmaltreated youth. Finally, there were no maltreatment group differences with respect to the effects of dopaminergic genetic risk on any of the EB outcomes, and dopaminergic risk did not significantly interact with parental closeness or friendship involvement after including appropriate gene–covariate and covariate–environment interactions and correcting for multiple testing. We discuss each of these findings in the context of previous literature and emphasize key considerations when interpreting these results.

First, as expected, adolescents with maltreatment histories had significantly higher levels of violence, nonviolent delinquency, and substance use in early adolescence compared with nonmaltreated youth, a pattern that persisted into adulthood. The differential patterns of growth for violent behavior versus nonviolent delinquency and substance use are consistent with prior evidence that the developmental trajectory of EB varies based on the type of behavioral outcome measured (Bongers, Koot, van der Ende, & Verhulst, 2004). Indeed, for the majority of youth, overtly aggressive and violent behaviors typically decrease with age starting in childhood (Côté, Vaillancourt, LeBlanc, Nagin, & Tremblay, 2006), whereas the quadratic patterns of growth for nonviolent delinquency and substance use are consistent with prevailing developmentally informative models of adolescent EB (Moffitt, 1993). Of importance, despite these differential growth patterns between types of EB, maltreated youth exhibited significantly higher violence, nonviolent delinquency, and substance use from age 13 to 32 compared with nonmaltreated youth. These group differences are consistent with studies showing that maltreatment not only predicts EB in adolescence (Egeland et al., 2002; Oshri et al., 2011), but also has enduring effects on EB in adulthood (Lansford et al., 2007). Because few studies have compared developmental trajectories of EB for maltreated versus nonmaltreated youth, it has been unclear if maltreatment affects the way EB develops into adulthood. Multigroup LGM showed that group differences in EB based on maltreatment were specific to a higher intercept rather than linear or quadratic change, suggesting that the enduring effects of maltreatment operate by elevating initial levels of EB that are maintained over time, rather than changing the pattern of EB development in adolescence or adulthood. Because we did not model development before early adolescence, however, it is unclear when this severity gap emerges. Studies that model these developmental patterns in preadolescence and childhood are needed to further elucidate how and when maltreatment begins to predict significant differences in EB, which will clarify key developmental periods to target through prevention programs for maltreated youth.

Beyond the developmental patterns of EB, our study also examined how childhood maltreatment affected later sensitivity to parental closeness. Overall, perceived parental closeness in early adolescence predicted lower initial levels of all EB outcomes, even after controlling for sex, race-ethnicity, and friendship effects. These cohesive results across multiple EB outcomes are consistent with a large body of literature supporting the protective effects of perceived support and emotional closeness with a parent (Branstetter, Low, & Furman, 2011; Chassin et al., 2005); however, these protective effects of parental closeness on violence and nonviolent delinquency in early adolescence (but not substance use) were significantly smaller in magnitude for maltreated versus nonmaltreated youth. Thus, one potential way severe and traumatic stressors such as maltreatment may lead to enduring EB over time is by blunting children's receptivity to later protective

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Table 5. Multigroup LGM parameter estimates of parental closeness, friendship involvement, and dopaminergic risk on substance use from ages 13 to 32 for maltreated and nonmaltreated youth

	Full sample (n = 9,421)			Nonmaltreated (74%)			Maltreated (26%)			Wald test	
Predicting substance use	*B* (*SE*)	ß	p^*	B (*SE*)	ß	p^*	B (*SE*)	ß	p^*	χ^2	p^*
Main environment effects[a]											
Intercept (age 13)											
Intercept	−1.238 (.239)	−1.28	<.001*	−1.216 (.272)	−1.23	<.001*	−1.298 (.345)	−1.389	<.001*		
Parental closeness	−.086 (.007)	−.370	<.001*	−.092 (.008)	−.365	<.001*	−.070 (.012)	−.357	<.001*	2.400	.121
Friendship involvement	.279 (.027)	.423	<.001*	.308 (.033)	.458	<.001*	.221 (.051)	.343	<.001*	1.967	.161
Linear slope (age 13–32)											
Intercept	.007 (.047)	.082	.882	−.038 (.072)	−.401	.600	.065 (.062)	.893	.292		
Parental closeness	.012 (.001)	.567	<.001*	.013 (.002)	.545	<.001*	.010 (.003)	.627	.001*	.907	.341
Friendship involvement	−.031 (.006)	−.530	<.001*	−.035 (.007)	−.550	<.001*	−.023 (.011)	−.452	.036	.902	.342
Quadratic (age 13–32)											
Intercept	<.001 (.002)	−.058	.922	.001 (.004)	.284	.753	−.002 (.003)	−.489	.576		
Parental closeness	<.001 (<.001)	−.541	<.001*	−.001 (<.001)	−.518	<.001*	<.001 (<.001)	−.561	.005*	.800	.371
Friendship involvement	.001 (<.001)	.525	<.001*	.001 (<.001)	.545	<.001*	.001 (.001)	.455	.060	.582	.446
Adding G×E effects[b]											
Intercept (age 13)											
Intercept	−1.114 (.305)	−1.143	<.001*	−1.262 (.335)	−1.251	<.001*	−1.071 (.424)	−1.091	.012		
DA risk	−.107 (.206)	−.105	.603	−.035 (.264)	−.033	.894	−.192 (.322)	−.187	.550	.160	.689
DA Risk × Parenting	.006 (.008)	.025	.428	.010 (.010)	−.038	.308	.005 (.015)	.022	.758	.093	.760
DA Risk × Friendship	.005 (.029)	.007	.861	.018 (.033)	.025	.574	−.030 (.049)	−.041	.539	.599	.439
Linear slope (age 13–32)											
Intercept	−.027 (.066)	−.284	.684	−.033 (.083)	−.304	.691	−.024 (.090)	−.241	.787		
DA risk	.019 (.040)	.188	.637	−.007 (.050)	−.063	.886	.030 (.065)	.278	.649	.211	.646
DA Risk × Parenting	−.001 (.002)	−.040	.596	−.001 (.002)	−.043	.558	−.002 (.003)	−.101	.493	.079	.778
DA Risk × Friendship	−.002 (.007)	−.023	.816	−.005 (.008)	−.061	.529	.006 (.010)	.080	.560	.757	.384
Quadratic (age 13–32)											
Intercept	.001 (.003)	.330	.657	.001 (.004)	.207	.811	.003 (.004)	.590	.516		
DA risk	−.001 (.002)	−.192	.631	<.001 (.002)	−.034	.944	−.001 (.003)	−.155	.805	.021	.885
DA Risk × Parenting	<.001 (<.001)	.030	.707	<.001 (<.001)	.013	.965	<.001 (<.001)	.149	.343	.577	.447
DA Risk × Friendship	<.001 (<.001)	.026	.815	<.001 (<.001)	.074	.505	<.001 (<.001)	−.105	.472	1.057	.304

Note: B = unstandardized parameter estimate; ß = standardized parameter estimate; DA = dopaminergic; G×E = Gene × Environment interaction; LGM = latent growth modeling; *significant effect after applying false discovery rate correction for multiple testing across all latent growth models; [a]models adjusted for sex and race-ethnicity; [b]models adjusted for sex, race-ethnicity, the main effects of parental closeness and friendship involvement, and all covariate–gene and covariate–environment interactions.

factors such as parental closeness and warmth in early adolescence. This is consistent with previous evidence that maltreatment impairs relational processes (e.g., attachment) involved in facilitating positive parent–child relationships (Cicchetti & Banny, 2014; Lowell et al., 2014), and suggests this may be particularly important in the context of parental closeness as a buffer for adolescent and young adult EB outcomes.

In considering these results, however, it is important to note that parents who engage in child abuse or neglect may also show lower parental warmth and closeness in early adolescence. Indeed, maltreatment was weakly but significantly correlated with parental closeness in this sample; thus, it is possible that the attenuated association between parental closeness and EB for maltreated youth reflects that adolescents feel less close to the parent who either maltreated them or was unable to protect them from maltreatment. Alternatively, youth who exhibit more violent and nonviolent delinquent behaviors may evoke negative parenting behaviors through evocative G×E correlations (rGE). Although parenting was not correlated with the polygenic dopamine score in our sample, some studies report that compared with adopted children without genetic risk (i.e., parental history of antisocial behavior), children with genetic risk for antisocial behavior were more likely to receive negative parenting from adoptive parents, supporting an evocative rGE (O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998). Much of the association between negative parenting and child behavior problems was not explained by the evocative rGE, however, suggesting that parenting also has an environmentally mediated effect on these EB outcomes (O'Connor et al., 1998). These findings support the plausibility that parental closeness plays a causal protective role in reducing EB over time, and our results suggest that youth exposed to childhood abuse or neglect may show reduced sensitivity to these effects in adolescence. Studies that experimentally change parenting behavior (e.g., intervention studies) and separate maltreatment from later parenting behavior (e.g., adoption studies of previously maltreated youth) are needed to substantiate these differential patterns of sensitivity for maltreated and nonmaltreated youth, as well as to test potential mediating mechanisms underlying these differences in sensitivity.

In contrast to the generally protective effects of parental closeness in early adolescence, friendship involvement initially predicted higher engagement in violence, nonviolent delinquency, and substance use at age 13, followed by a greater decrease in these EB outcomes across time. These results highlight the differential influences of dyadic parenting from dyadic friendship effects in adolescence and emphasize the need to more closely examine what close friendship entails, particularly in the developmental context of adolescence. For example, the current study used a broad measure of friendship involvement (e.g., how much time spent with a friend, how often they communicate or talk about problems), which can reflect several separable aspects of friendship, ranging from emotional closeness/support to number of hours spent in each other's presence. For example, exposure to peer deviant behavior and substance use is consistently related to adolescent EB over time (Branstetter et al., 2011), and adolescents may tend to choose friends that engage in similar levels of EB (Hou et al., 2013; Poulin, Kiesner, Pedersen, & Dishion, 2011). Our findings highlight the need to better understand the mechanisms underlying the association between friendship involvement and EB in early adolescence, which may differ from the mechanisms underlying the overall protective effects of a close friendship on the developmental pattern of these outcomes in the transition from adolescence to adulthood.

Similar to its effect on sensitivity to parental closeness, maltreatment also attenuated sensitivity to friendship involvement on violence and nonviolent delinquency in early adolescence. This difference became particularly pronounced when examining EB change over time: friendship involvement predicted greater decreases in both violence and nonviolent delinquency for nonmaltreated youth, but this protective effect was not observed for maltreated youth. That maltreated youth showed a blunted sensitivity to friendship effects, social relationships outside of the context of the home, suggests that childhood maltreatment may attenuate later sensitivity to social relationships overall and not just to the same parents who potentially maltreated the adolescents in childhood. This decreased responsivity to social relationships is consistent with the adaptive calibration model (Del Giudice et al., 2011) and supports conceptualization of childhood maltreatment as a severe and traumatic stressor with enduring effects on environmental sensitivity across development. Given that our study did not directly assess the severity of physical and psychological consequences of maltreatment (e.g., injuries, posttraumatic stress symptoms), studies are needed that model these potential mediators within the context of adaptive calibration model, a critical next step to understanding the mechanisms through which early maltreatment may influence sensitivity to parenting and peer relationships in adolescence.

Finally, we explored whether group differences in sensitivity to parenting and friendship effects may be influenced by dopamine genes previously linked to environmental sensitivity (Bakermans-Kranenburg & van IJzendoorn, 2011; Belsky & Beaver, 2011). Given the heightened susceptibility of candidate G×E studies to Type I error (Duncan & Keller, 2011), our models corrected for multiple testing using a FDR adjustment and accounted for each covariate's interaction term with dopaminergic risk and the environment variables (parental closeness, friendship involvement) to minimize the potential for spurious G×E caused by gene–covariate and covariate–environment interactions (Keller, 2014). Dopaminergic risk marginally moderated the association between friendship involvement and violence, but none of these GxE survived correction for multiple testing. Furthermore, neither the direct effects of dopaminergic risk nor any of the G×E effects differed between maltreated and nonmaltreated youth. Given the overall dearth of G×E studies modeling these effects in the context of developmentally sensitive growth models, our results emphasize the need for other well-powered replication studies of G×E that investigate EB across time while rigorously controlling for potential confounding factors and multiple testing. Furthermore, although our study focused on the three most commonly investigated dopaminergic variants related to differential susceptibility and EB (Bakermans-Kranenburg & van IJzendoorn, 2011), it is likely that other candidate genes that influence dopamine efficiency (e.g., COMT, DRD2-141C) and other neurotransmitter systems (e.g., serotonin, GABA) influence these processes as well. Well-powered longitudinal studies are therefore needed to further evaluate whether these and other genetic markers contribute to clinically meaningful differences in sensitivity to the social environment across later stages of development, beyond the direct effects of early environmental exposures (e.g., maltreatment) on EB.

These results should be interpreted in the context of several important study limitations. First, because adolescents reported on perceived parental closeness, friendship involvement, maltreatment history, and EB, findings are subject to shared method variance. Future replication studies including additional informants and assessment procedures are needed, although the convenience

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and efficiency of self-report measures is an important consideration in large-scale survey studies such as Add Health. Furthermore, by including temporal separation between constructs (e.g., parental closeness and friendship at Wave 1 and EB at Waves 1–4), some reduction of method bias was afforded in this study (Podsakoff, MacKenzie, & Podsakoff, 2012). Second, childhood maltreatment was retrospectively assessed, which may underestimate the frequency of maltreatment compared with prospective longitudinal studies of maltreatment (Shaffer, Huston, & Egeland, 2008). Similarly, the present study explored how maltreatment broadly affects EB development and sensitivity to social factors; however, the timing, type, and severity of maltreatment may differentially affect these developmental processes as well (Jackson, Gabrielli, Fleming, Tunno, & Makanui, 2014; Manly, Cicchetti, & Barnett, 1994). Although these questions were beyond the scope of this study, we encourage future studies to further elucidate these potential differences in childhood maltreatment that may differentially affect youth's sensitivity to social factors in adolescence.

Overall, our findings underscore the enduring effects of childhood maltreatment on EB development from adolescence to young adulthood, and they suggest that one way maltreatment contributes to enduring EB is by dampening sensitivity to parenting and peer factors in adolescence. Understanding how and when youth are differentially sensitive to the social environment is a central topic in developmental psychopathology and key to tailoring psychosocial interventions for youth at risk for EB. This recognition is evident in the influx of studies in the past decade exploring genetic effects underlying differential susceptibility to the environment. Although G×E studies continue to represent a promising avenue for understanding individual differences in environmental sensitivity, our results highlight the need to reconsider the role of development itself and, specifically, the dynamic nature of the environment across time when investigating complex developmental phenotypes such as EB. Given the multiple factors across biological and social domains that likely affect environmental sensitivity, we encourage more studies to actively integrate dynamic models of EB (e.g., including multiple measures of environmental stress and support across time) to further elucidate the developmental mechanisms underlying EB development across the life span.

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