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Regular Article

Polygenic risk and hostile environments: Links to stable and dynamic antisocial behaviors across adolescence

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Abstract

Adverse environments are linked to elevated youth antisocial behavior. However, this relation is thought to depend, in part, on genetic susceptibility. The present study investigated whether polygenic risk for antisociality moderates relations between hostile environments and stable as well as dynamic antisocial behaviors across adolescence. We derived two antisocial-linked polygenic risk scores (PRS) ($N = 721$) based on previous genome-wide association studies. Forms of antisocial behavior (nonaggressive conduct problems, physical aggression, social aggression) and environmental hostility (harsh parenting and school violence) were assessed at age 13, 15, and 17 years. Relations to individual differences stable across adolescence (latent stability) vs. time-specific states (timepoint residual variance) of antisocial behavior were assessed via structural equation models. Higher antisocial PRS, harsh parenting, and school violence were linked to stable elevations in antisocial behaviors across adolescence. We identified a consistent polygenic-environment interaction suggestive of differential susceptibility in late adolescence. At age 17, harsher parenting was linked to higher social aggression in those with higher antisocial PRS, and lower social aggression in those with lower antisocial PRS. This suggests that genetics and environmental hostility relate to stable youth antisocial behaviors, and that genetic susceptibility moderates home environment-antisocial associations specifically in late adolescence.

Keywords: antisocial behavior; adolescence; environment adversity; longitudinal; polygenic risk score

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Antisociality is a cluster of traits and behaviors centered around disregarding, harming, and violating the rights of others. The expression of antisocial behaviors is heterogenous; there is substantive variation in severity, form, time of onset, and persistence (Gard et al., 2019). Antisocial behaviors are theorized to fall into three etiologically and developmentally distinct, but related, types: physical aggression (acts or threats of bodily harm), social aggression (harming others via relationship or reputational damage), and nonaggressive conduct problems (rule-breaking, lying; Burt et al., 2012; Burt, 2012; 2013). Forms of antisocial behavior can be moderately to strongly linked to one another (Acland et al., 2021; Cheng et al., 2023; Olson et al., 2013). Yet, the expression of these behaviors can fluctuate asynchronously over time, which may be brought about by distinct environmental and genetic influences, especially upon entering adolescence (Karriker-Jaffe et al., 2013; Niv et al., 2013). The nuances of these interactive processes are not well-understood, and are thus, the focus of the present study.

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The development of antisocial behaviors

Antisocial behaviors seldom begin in adulthood (Harden et al., 2015; Moffitt, 2018). Physical aggression peaks in early childhood, while nonaggressive conduct problems peak in adolescence (Côté et al., 2006; Givens & Reid, 2019; Niv et al., 2013; Tremblay et al., 2018; Zheng & Cleveland, 2013). Both decline with age after their peak, emphasizing the importance of studying the development of antisocial behaviors pre-adulthood. Twin studies indicate that physical, stable aggression is particularly heritable (~65%), more so than nonaggressive conduct problems (Burt, 2012; Lacourse et al., 2014). Nonaggressive antisocial behaviors (e.g., rule-breaking, stealing, truancy) are found to be more dynamic, more affected by shared environmental factors, and exhibit less genetic stability (Burt, 2012; Eley et al., 2003).

Distinctions between aggression and rule-breaking may be best understood in adolescence as the unique influencers of each behavior emerge more consistently post-puberty (Harden et al., 2015;

Niv et al., 2013). For instance, the effect of family-level environmental factors on aggression and rule-breaking becomes increasingly differentiated when children enter adolescence (Harden et al., 2015). Additionally, some genetic influences on antisocial behavior are not observed until after puberty and the influence of genetics on nonaggressive conduct problems increases from childhood to adolescence (Burt & Neiderhiser, 2009; Harden et al., 2015; Jacobson et al., 2002; Niv et al., 2013). In sum, research suggests that differing forms of antisocial behavior have distinct etiological and developmental pathways, and that adolescence is a key period for understanding these differences.

Hostile environments and antisocial behavior

Exposure to hostile environments is an established risk factor for antisocial behavior (Bacchini et al., 2011; Estrada & Baskin-Sommers, 2023). Namely, parent-child coercion and peer antisociality are identified as two critical microsystem factors for the maintenance and escalation of antisocial problems (Dishion & Patterson, 2016). Harsh parenting styles are linked to increased aggression and antisocial behaviors more generally over childhood and adolescence (Côté et al., 2006; Compton et al., 2003; Evans et al., 2012; Girard et al., 2019; Kawabata et al., 2011; Li et al., 2011). Similarly, research suggests that being a victim of family violence and witnessing violence at school independently relate to increased antisocial behavior over adolescence (Bacchini et al., 2015; Calvete & Orue, 2011; O'Keefe, 1997). Direct contact with violence may not even be necessary, as fear evoked from living in violent neighborhoods is linked to poorer mental health in youth even when personal exposure is controlled (Cruz et al., 2021). Social proximity to antisocial behaviors at school has been associated to their amplification and spread (Dimant, 2019; Kalvin & Bierman, 2017; Kornienko et al., 2018). Thus, sharing environments with hostile actors, whether that be parents or peers, can lead to distress in youth and the mirroring of those behaviors.

Together this suggests that both school and home environmental hostility are important etiological factors of antisocial behavior. However, adolescents who experience harsh parenting or report having violent peers at school do not necessarily go on to display elevated antisocial behavior. Such variability in the face of these criminogenic environments point to possible individual-level factors that may exacerbate or buffer the associations between environments and antisocial behavior. A key factor that may lead youth to be susceptible to environmental adversity is genetic risk for antisocial behavior.

Polygenic-environment interactions

Heritability of behavioral phenotypes is highly polygenic, and genome wide association studies (GWAS) are increasingly used to identify the polygenic contribution (via single nucleotide polymorphisms; SNPs) to antisocial behavior (Gard et al., 2019; Pappa et al., 2016; Tielbeek et al., 2022). These genetic variation candidates can be used to create composite scores (i.e., polygenic risk scores; PRS) that represent individuals' continuously distributed genetic risk for a particular trait. These derived PRS allow for the assessment of polygenic-environment interactions. For instance, a higher PRS for aggression was found to be related to greater lifetime incarceration risk, however, this effect was substantially reduced if at least one parent graduated high school (Barnes et al., 2019). Similarly, children with low family instability combined with low polygenic risk for aggression had steeper declines in aggression from ages 7 to 14 (Womack et al., 2021). A higher PRS for externalizing problems was also linked to increased adolescent externalizing symptoms, especially when parental monitoring was low (Salvatore et al., 2015). In somewhat contrast to these other studies that show more genetic and environment risk predicts greater antisociality, Musci et al. (2019) found that middle schoolers with a *lower* PRS for conduct problems were actually more likely to engage in impulsive, aggressive behaviors if exposed to community violence. This research demonstrates that polygenic risk for antisociality can moderate links between environmental factors and the development of antisocial behaviors. Thus, how genetic liability moderates environmental-antisocial relations may depend on type and feature of the environment measured (e.g., home vs. outside home). To our knowledge, there has been no investigation that has attempted to disentangle these processes by assessing polygenic-environment interactions using multiple antisocial-linked PRS, environment contexts, forms of antisocial behavior, and whether relations affect stable vs. unstable behaviors with longitudinal data.

Present study

Using a sample followed longitudinally, this study examined whether genetic liability for antisociality moderated the relations between home and school environmental hostility and antisocial behaviors across adolescence. Specifically, we assessed (1) how genetic susceptibility (via two antisocial-linked PRS) moderated relations between harsh parenting and school violence and antisocial behaviors, (2) whether this moderation operated differently for aggressive versus nonaggressive and socially vs. physically aggressive forms of antisocial behavior, and (3) whether interactions differed between antisocial behaviors that are stable across adolescence vs. behaviors that fluctuate between ages.

Based on past research suggesting that physical aggression, nonaggressive conduct problems, and social aggression are distinct constructs with differing developmental trajectories and etiologies, we expected our model to have better fit when forms of antisocial behavior were modeled as separate, but covarying constructs, as opposed to modeling them as one general factor of antisocial behavior. Physical, stable aggression is estimated to be highly heritable, whereas nonaggressive conduct problems have lesser heritable components, which increase over development (Burt et al., 2015; Burt, 2012). Nonaggressive problems are less stable across time and more influenced by environmental factors, however, environment still plays a role in physical aggression (Burt, 2012).

Thus, we hypothesized that greater levels of home and school environment hostility would link to (1) higher stable physical aggression, but only in genetically susceptible youth, and (2) higher stable and dynamic nonaggressive problems. Social aggression was included to assess whether any relations found were unique to physical aggression, as opposed to all forms of aggression; we expected that the antisocial PRS would be specifically linked to stable physical aggression, as opposed to social aggression. Additionally, the strength of associations between PRS and antisociality may depend on sex, thus we explored whether relations differed between sexes (Tielbeek et al., 2017). Sex and income were assessed as control variables given their established relations to antisocial behavior (Côté et al., 2006).

Method

Participants

Data came from the Quebec Longitudinal Study of Child Development (QLSCD): a well-established, population-based, longitudinal birth cohort in Canada (conducted by Institut de la

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Statistique du Québec) that began in 1997/98 ($N = 2120$). Participants were recruited from the Quebec Birth Registry via a stratified procedure based on location and birth rate. Data collection is ongoing and has been collected every one or two years from birth into adulthood. Further detail about the cohort can be found online (https://www.jesuisjeserai.stat.gouv.qc.ca/) and in the cohort profile (Orri et al., 2021). In the present study, we used data from when participants were 13, 15, and 17 years of age. A subset of the QLSCD sample reporting European ancestry was genotyped ($n = 951$), resulting in a final sample size of $n = 721$ for the present study after genetic quality control (see missing data section for genetic subsample information).

Procedure

The QLSCD protocol was approved by the Institut de la statistique du Québec and the St-Justine Hospital Research Centre ethics committees, and informed assent and consent was acquired at each data collection. Interviewer Completed Computerized Questionnaires (ICCQ) were carried out mainly by phone by a researcher interviewing the person most knowledgeable about the child (the mother in more than 98% of cases) to collect sociodemographic and parenting information. At age 10, blood and saliva samples were collected ($n = 951$) and DNA extraction was performed as soon as the sample were received at the lab (~ second day after sample draw).

Measures

Polygenic risk scores

Two GWAS on antisocial behavior (ASB) were used to derive PRS in the present study as participant characteristics (e.g., age), measures used (e.g., informant of antisocial behavior) and sample size of the original GWAS can affect the PRS's predictive value. One ASB-PRS was derived from the Pappa et al. (2016) GWAS, which included participants relatively close in age to the present sample (i.e., children and early adolescents; $N = 18{,}988$). Measures were caregiver-reported and included items that tapped into different facets of antisocial traits and behaviors (e.g., items used such as "Mean to others," "Disobedient," "Selfish," "Attacks people," "Often lies or cheats"). The second ASB-PRS was derived from the Tielbeek et al. (2022) GWAS, which included largely adults, but had a larger sample ($N = 85{,}359$). It also included a range of antisocial measures, many of which were self-reported, as used in the present study. These scores are referred to ASB-PRS_c (Pappa et al., 2016; child sample GWAS) and ASB-PRS_a (Tielbeek et al., 2022; adult sample GWAS).

DNA in the QLSCD sample was genotyped using the Illumina Infinium PsychArray-24 and the 1000 Genomes Phase 3 reference panel was used for quality control and imputation. After quality control and imputation, 721 individuals and several million genetic variants remained (see Appendix S1 for more details). The PRS was calculated using PRS-CS (see Ge et al., 2019). Here, the global shrinkage parameter phi was fixed to 0.01, as suggested for highly polygenic traits (Ge et al., 2019). Polygenic scores were then calculated using imputed dosage data in PLINK 1.9 using the PRS-CS adjusted summary statistics. PRS were transformed into standardized scores in main analyses.

To account for population stratification and outliers, 20 genetic ancestry principal components were included as covariates in the models. After controlling for the false discovery rate, none of the 20 components were significantly ($p < .05$) related to antisocial behaviors. Inclusion of ancestry components in the models did not substantially alter the estimates of the main predictor variables.

School violence

At age 13, 15, and 17 years, youth reported on violence that took place in their school environment using the Major Perceived Violence 3-item subscale (Janosz et al., 2008). Participants were asked to report how often they observed or have been informed of the following behaviors occurring at school during the present school year: "Students being physically assaulted by other students (beaten up, punched, kicked)," "Students who intimidate (threaten) adults at the school," and "Students who physically attack adults at the school." Participants rated each item on a 5-point Likert scale ranging from 1 = *Never* to 5 = *Almost every day*. Scale reliability was largely acceptable: average inter-item $r = 0.35, 0.53, 0.49$ for age 13, 15, 17, respectively[1]. In principal component factor analyses, all items loaded onto a single factor (loadings all > 0.7 at each age), suggesting all items tap into the same dimension. For main analyses, all items were standardized then averaged together.

Harsh parenting

At age 13, 15, and 17, the person most knowledgeable about the child was interviewed about their parenting behavior using a 4-item harsh parenting subscale assessing the frequency of their violent and harsh interactions with their child. Caregivers were asked (over the last 12 months): "How often did you hit (child's name) when (child's name) was difficult?" and "How often did you get angry when you punished (child's name)?"; when (child's name) broke the rules or did things that (child's name) was not supposed to "how often did you raise your voice, scold or yell at (child's name)?" and "how often did you use physical punishment?". The first item was rated on a Likert scale from 1 = *Never* to 7 = *Several times a day*, and the latter three items were rated from 1 = *Never* to 5 = *All the time/Always*. Scale reliability was acceptable: average inter-item $r = 0.30, 0.25, 0.20$ for age 13, 15, 17, respectively. Principal component analyses conducted at each time point suggested items loaded onto two dimensions: (1) harsh parenting (all loadings > 0.4, except for the two violent items at age 17 which were > 0.3) and (2) violent parenting (> 0.5 loadings for physically violent items, < −0.2 loadings for the remaining items). This suggests that all items tap into a common harsh parenting factor, and that corporal punishment items also tap into a dimension specific to violent parenting. Since we are more interested in harsh parenting, these four items were standardized and then averaged together.

Antisocial behavior

At age 13, youth self-reported their nonaggressive conduct problems (stealing, lying, cheating, rule-breaking; 6 items, $\alpha = 0.70$), physical aggression (physically harming or threatening to harm others; 8 items, $\alpha = 0.83$), and social aggression (emotional harming others, damaging relationships/reputation; 4 items, mean inter-item $r = 0.37$) during the last six months via the Social Behavior Questionnaire (SBQ; Collet et al., 2023)[2].

At age 15 and 17, youth completed the Mental Health and Social Inadaptation Assessment for Adolescents (MIA; Côté et al., 2017).

[1]Cronbach's alpha penalizes for number of scale items. For scales with fewer than 10 items, inter-item correlations are recommended to assess scale reliability (Pallant, 2020, p. 102). Average inter-item correlations are recommended to be between 0.15–0.50 (Clark & Watson, 1995), the lower range is expected for scales capturing more heterogenous constructs.

[2]SBQ includes items adapted from the Canadian National Longitudinal Study of Children and Youth (Statistics Canada, 1996), Child Behavior Checklist (Achenbach & Edelbrock, 1991), the Ontario Child Health Study Scales (Byles et al., 1988), the Children's Behaviour Questionnaire (Behar, 1977), and the Preschool Behaviour Questionnaire (Tremblay et al., 1992).

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The MIA is a 113-item scale aimed at measuring DSM-5 symptoms of internalizing and externalizing disorders over the last 12 months. Items pertaining to nonaggressive conduct problems (14 items, $\alpha = 0.84$, 0.79 for ages 15 and 17, respectively), physical aggression (12 items, $\alpha = 0.85$, 0.87 for ages 15 and 17, respectively), and social aggression (5 items, mean inter-item $r = 0.31$, 0.26 for ages 15 and 17, respectively) were used to create subscales. For both the SBQ and MIA, youth were asked to rate each item on a 3-point Likert scale from 1 = *Never/not true* to 3 = *Often/very true*. For main analyses, all items were standardized then averaged together. To support the assumption that items from the SBQ tap into the same antisocial constructs assessed in the MIA, several steps were taken (see Appendix S2 and S3). Whether forms of antisocial behaviors were best modeled as distinct or as a general antisocial factor was also assessed (see Analytic Plan section).

Control variables

Sex and income were included as covariates to control for their potentially confounding influence on antisocial behavior. Sex was parent-reported at birth and was coded as female = 0, male = 1. Income was parent-reported at age 13, 15, and 17 via the ICCQ on a 9-point Likert scale ranging from 1 = less than $10,000 to 9 = $100,000 or more annually. Income items were standardized, and across timepoints had a mean inter-item $r = .77$ and loaded > 0.9 onto one common factor via a principal component factor analysis. Parental education was evaluated as a potential control variable; however, it was not significantly related to any antisocial outcome when income was controlled, and thus was not included in the analytical models. Both household income and the mother's education at birth were used to evaluate potential biases in the selected genetic subsample and sample attrition.

Data analyses

Descriptive statistics and zero-order Spearman correlations were conducted on all study variables. Correlations between ASB-PRS and environmental factors were estimated to assess polygenic-environment associations (rGE), a known confounder for assessing genetic and environmental relations to phenotypes. The ASB-PRS_c and ASB-PRS_a were not significantly correlated. Model specification sensitivity testing was performed, whereby the ASB-PRS_a was removed from models to assess its influence on ASB-PRS_c (and vice versa). Removing each ASB-PRS did not substantially change the other's p-values for main findings. In concert with their low correlation with one another, this supports that these PRS have independent contributions to outcomes and, therefore, were included in models together.

Path models were then created using Mplus (v. 1.8.8) to address main research questions. First, to assess whether antisocial behaviors were best conceptualized as distinct forms or one uniform indicator of antisocial behavior, a model with one general antisocial factor (all antisocial scales included) was compared to a three-factor model (nonaggressive conduct, physical aggression, social aggression modeled separately). Stable forms of antisocial behavior were assessed as latent variables across timepoints, e.g., age 13, 15, and 17 physical aggression were regressed on a latent variable representing stable physical aggression. Forms of antisocial behavior were allowed to covary within each timepoint, e.g., age 13 physical aggression, age 13 social aggression, and age 13 nonaggression conduct covaried. Models were compared using the Satorra-Bentler Scaled Chi-Square Difference Test (Satorra & Bentler, 2010). To assess model fit, SRMR < .08, RSMEA < .06, CFI > .95 values were used to indicate models with acceptable fit (Hooper et al., 2007). To adjust for the increased risk of a false positive due to multiple comparisons, within each model we adjusted all p-values representing links between independent and dependent variables using the Benjamini and Hochberg (1995) method via the *R* package *stats (R Core Team, 2021)*.

Model 1: links to stable antisocial behaviors

The retained latent path model was then extended to examine how environmental, genetic, and control variables were associated with stable antisocial behaviors across adolescence (see Fig. 1 for illustration of model). Income, school violence, and harsh parenting were averaged across timepoints to assess level of exposure to these environmental factors across adolescence. To assess how the ASB-PRS moderated associations between home and school environment hostility and stable antisocial behaviors, four interaction terms were created by multiplying the relevant centered variables (e.g. ASB-PRS_c*school violence). Exogenous (independent) variables were allowed to covary. Latent antisocial variables were regressed on all independent variables. Models 1 and 2 were run in two steps: first with all controls and main predictors, then again but with interaction terms added. Lastly, multigroup path analyses were performed to assess whether sex moderated associations.

Models 2a,b,c: links to dynamic antisocial behaviors

Next, latent path models were respecified to assess which factors were associated with time-specific antisocial behaviors across adolescence (see Fig. 2 for example model). To assess dynamic antisocial behaviors, the model design was the same as model 1 except that variance not explained by stable antisocial behaviors were the dependent variables (i.e., latent variable residuals). Three models were created, one for each timepoint (models 2a, b, c for age 13, 15, and 17, respectively). Predictors were matched for each timepoint, e.g., predictors assessed at age 15 were included in the residual model for antisocial behaviors specific to age 15 (model 2b; see Appendix S4 and S5 for Mplus syntax of models).

Missing data

Participants that did not have genetic data available were significantly more likely to be male ($r = .10$, $p < .001$), have lower household income at birth ($r = -.08$, $p < .001$), age 13 ($r = -.08$, $p = .004$) and 15 ($r = -.06$, $p = .025$), and have mothers with less education at birth ($r = -.07$, $p < .001$). Within the subsample with genetic data, the attrition rate was 3% ($n = 18$) i.e., no data collected during adolescence. Each study variable had between 7% ($n = 57$; age 15 antisocial behavior) to 19% (149; age 17 school violence) missing data, with the exception of sex (complete data). At-birth household income and mother's education were not significantly related to adolescent attrition within the genetic subsample. Observed values for sex and all adolescent variables were significantly associated with data missingness for at least one time point; thus, full information maximum likelihood was used to handle missing data. Specifically, the maximum likelihood with robust standard errors estimator was used in all models, as research indicates this estimator can accommodate severe departure from normality as long as models have good fit indices and a sample size over 250 (Lai, 2018). All predictors/covariates except ASB-PRS (not related to data missingness) were specified to estimate missing data (Enders & Bandalos, 2001). Models were specified to include all participants that had genetic data available ($n = 721$).

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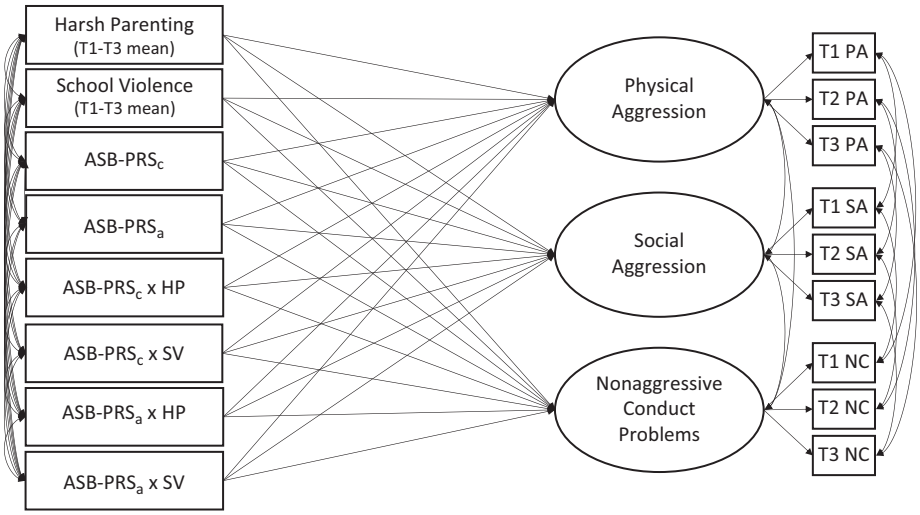

Figure 1. Model 1: risk factors predicting stable antisocial behaviors. Illustrates model 1 which assessed how genetic and environmental factors relate to forms of antisocial behavior stable across adolescence. The ASB-PRS$_c$ and ASB-PRS$_a$ were derived from the Pappa et al. (2016; child sample GWAS) and Tielbeek et al. (2022; adult sample GWAS), respectively. ASB-PRS = antisocial behavior polygenic risk score; HP = harsh parenting; SV = school violence; PA = physical aggression; SA = social aggression; NC = nonaggressive conduct problems; T1 = age 13; T2 = age 15; T3 = age 17.

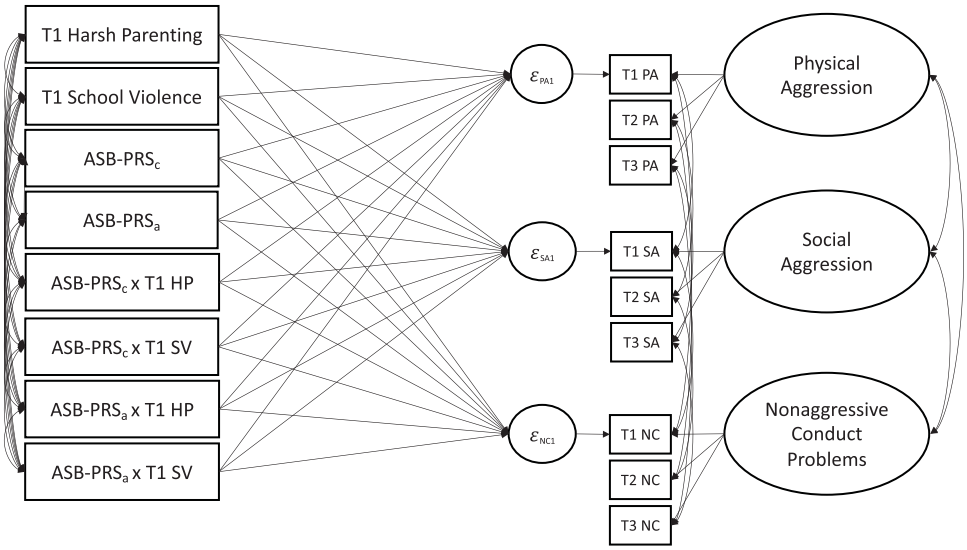

Figure 2. Model 2a: risk factors predicting antisocial behaviors specific (residual) to age 13. Illustrates model 2a which assessed how genetic and environmental factors relate forms of antisocial behavior that were specific to T1, i.e., antisocial variance at age 13 unexplained by stable behaviors. The ASB-PRS$_c$ and ASB-PRS$_a$ were derived from the Pappa et al. (2016; child sample GWAS) and Tielbeek et al. (2022; adult sample GWAS), respectively. ASB-PRS = antisocial behavior polygenic risk score; HP = harsh parenting; SV = school violence; PA = physical aggression; SA = social aggression; NC = nonaggressive conduct problems; T1 = age 13; T2 = age 15; T3 = age 17.

Results

Descriptive statistics

The means, SDs, ranges, and zero-order correlations for all study variables are provided in Tables 1 and 2 (see Table S1 for stability correlations of environmental predictors). Income and harsh parenting were strongly correlated between timepoints, while school violence was modestly correlated between timepoints. This indicates that school violence fluctuates over adolescence, while harsh parenting and income have greater stability. In the zero-order correlations, being reported as male at birth was associated with greater physical aggression at all timepoints and lower social aggression at age 13. Lower income, harsher parenting, and more reported school violence were significantly associated with higher antisocial behavior (all forms) across adolescence, with the exception of income and age 17 social aggression. Higher ASB-PRS$_c$ was associated with greater antisocial behaviors (all forms) at age 17, nonaggressive conduct problems and social aggression at age 15. ASB-PRS$_a$ was not significantly linked to any antisocial outcome. The strengths of relations between forms of antisocial behavior varied substantially across forms and timepoints ($r = .07–.56$). ASB-PRS were not significantly related to environmental factors, minimizing the confounding effect of underlying rGE.

Latent structure of antisocial behavior across adolescence

The Likelihood Ratio Test was significant for the analysis comparing a one vs. three antisocial behavior factor model and was nonsignificant when comparing models that were allowed to vary between sexes vs. not. This suggests that models that differentiated between forms of antisocial behavior — i.e., a three-factor model: physical aggression, social aggression, nonaggressive conduct problems — and that were invariant between sexes had significantly better model fit (see Table 3). The final model's fit indices were acceptable; thus, the three-factor model was retained for all subsequent analyses.

Genetic and environmental links to stable antisocial behaviors

Model 1 estimates and fit indices are included in Table 4. After adjusting for false discovery rate, ASB-PRS$_c$ was significantly associated with stable nonaggressive conduct problems, while ASB-PRS$_a$ was linked to stable physical aggression. However, both

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Table 1. Descriptive statistics by timepoint for study variables

	13-years-old (T1)				15-years-old (T2)				17-years-old (T3)			
	n	*M*	*SD/MAD*	Range	*n*	*M*	*SD/MAD*	Range	*n*	*M*	*SD/MAD*	Range
Income	1274	7.22	1.87	1.00–9.00	1383	7.28	1.84	1.00–9.00	1243	7.60	1.69	1.00–9.00
Harsh parenting	1290	1.85	0.49	1.00–3.75	1399	1.70	0.46	1.00–3.75	1252	1.56	0.43	1.00–3.50
School violence[b]	1224	1.00	0.00	1.00–5.00	1427	1.33	0.00	1.00–5.00	1206	1.00	0.00	1.00–5.00
Physical aggression[a,b]	1231	1.00	0.00	1.00–3.00	1443	1.00	0.33	1.00–2.92	1268	1.00	0.00	1.00–2.25
Social aggression[a]	1230	1.28	0.35	1.00–3.00	1443	1.38	0.34	1.00–3.00	1269	1.35	0.31	1.00–2.80
Nonaggressive conduct[a,b]	1231	1.00	0.00	1.00–2.83	1443	1.07	0.07	1.00–3.00	1269	1.07	0.07	1.00–2.62
ASB-PRS$_c$	721	396	0.89	394–399	–	–	–	–	–	–	–	–
ASB-PRS$_a$	721	309	0.56	307–311	–	–	–	–	–	–	–	–

Note. Unstandardized study variables by timepoint. Sex, income, and harsh parenting were caregiver-reported, while school violence and all antisocial behaviors were self-reported by the participating youth. Genetic data was collected at age 10. The ASB-PRS$_c$ and ASB-PRS$_a$ were derived from the Pappa et al. (2016) and Tielbeek et al. (2022) GWAS, respectively. *M* = mean or median, *SD* = standard deviation, *MAD* = median absolute deviation, *ASB-PRS* = antisocial behavior polygenic risk score. Data were compiled from the final master file of the Québec Longitudinal Study of Child Development (1998–2018), ©Gouvernement du Québec, Institut de la statistique du Québec.
[a]Antisocial behaviors were measured via the SBQ at T1 and the MIA at T2 and T3.
[b]Values were skewed (+/− 2): median and MAD were reported for these variables, respectively.

Table 2. Zero-order correlations for all study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Sex	–														
2. Income[a]	−.01	–													
3. Harsh parenting[a]	.08**	−.03	–												
4. School violence[a]	.06*	−.17***	.08**	–											
5. ASB-PRS$_c$	.02	.01	.05	.05	–										
6. ASB-PRS$_a$	−.07	.00	.02	-.06	.03	–									
7. T1 physical aggression	.24***	−.09**	.18***	.22***	.03	.01	–								
8. T2 physical aggression	.23***	−.13***	.14***	.23***	.04	.03	.32***	–							
9. T3 physical aggression	.16***	−.14***	.12***	.16***	.10*	.06	.22***	.37***	–						
10. T1 social aggression	−.08**	−.06*	.11***	.21***	−.01	.02	.36***	.15***	.07*	–					
11. T2 social aggression	.02	−.07**	.15***	.25***	.08*	.04	.25***	.42***	.25***	.32***	–				
12. T3 social aggression	.04	−.05	.16***	.22***	.09*	.07	.21***	.27***	.38***	.28***	.48***	–			
13. T1 nonagg. conduct	.04	−.07*	.14***	.21***	.04	.07	.43***	.23***	.17***	.35***	.24***	.22***	–		
14. T2 nonagg. conduct	.01	−.09***	.18***	.27***	.10*	.06	.23***	.42***	.26***	.26***	.53***	.38***	.33***	–	
15. T3 nonagg. conduct	.02	−.11***	.21***	.26***	.13**	.06	.17***	.27***	.36***	.20***	.36***	.49***	.26***	.56***	–

Note. Sex, income, and harsh parenting were caregiver-reported, while school violence and all antisocial behaviors were self-reported by youth. Sex was coded as assigned at birth as male = 1 and female = 0. Spearman correlation coefficients are reported; cases were excluded using pairwise method (*ns* = 612 to 1537). The ASB-PRSc and ASB-PRSa were derived from the GWAS Pappa et al. (2016) and Tielbeek et al. (2022), respectively. ASB-PRS = antisocial behavior polygenic risk score, T1 = age 13, T2 = age 15, T3 = age 17, nonagg. = nonaggressive. *$p < .05$, **$p < .01$, ***$p < .001$. Data were compiled from the final master file of the Québec Longitudinal Study of Child Development (1998–2018), ©Gouvernement du Québec, Institut de la statistique du Québec.
[a]Income, harsh parenting, and school violence were averaged across T1, T2, and T3 for this table.

scores had similar coefficient and p-values for physical aggression and nonaggressive conduct ($\beta = .08$–$.11$, $ps = .02$–$.05$) and each explained a similar amount of variance (~1%) of these two antisocial behaviors. ASB-PRS did not significantly moderate any environment-stable behavior links.

Several environmental and demographic associations with outcomes were found. Harsh parenting was significantly associated with higher stable levels of adolescent nonaggressive conduct problems and social aggression ($\Delta R^2 = .03$, $.02$, respectively). School violence and income were positively and negatively (respectively) associated with stable physical aggression, social aggression, and nonaggressive conduct problems (school violence $\Delta R^2 = .11$, $.11$, $.14$; income $\Delta R^2 = .03$, $.01$, $.02$, respectively). Assigned male-at-birth was significantly positively associated with stable physical aggression ($\Delta R^2 = .04$).

Genetic and environmental links to time-specific (residual) variance in antisocial behaviors

Models 2a-c estimates and fit indices are included in Table 5. After adjusting for multiple comparisons, ASB-PRS were not significantly associated with any form of time-specific antisocial behavior. However, interestingly both ASB-PRS showed a significant consistent polygenic-environment interaction prior to p-value

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Table 3. Likelihood ratio tests for comparing stable antisocial behaviors models

		SRMR	RMSEA	CFI	*df*	AIC	BIC	χ^2	χ^2 Δ	*df* Δ	*p*
One vs. three factor models for antisocial behavior	One latent factor	.03	.04	0.88	314	−37,377	−36,674	586			
	Three latent factors	**.02**	**.02**	**0.98**	**251**	**−37,574**	**−36,782**	**295**	**291**	**63**	**< .001**
Multigroup model by sex	Constrained model	.04	.03	0.92	594	−38,629	−37,288	807			
	Unconstrained model	.03	.03	0.93	498	−38,599	−37,124	700	107	96	.113

Note. Model fit indices were compared in (1) a one (overall antisocial behavior) vs. three (social aggression, physical aggression, and nonaggressive conduct problems) latent factor model and (2) a model that constrained relations to be the same between sexes vs. a model that allowed relations to vary by sex. Model comparisons were performed using Satorra-Bentler Scaled Chi-Square Difference Tests, models with significant improvements in fit indices were bolded. The 3-factor model of antisociality and a model where latent factor and regression coefficients were *not* allowed to vary between sexes were supported. SRMR = standardized root mean squared residual; RMSEA = root mean square error of approximation; CFI = comparative fit index; DF = degrees of freedom; AIC = akaike information criterion; BIC = sample-size adjusted bayesian information criterion. Data were compiled from the final master file of the Québec Longitudinal Study of Child Development (1998–2018), ©Gouvernement du Québec, Institut de la statistique du Québec.

Table 4. Model 1: latent path model for stable antisocial behaviors across adolescence

		Nonagg. conduct problems (R^2 = 0.30)				*Physical aggression* (R^2 = 0.29)				*Social aggression* (R^2 = 0.24)			
	Predictors	*β*	*SE*	95% CI	*p*	*β*	*SE*	95% CI	*p*	*β*	*SE*	95% CI	*p*
Step 1	Sex	−0.05	.039	[−0.10, 0.03]	.248	**0.16**	**.051**	**[0.03, 0.18]**	**.001**	−0.07	.041	[− 0.09, 0.01]	.088
	Income	**−0.14**	**.045**	**[−0.11, − 0.02]**	**.002**	**−0.17**	**.048**	**[−0.10, − 0.01]**	**< .001**	**−0.13**	**.045**	**[−0.07, −0.01]**	**.004**
	Harsh parenting	**0.16**	**.046**	**[0.05, 0.20]**	**.001**	0.09	.037	[0.00, 0.10]	.020	**0.15**	**.042**	**[0.03, 0.13]**	**< .001**
	School violence	**0.40**	.066	**[0.15, 0.38]**	**< .001**	**0.35**	**.075**	**[0.09, 0.26]**	**< .001**	**0.36**	**.061**	**[0.09, 0.25]**	**< .001**
	ASB-PRS$_c$	**0.11**	**.045**	**[0.00, 0.10]**	**.017**	0.09	.048	[− 0.01, 0.07]	.052	0.07	.044	[0.00, 0.05]	.091
	ASB-PRS$_a$	0.08	.040	[0.00, 0.12]	.051	**0.10**	**.040**	**[0.01, 0.09]**	**.016**	0.05	.041	[−0.02, 0.07]	.195
Step 2	ASB-PRS$_c$ x HP	0.04	.043	[−0.05, 0.12]	.390	0.01	.043	[−0.05, 0.07]	.788	0.04	.049	[−0.04, 0.09]	.473
	ASB-PRS$_c$ x SV	0.07	.077	[−0.06, 0.16]	.362	0.10	.084	[−0.04, 0.15]	.255	0.00	.060	[−0.06, 0.06]	.979
	ASB-PRS$_a$ x HP	0.04	.044	[−0.07, 0.18]	.392	0.06	.042	[−0.02, 0.15]	.177	0.06	.042	[−0.03, 0.15]	.176
	ASB-PRS$_a$ x SV	0.06	.098	[−0.15, 0.28]	.561	0.15	.110	[−0.03, 0.29]	.177	−0.01	.076	[−0.13, 0.12]	.941

Note. Latent path model estimates for youths' genetic, environmental, and stable antisociality. Genetic ancestry scores were included as covariates. Model fit indices: RMSEA = .02, CFI = .98, SRMR = .02, $\chi^2(251, N = 721) = 295$, $p = .03$. Bolded values indicate $p < .05$ after false discovery rate was adjusted. ASB-PRS = antisocial behavior polygenic risk score, HP = harsh parenting, SV = school violence, β = standardized beta, SE = standardized standard error, CI = confidence interval. The ASB-PRS$_{c,a}$ were derived from the GWAS Pappa et al. (2016; child population) and Tielbeek et al. (2022; adult population), respectively. Data were compiled from the final master file of the Québec Longitudinal Study of Child Development (1998–2018), ©Gouvernement du Québec, Institut de la statistique du Québec.

adjustment (model 2c). At age 17 and in youth with lower ASB-PRS (> 0.5 SD below mean), harsher parenting was significantly associated with lower social aggression (see Figure S1 for Johnson Neyman graphs showing regions of significance). Conversely, in those with higher ASB-PRS (> 2 SDs above mean), harsher parenting was significantly associated with higher social aggression (Fig. 3; $\Delta R^2 = .04, .04$).

There were also two environmental and demographic links to time-specific outcomes. Being male was significantly positively associated with higher physical aggression specifically at age 13 (model 2a; $\Delta R^2 = .04$) and school violence was positively associated with nonaggressive conduct problems at specifically age 17 (model 2c; $\Delta R^2 = .05$).

Discussion

The present study employed longitudinal latent analyses to assess how genetics and environmental hostility interact to affect stable and dynamic antisocial behaviors across adolescence. We assessed two antisocial-linked PRS, two forms of environmental hostility (harsh parenting and school violence), and three forms of antisocial behavior (physical aggression, social aggression, and nonaggressive conduct problems) across three timepoints (age 13, 15, 17 years) in a population-based sample.

Our findings support that forms of antisocial behavior should be treated as distinct, but related, constructs. We identified associations between genetics and environment and stable antisocial behavior. Polygenic risk for antisociality also moderated the association between environmental hostility and late adolescent changes in antisocial behavior. These findings contribute several novel insights into the extent to which genes and key environmental adversity factors independently and jointly contribute to distinct forms of antisocial behavior in youth.

Polygenic-environment interactions and antisocial behavior

Consistent with our expectations, one of the antisocial-linked PRS (ASB-PRS$_a$) was significantly associated with only stable physical aggression. The other antisocial PRS (ASB-PRS$_c$) was significantly associated with only stable nonaggressive conduct problems. However, both antisocial PRS had similar relation estimates and explained similar modest amounts of variance (~1% each) of both physical aggression and nonaggressive conduct. This suggests that nonaggressive conduct problems and physical aggression share some genetic overlap that is distinct from social aggression.

Contrary to our expectations, we did not find support that the selected polygenic scores moderated relations between environment and stable antisocial behavior. Although the two GWASs

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Table 5. Models 2a,b,c: latent path model for time-specific (i.e., residual) antisocial behaviors across adolescence

		Nonagg. conduct (R^2 = .08$_{T1}$, .20$_{T2}$, .14$_{T3}$)				*Physical agg.* (R^2 = .14$_{T1}$, .17$_{T2}$, .13$_{T3}$)				*Social agg.* (R^2 = .08$_{T1}$, .12$_{T2}$, .20$_{T3}$)			
	Predictors	*β*	*SE*	95% CI	*p*	*β*	*SE*	95% CI	*p*	*β*	*SE*	95% CI	*p*
Model 2a: Age 13 (T1)	Sex	0.05	0.04	[−0.04, 0.13]	.286	**0.18**	**0.04**	**[0.10, 0.26]**	**< .001**	−0.07	0.04	[−0.15, 0.01]	.068
	Income	−0.02	0.04	[−0.11, 0.06]	.611	−0.06	0.05	[−0.14, 0.03]	.223	−0.05	0.05	[−0.14, 0.04]	.255
	Harsh parenting	0.11	0.05	[0.00, 0.21]	.048	0.13	0.05	[0.04, 0.22]	.005	0.09	0.05	[−0.01, 0.18]	.073
	School violence	0.11	0.07	[−0.02, 0.24]	.090	0.20	0.07	[0.06, 0.33]	.005	0.14	0.06	[0.03, 0.25]	.014
	ASB-PRS$_c$	0.08	0.04	[0.00, 0.15]	.046	0.02	0.04	[−0.06, 0.10]	.602	−0.04	0.04	[−0.12, 0.05]	.376
	ASB-PRS$_a$	0.04	0.04	[−0.04, 0.11]	.339	0.06	0.04	[−0.02, 0.13]	.133	−0.02	0.04	[−0.10, 0.05]	.506
	ASB-PRS$_c$ x HP	0.00	0.06	[−0.11, 0.11]	.987	−0.02	0.05	[−0.12, 0.07]	.633	0.00	0.05	[−0.10, 0.10]	.978
	ASB-PRS$_c$ x SV	0.06	0.06	[−0.06, 0.17]	.318	−0.03	0.08	[−0.19, 0.12]	.691	0.01	0.05	[−0.09, 0.10]	.918
	ASB-PRS$_a$ x HP	−0.05	0.04	[−0.12, 0.03]	.255	0.00	0.05	[−0.09, 0.10]	.979	0.01	0.04	[−0.07, 0.09]	.773
	ASB-PRS$_a$ x SV	0.04	0.06	[−0.08, 0.15]	.543	0.04	0.05	[−0.05, 0.14]	.362	0.02	0.05	[−0.07, 0.11]	.701
Model 2b: Age 15 (T2)	Sex	−0.05	0.08	[−0.20, 0.10]	.538	−0.24	0.15	[−0.54, 0.06]	.114	0.03	0.06	[−0.09, 0.16]	.612
	Income	0.17	0.09	[0.00, 0.35]	.056	0.14	0.12	[−0.10, 0.37]	.254	0.02	0.07	[−0.11, 0.16]	.765
	Harsh parenting	−0.21	0.10	[−0.40, −0.02]	.032	0.01	0.07	[−0.13, 0.14]	.942	−0.04	0.07	[−0.18, 0.09]	.516
	School violence	0.16	0.14	[−0.11, 0.42]	.245	0.06	0.19	[−0.31, 0.43]	.735	0.08	0.11	[−0.13, 0.29]	.455
	ASB-PRS$_c$	0.00	0.06	[−0.11, 0.11]	.945	0.03	0.06	[−0.08, 0.15]	.545	0.04	0.05	[−0.06, 0.14]	.465
	ASB-PRS$_a$	0.04	0.05	[−0.06, 0.14]	.467	0.00	0.05	[−0.10, 0.11]	.965	0.03	0.05	[−0.07, 0.12]	.600
	ASB-PRS$_c$ x HP	0.02	0.08	[−0.13, 0.18]	.757	0.09	0.09	[−0.09, 0.26]	.338	−0.08	0.06	[−0.20, 0.05]	.222
	ASB-PRS$_c$ x SV	−0.16	0.14	[−0.44, 0.12]	.251	−0.13	0.19	[−0.50, 0.24]	.500	−0.06	0.08	[−0.22, 0.11]	.507
	ASB-PRS$_a$ x HP	0.02	0.07	[−0.11, 0.15]	.721	0.05	0.06	[−0.07, 0.18]	.402	−0.04	0.06	[−0.15, 0.07]	.445
	ASB-PRS$_a$ x SV	0.02	0.15	[−0.27, 0.32]	.876	−0.07	0.20	[−0.47, 0.32]	.713	−0.11	0.07	[−0.25, 0.04]	.148
Model 2c: Age 17 (T3)	Sex	−0.01	0.05	[−0.12, 0.09]	.821	−0.02	0.09	[−0.21, 0.16]	.848	0.02	0.06	[−0.10, 0.15]	.704
	Income	−0.01	0.07	[−0.15, 0.14]	.897	0.08	0.11	[−0.13, 0.30]	.481	0.11	0.08	[−0.05, 0.28]	.183
	Harsh parenting	0.04	0.05	[−0.06, 0.14]	.401	−0.14	0.08	[−0.30, 0.02]	.099	−0.05	0.09	[−0.24, 0.14]	.610
	School violence	**0.24**	**0.07**	**[0.10, 0.39]**	**.001**	0.17	0.09	[0.00, 0.34]	.052	0.12	0.09	[−0.05, 0.29]	.173
	ASB-PRS$_c$	0.08	0.04	[−0.06, 0.15]	.062	0.03	0.04	[−0.11, 0.09]	.521	0.03	0.05	[−0.11, 0.12]	.483
	ASB-PRS$_a$	0.02	0.04	[−0.10, 0.09]	.708	0.07	0.04	[−0.07, 0.15]	.097	0.01	0.05	[−0.11, 0.11]	.755
	ASB-PRS$_c$ x HP	−0.02	0.05	[−0.12, 0.08]	.641	0.01	0.05	[−0.08, 0.10]	.885	0.19	0.07	[0.05, 0.32]	.008
	ASB-PRS$_c$ x SV	0.19	0.07	[0.05, 0.33]	.009	0.21	0.10	[0.02, 0.40]	.034	−0.02	0.07	[−0.15, 0.12]	.802
	ASB-PRS$_a$ x HP	0.02	0.06	[−0.09, 0.13]	.693	0.06	0.05	[−0.03, 0.15]	.212	0.19	0.07	[0.06, 0.32]	.004
	ASB-PRS$_a$ x SV	−0.01	0.09	[−0.18, 0.17]	.954	0.09	0.12	[−0.14, 0.32]	.444	0.11	0.07	[−0.03, 0.26]	.132

Note. Latent path model estimates for youths' genetic, environmental, and time−specific antisocial behaviors. Model fit indices: RMSEAs = .02, CFI = .97.98, SRMRs = .02, $\chi^2(167, N = 721) = 213, 234, 229$ (respectively), *ps* < .05. Bolded values indicate < .05 after false discovery rate was adjusted. Interactions terms were added in a second step in the model. The ASB − PRS$_{c,a}$ were derived from the GWAS Pappa et al. (2016; child population) and Tielbeek et al. (2022; adult population), respectively. Agg = aggression, ASB − PRS = antisocial behavior polygenic risk score, HP = harsh parenting, SV = school violence, β = standardized beta, SE = standardized standard error, CI = confidence interval. Data were compiled from the final master file of the Québec Longitudinal Study of Child Development (1998–2018), ©Gouvernement du Québec, Institut de la statistique du Québec.

used to derive the present PRS were the largest to-date, sample sizes between 19,000 and 85,000 are still relatively small; a larger GWAS sample increases statistical power, especially for lower-frequency variants (Ioannidis et al., 2009). Thus, larger GWAS on antisocial behavior would likely result in a PRS with greater predictive power than used here. Additionally, genome-wide complex trait analysis heritability estimates in the published GWAS ranged from 10% and 54% depending on the cohort, suggesting substantial phenotypic heterogeneity; the performance of these PRS likely vary substantially based on sample characteristics. The present study provided meaningful insights into the effectiveness of these antisocial PRS, which is critical for moving the field forward.

Unexpectedly, we also identified a novel time-specific link. Polygenic risk for antisocial behavior may result in differential susceptibility to home environment in late adolescence. Specifically, those with relatively high genetic risk for antisociality were more likely to exhibit social aggression when exposed to harsher parenting, but also showed relatively lower social aggression when exposed to less harsh parenting. Interestingly, although a harsher parenting style related to greater stable social aggression in youth, harsher parenting resulted in a dip in social aggression in older adolescents who had lower genetic antisocial risk. This finding was consistent between the two tested antisocial PRS; each explained a notable amount of social aggression (~4%)

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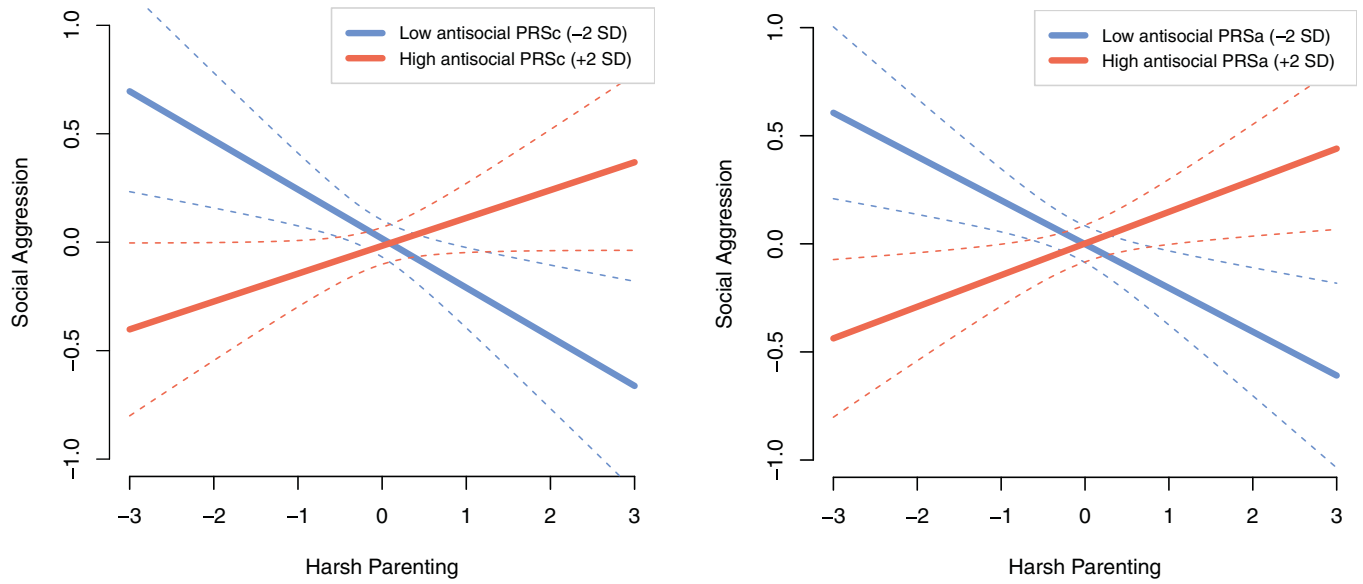

Figure 3. Polygenic-environment interaction in late adolescence. Plots illustrate the relations between harsh parenting (standardized) and time-specific social aggression at age 17 (i.e., residual variance) with high or low (+/− 2 SD) antisocial behavior polygenic risk scores (ASB-PRS). The ASB-PRS$_c$ and ASB-PRS$_a$ were derived from the Pappa et al. (2016; child sample GWAS) and Tielbeek et al. (2022; adult sample GWAS), respectively. Johnson Neyman plots (see Figure S1) indicate the negative (blue) and positive (red) relations between harsh parenting and social aggression are significant when PRS are less than approximately 0.5 SDs below and 2 SDs above the mean, respectively. Each plot was computed using a trimmed model which includes only significant predictors from model 2c; ABS-PRS$_c$ and ABS-PRS$_a$ were not included in the same model for computing these plots. SD = standard deviation. Data were compiled from the final master file of the Québec Longitudinal Study of Child Development (1998–2018), ©Gouvernement du Québec, Institut de la statistique du Québec.

specific to age 17. Perhaps, the genetic variants included in the PRS also influence other traits that then affect the expression of antisocial behavior in unexpected ways. For example, youth with a lower genetic risk may become more inhibited and withdrawn when harshly punished, reducing both prosocial and antisocial interactions. This may be specific to late adolescence due to normative increases in their abilities to inhibit base impulses (Hammond et al., 2012). Alternately, they may know they are nearing independence from their parents and are thus more willing to temporarily modify their behavior to reduce conflict in the home. However, these are speculative explanations for this unanticipated finding; further research will need to replicate this relation, understand the mechanisms involved, and determine whether this change in social aggression is temporary or sustained into adulthood.

This polygenic-environment link is complimentary to much of the previous literature reporting that a combination of high polygenic and environmental risk associates to greater antisocial problems (Barnes et al., 2019; Salvatore et al., 2015; Womack et al., 2021). Similar to our finding that low genetic risk combined with an adverse environment may increase antisocial behavior, Musci et al. (2019) reported that middle schoolers with a *lower* PRS for conduct problems were more impulsive-aggressive when exposed to community violence. Perhaps this means that youth with low antisocial genetic risk respond to their environmental dynamics in ways that do not follow our established understanding of these processes. These type of complex, temporally sensitive interactions could help explain why some youth respond in opposite ways to the same stimuli. This research highlights the need for further studies on how environment and genetics interact to explain the substantive heterogeneity and instability of antisocial behavior across development.

Interestingly, we found that the PRS used in the present study were not significantly correlated and each explained unique portions of antisocial behavior. Each PRS was derived from a GWAS with very different methodologies (e.g., parent- vs. self-reports/records and childhood vs. adulthood). This could have implications for the PRS literature more broadly, in that different methodologies may capture different facets of antisocial genetics. Antisocial behaviors fluctuate across contexts and developmental periods. Thus, selecting for genes that are predictive of antisociality across discrepant methodologies (e.g. meta-GWAS) may limit potential for identifying genetic contributions to antisociality. This could explain why antisocial PRS are most predictive when studies use methodologies that are more closely in-line with the original GWAS (e.g., Kretschmer et al., 2021).

Environment and antisocial behavior across adolescence

We expected based on past twin studies that environmental factors would be especially influential on youths' nonaggressive conduct problems (Burt, 2012), which was partially supported by our findings. Harsh parenting was significantly associated with both nonaggressive conduct problems and social aggression, but not physical aggression. This is suggestive of social aggression and nonaggressive problems having greater overlap in adolescent environmental etiologies. Although generally in-line with our expectations, this result is in contrast to one of the few studies that have explored a similar question; Becht et al. (2016) reported that harsh parenting (yelling at, insulting, or hitting child) predicted a high aggression trajectory over childhood and adolescence (age 9 to 15), but not rule-breaking. These contrasting results could be seen as support for other research showing that shared environmental factors have less influence on physical aggression *after* childhood (Burt, 2009). Albeit, other researchers have reported the converse (Eley et al., 2003).

Further complicating matters, parenting can also be a part of children's nonshared environment; parents may treat siblings

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differently and each child may have a unique perspective on similar parenting practices (Turkheimer & Waldron, 2000). If the influence of parenting on aggression decreases after childhood, perhaps early hostile parenting increases early-onset physical aggression making whether one exhibits chronically high physical aggression in adolescence a result of childhood factors. Thus, parenting may no longer influence physical aggression in adolescence, but would still affect other more malleable antisocial behaviors.

Although more violent school environments were associated with all forms of stable antisocial behaviors, school violence explained 3% more variance in stable nonaggressive conduct problems than other forms antisocial behavior and was related to time-specific increased nonaggressive problems in late adolescence. This provides some support for environmental hostility being especially related to stable and dynamic nonaggressive problems, which partially supports our initial hypothesis. We also expected that environment would only be related to stable physical aggression among genetically susceptible youth, which was not supported by our results. Interestingly, school violence explained a substantial amount of variance for all antisocial behaviors relative to harsh parenting (e.g., $R^2 = .14$ vs. .03 for stable nonaggressive conduct problems). Family-level factors decrease in influence on rule-breaking and aggression as children age, which may partially explain this discrepancy (Harden et al., 2015). It is also possible that more violent youth are more aware of violent behavior at the school as they enact some of this violence and/or associate with more antisocial peers. The magnitude of this relation may also have been affected by school violence and antisocial behavior both being self-reported measures (i.e., shared informant bias). To our knowledge, we are the first to investigate how school violence may relate to distinct forms of antisocial behavior across adolescence. Thus, more research encompassing teacher- and peer-reports is needed to help elucidate why school violence explained such a large proportion of antisocial behavior.

Limitations and strengths

Some of the strengths and limitations of our study have already been discussed above. In our model, we conducted analyses that allowed us to differentiate between behaviors that were stable across adolescence, as opposed to time-specific behavior. Understanding what factors (using two informants and biological data) are associated with changes in antisocial behavior is an understudied area, thus we were able to provide novel insights into the dynamics of antisocial behavior over a critical developmental period. Additionally, multi-informant, multi-timepoint study designs are considered best practice for developmental research, which is a strength of this study. However, we used two different antisocial scales for age 13 vs. 15 and 17 years. They had similar items, Likert scales, and physical aggression scales were similarly correlated with one another — e.g., correlations between ages 13 and 15 vs. 15 and 17 $r = .32$ vs. .37 — however, ages 15 and 17 were ~ 0.2 more correlated than ages 13 and 15 in nonaggressive conduct problems and social aggression scales. It is unclear whether this is due to scale differences or developmental dynamics. Thus, the sex relation to antisocial behavior specific to age 13 should be treated with caution. Further research is needed to replicate our work to determine whether developmental dynamics had an impact on findings.

Although we used multiple informants for this study, the choice of informants could have impacted the results. For example, we asked informants to report on their own behavior, i.e., parents self-reported their harsh parenting behavior. Self-reports may lead to under-reporting of problem behaviors due to social desirability biases. To tease out how these informant choices may have affected our findings, research incorporating multiple informants for each measure is needed.

As is best practice, we adjusted our alpha significance threshold to reduce the likelihood of reporting a false positive. However, such adjustments can increase the likelihood of a false negative. More research is needed to discern whether our adjustments were overly conservative. As the original Pappa et al. (2016) and Tielbeek et al. (2022) GWAS were conducted on a European sample, we restricted our sample to include only those of European decent. This has the benefit of maximizing the predictive power of the PRS and avoiding interpretation problems that can occur if the PRS is not generalizable to differing ethnicities (Ioannidis et al., 2009; Martin et al., 2017). However, this means that our findings likely will not generalize to individuals of differing ethnic backgrounds. Lastly, our findings are correlational, and although our models are built on directional hypotheses, we are ultimately unable to determine whether identified associations are causal. For example, Haan et al. (2012) reported that the relation between overreactive parenting and antisocial behavior in adolescence is reciprocal, thus engaging in antisocial behavior more frequently may also lead parents to use more harsh parenting strategies. Similarly, coercion theory states that problem behavior and harsh child-parent interactions feed into each other, and this dynamic begins early in life (Shaw et al., 2000; Sitnick et al., 2015). Thus, investigations starting in early childhood may be needed to tease apart the long-term interactions between harsh parenting and antisociality.

Conclusion

Our findings indicate independent genetic and environmental contributions to stable adolescent antisocial behavior and the presence of a polygenic-environment interaction in late adolescence. Specifically, amongst late adolescents with higher genetic risk for antisociality, harsher parenting was associated with higher social aggression in late adolescence. Alternately among late adolescents with lower genetic risk for antisociality, harsher parenting was linked to lower social aggression. Harsher parenting was also associated with greater stable social aggression and nonaggressive conduct problems across adolescence. School violence was not moderated by antisocial PRS but was independently associated with all forms of stable antisocial behavior, especially nonaggressive problems at age 17. Together, this suggests that genetics and environmental hostility link to stable youth antisocial behaviors, whereas genetic-environment interactions may be more influential on late adolescent changes in antisocial behavior. This research highlights the interplay between biological and environmental factors and how they may be critical for understanding stable and fluctuating antisocial behaviors over development.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S095457942400004X.

Data availability statement. The data analyzed in this study was obtained from the Institut de la statistique du Québec and, as stipulated in the clauses 10 and 11 of the Institut de la statistique's Québec Act (Canada), the access to the data is restricted to the parties identified in the partnership agreement signed to ensure the conduct of the study and which describes the author's right. In the QLSCD cohort, the participants only consented to share their data with the

study's financial partners and affiliated researchers and their collaborators. Those partners and researchers only have access to the data after signing a data sharing agreement. Requests to access these data can be directed to the Institut de la statistique du Québec's Research Data Access Services - Home (www.quebec.ca). For more information, contact Marc-Antoine Côté-Marcil (SAD@stat.gouv.qc.ca).

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