



# Developmental Trajectories of Delinquent and Aggressive Behavior: Evidence for Differential Heritability

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## Abstract

The developmental course of antisocial behavior is often described in terms of qualitatively distinct trajectories. However, the genetic etiology of various trajectories is not well understood. We examined heterogeneity in the development of delinquent and aggressive behavior in 1532 twin youth using four waves of data collection, spanning ages 9–10 to 16–18. A latent class growth analysis was used to uncover relevant subgroups. For delinquent behavior, three latent classes emerged: Non-Delinquent, Low-Level Delinquent, and Persistent Delinquent. Liability for persistent delinquency had a substantial genetic origin (heritability = 67%), whereas genetic influences were negligible for lower-risk subgroups. Three classes of aggressive behavior were identified: Non-Aggressive, Moderate, and High. Moderate heritability spanned the entire continuum of risk for aggressive behavior. Thus, there are differences between aggressive behavior and non-aggressive delinquency with respect to heterogeneity of etiology. We conclude that persistent delinquency represents an etiologically distinct class of rule-breaking with strong genetic roots.

**Keywords** Delinquency · Aggression · Developmental trajectory · Heritability

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## Introduction

Antisocial behavior is a broad term that encompasses delinquent and aggressive behaviors. Delinquent behaviors are generally defined as those which violate societal norms, values and laws, whereas aggressive behaviors are defined as those which hurt, harm or injure another [1–4]. Research has shown that early behavior problems strongly predict future antisocial behavior, and are associated with a wide range of negative outcomes, including academic underachievement, unemployment, psychiatric illness, substance abuse, and unstable relationships [5–8]. Thus, antisocial behavior constitutes both an economic and a social burden to individuals, their families, and entire communities [9]. Understanding the developmental patterns of antisocial behavior from childhood through adolescence can inform intervention efforts and help reduce the aforementioned burdens.

The relationship between age and antisocial behavior is well known. Overall, antisocial behavior tends to increase in early adolescence, reach a peak in mid-adolescence and then decline, at first quickly, but then more gradually. This relationship between age and antisocial/criminal behavior has been evident for several decades and across diverse demographic samples [10–13]. Even though the relationship

between age and antisocial/criminal behavior is evident, antisocial behavior in childhood will not always result in antisocial behavior during adolescence; not all antisocial youths continue to engage in such acts during adulthood, and the majority of individuals who have previously engaged in antisocial behavior seem to desist during late adolescence or early adulthood.

## Developmental Trajectories

These developmental patterns form the basis for Moffitt's famous taxonomy of a life-course persistent trajectory versus an adolescent-onset antisocial trajectory in which *age-of-onset* is the key [14]. Similar to Moffitt's developmental taxonomy, an earlier theory by DiLalla and Gottesman [15] also focuses on age-of-onset. They differentiate among three trajectories: 'continuous antisocials'—delinquent youths who continue to be criminal as adults; transitory delinquents—adolescent delinquents who desist from crime in adulthood; and 'late bloomers', who begin offending in adulthood.

Other developmental theories posit different trajectories that describe when youth develop (and desist from) antisocial behavior [16, 17]. Newer models focus on *antisocial behavioral subtypes*, namely aggressive behavior versus delinquent/rule-breaking behavior [4, 18, 19]. For example, Loeber and Hay [20] have suggested two pathways: *the overt pathway*—that escalates across time from minor aggression (e.g., annoying others, bullying), to physical fighting (e.g., gang fighting), and lastly into full-blown violence (e.g., rape, aggravated assault, robbery)—and *the covert pathway*, which constitutes minor covert behaviors (e.g., shop-lifting and lying), property damage (e.g., vandalism and fire-setting), and later serious delinquency (e.g., fraud, burglary, and major theft).

The presence of distinct trajectories of antisocial behavior has received much empirical support [21–30]; for an exception see Walters [31]. Some of the identified trajectories include 'stable high' (childhood onset/life course persistent), 'decreasing' (childhood limited), 'increasing' (adolescent onset/transitory), 'stable low' (low offending across development/chronic low level), and 'abstainers' (those who refrain from antisocial and criminal activities throughout the life-course). Although it has been suggested that these developmental trajectories may not be analogous in females [32], there is substantial evidence that females follow similar trajectories as males. A comprehensive review of 46 studies identified early-onset/life-course-persistent, childhood-limited, adolescence-limited, and adulthood onset trajectories in females, and concluded that these trajectories were similar to the ones previously identified in males [33].

Further refinement according to the subtype of antisocial behavior have illuminated differences in developmental course. Studies exclusively examining aggressive behavior

have typically found a decreasing (i.e., childhood-limited) group [23, 34, 35], whereas delinquent behavior is found to increase during adolescence in parallel with the onset of puberty [7]. These findings suggest that aggressive and delinquent behavior follow different developmental trajectories. The precise number of antisocial trajectories varies from one study to another, depending on the subtype under examination (i.e., aggressive vs. delinquent behavior) and the design of the study (e.g., self-reported vs. caregiver-reported measures; prospective longitudinal vs. retrospective).

Various developmental trajectories have been found to be associated with different underlying risk factors and outcomes. The stable high (i.e., childhood onset/life course persistent) trajectory has been associated with early neurological impairments, cognitive difficulties, and severe family dysfunction, and also carries poor long-term outcomes such as school drop-out and incarceration [8, 24]. In contrast, increasing (i.e., adolescent onset/transitory) antisocial behavior coincides with the onset of puberty, and is thought to be a relatively normative response to the "maturity gap"—the "role-less" years between biological maturation and access to mature privileges and responsibilities [29, 36]. These rebellious adolescents are often poorly monitored by their parents but eventually snap out of their antisocial activities by late adolescence or early adulthood. However, there is also evidence that adolescent-onset antisocial behavior is not entirely without risk—some individuals on this track will continue to exhibit antisocial behavior due to "developmental snares" (e.g., addiction, unwanted pregnancy, and incarceration) [24, 25]. More recent literature indicates that adolescent-limited offenders exhibit poorer psychological functioning (including greater negative emotionality) in adulthood relative to nonoffenders [37].

The 'stable low' pattern has been associated with mental health problems, whereas abstainers (those who refrain from antisocial and criminal activities throughout the life-course) were originally hypothesized to possess unappealing personality characteristics that prevent them from taking part in normative peer social groups during adolescence [25]. The latter perspective has been challenged by evidence, to the contrary, that abstainers are characterized by superior social adjustment and mental health [38, 39]. Decreasing (childhood-limited) antisocial behavior has been found to have similar risk factors and long-term prognosis as the stable low group [25].

## Genetic and Environmental Etiology

Twin studies provide evidence that genetic influences contribute more strongly to stable high (childhood onset/life course persistent), than to increasing (i.e., adolescent onset/transitory) antisocial behavior [40–42]. According to a meta-analysis, the underlying etiologies of aggressive

and delinquent behavior differ in two key ways: aggressive behavior is more heritable than rule-breaking (delinquent) behavior (65% vs. 48%), and rule-breaking behavior is influenced by the family-wide environment (18% of the total variance explained) while aggressive behavior is not [43, 44]. Thus, aggressive behavior is highly heritable, whereas delinquent behavior is influenced by both genetic and family-wide environmental factors.

Aggressive behavior is quite prevalent in early childhood. Those that display aggressive behavior early typically continue to display aggressive behavior throughout the lifespan. In contrast, delinquent behavior is most prevalent during adolescence and shows lower levels of stability than aggressive behavior [44, 45]. Despite what is known about the distinct developmental courses of these antisocial subtypes, very few studies have examined the degree of genetic liability for various trajectories of delinquent and aggressive behavior.

The present study builds on previous research by identifying and exploring the etiological basis of developmental trajectories of delinquent and aggressive behavior across four waves of data collection (from ages 9–10 to 16–18 years) in a large community sample of twins. Previous research has identified around five different trajectories of antisocial behavior: stable high (childhood onset/life course persistent), decreasing (childhood limited), increasing (adolescent onset/transitory), stable low, and abstainers (those who refrain from antisocial and criminal activities throughout the life-course). Here, we explore whether we can identify similar trajectories of delinquent and aggressive behavior using an empirical approach: latent class growth analysis. This mixture modeling technique is appropriate for longitudinal data and is designed to uncover the existence of subgroups (classes) in a population. Given that previous research has demonstrated that delinquent and aggressive behavior are influenced by genetic factors [43, 44], we also sought to ascertain the degree of genetic influence on any emerging classes. We hypothesized that genetic factors are more important in explaining twin concordance for aggressive-behavior subgroups than for delinquent-behavior subgroups.

## Method

### Participants

The current sample consists of 1532 participants (766 same-sex and opposite-sex twin pairs, 49.7% male) from the University of Southern California (USC) Risk Factors for Antisocial Behavior (RFAB) twin study. This study is a prospective investigation of the interplay of genetic, environmental, social, and biological risk factors on the development of antisocial and aggressive behaviors

from childhood to emerging adulthood [46]. Participating families were recruited from the greater Los Angeles community, and the sample is representative of the ethnic and socioeconomic diversity of the area. During Wave 1, participants were 9–10 years old ( $M=9.59$ ,  $SD=0.58$ ); at Wave 2, 11–13 years old ( $M=11.79$ ,  $SD=0.92$ ); at Wave 3, 14–15 years old ( $M=14.82$ ,  $SD=0.83$ ); at Wave 4, 16–18 years old ( $M=17.22$ ,  $SD=1.23$ ).

Twin zygosity was determined through DNA microsatellite analysis for 87% of same-sex twin pairs. Seven concordant and zero discordant markers were necessary for a monozygotic assignment, while one or more discordant markers indicated dizygotic status. For the remaining same-sex twin pairs, zygosity was established by questionnaire items about the twins' physical similarity and the frequency with which people confuse them. The questionnaire was used only when DNA samples were insufficient for one or both twins in a pair. When both questionnaire and DNA results were available, there was a 90% agreement between the two. For complete details about procedures and measures, see Baker et al. [47]. The study had been approved by an ethical review board; project title: Social and Moral Development: Phase III; IRB Protocol Number: UP-11-00503.

### Measures

The Child Behavior Checklist (CBCL) is a highly reliable and well-validated instrument that assesses externalizing and internalizing childhood behavioral problems based on parental ratings [48]. Each of the 113 items pertains to behavior witnessed by the parent in the preceding 6 months and is scored on a three-point scale (0 = *not true*, 1 = *somewhat or sometimes true*, 2 = *very true or often true*) [49]. The Delinquent Behavior subscale consists of 13 items, including nonaggressive antisocial behaviors such as lying and stealing. The Aggressive Behavior subscale is made up of 20 items that indicate both physically aggressive behaviors (e.g., destroys others' belongings, fights with other children) as well as aspects of an aggressive personality (argues a lot, brags and boasts, etc.).

The CBCL was administered during Waves 1 through 4. An overwhelming majority (> 92%) of caregiver ratings were obtained from the twins' biological mother. Parents rated both of their twin children's behaviors at the same time. Cronbach's Alpha for Delinquent Behavior at ages 9–10, 11–13, 14–15, and 16–18 were 0.61, 0.66, 0.78, and 0.70, respectively. Cronbach's Alpha for Aggressive Behavior at these four time points were higher: 0.88 (ages 9–10), 0.88 (ages 11–13), 0.89 (ages 14–15), and 0.84 (ages 16–18). The Delinquent Behavior and the Aggressive Behavior subscales were created by taking the arithmetic mean of the completed items and multiplying by the total number of items. Effectively, this resulted in a sum score for each subscale.

## Attrition

Since attrition may bias estimates in longitudinal analyses [50], we carried out analyses to assess whether baseline aggression and delinquency scores differ between participants who participated in more than one wave (responders) and those who dropped out of the study (non-responders). For those participants who began in Wave 1, logistic regression analyses revealed that higher aggressive behavior scores at Wave 1 significantly predicted later attrition (OR 1.05,  $p=0.04$ ). Delinquent behavior scores at Wave 1 did not significantly predict attrition (OR 1.03,  $p=0.62$ ).

## Statistical Analyses

### Latent Class Growth Analyses

Latent class growth analyses (LCGA) [51] were used to explore the developmental trajectories of delinquent and aggressive behavior from Wave 1 (ages 9–10) to Wave 4 (16–18 years old). To account for skewed distributions of these two behaviors, we fit a Poisson regression model to the data. This is easily handled in Mplus version 7.1 [52] by specifying the measurement scale as ‘count’, such that the proper error distribution is produced by the maximum-likelihood estimator. The developmental trajectory of each latent group across waves was represented by a latent growth curve model, with two growth factors estimated: the intercept (G0, also known as the baseline) and slope (G1, also known as rate). The LCGA assumed homogeneity of individual trajectories within a single group, thus constraining the variance/covariance of the intercept and slope to zero but allowed for differences in these two parameters across latent groups. The weight coefficients for the slope, assumed to be uniform across class, were set to be 0 and 1 for the first wave and the last wave, respectively. The slope loadings were freely estimated for the other waves. Five LCGA models were fit to the data, with the number of latent groups ranging from 2 to 6.

Latent class growth analyses were conducted in Mplus using the maximum likelihood method with robust standard errors. Missing values were handled by full information maximum likelihood estimation. The preferred number of latent groups was determined by the Lo–Mendell–Rubin (LMR) likelihood ratio test [53]. The  $p$ -value yielded by this test determines whether one can reject the null hypothesis that a neighboring class model (with one fewer class) is viable. We first examined whether a model with two latent classes is superior to one where no classes are present. We then progressively tested the fit of less parsimonious models (i.e., where one more class was added). This process was continued until the Lo–Mendell–Rubin likelihood test for a given model was nonsignificant ( $p > 0.05$ ).

We also used values of Akaike Information Criterion (AIC) and the sample-size adjusted Bayesian Information Criterion (SABIC) to guide model selection. Lower AIC and SABIC values signify a more optimal solution. Both criteria exact a penalty when introducing additional parameters to the model (i.e., increasing the number of classes). Ideally, these values should reach an absolute minimum or at least plateau as more and more latent classes are added.

Once a class solution was selected, we extracted the most likely class membership for each participant. We accommodated for the clustered nature of observations (i.e., twins nested within families) in Mplus. Because each class membership is binary, tetrachoric correlation coefficients were used to express the twin correlation of liability for a given antisocial trajectory. By glancing at the tetrachoric correlations in monozygotic (MZ) and dizygotic (DZ) twins, it is easy to infer the relative importance of genetic and environmental influences on the various trajectory groups. A higher coefficient for MZ than for DZ twins indicates that genetic influences are important for the behavior under study.

Lastly, we used liability threshold twin modelling to formally estimate the heritability of each antisocial trajectory [54]. The heritability coefficient represents the proportion of individual differences in liability attributable to genetic effects. Since genetic influences were assumed to be additive, the genetic correlation was specified as 0.5 in DZ twins and 1 (unity) in MZ twins. The structural equation model also included a family-wide environmental parameter, which was fixed at unity for both twin types. The family-wide environment represents any influences (e.g., neighborhood quality and language spoken at home) that foster greater similarity among reared-together siblings irrespective of genetic relatedness.

## Results

### Descriptive Statistics

Table 1 presents the means and standard deviations for the *Rule-Breaking (Delinquent)* and *Aggressive Behavior* subscales stratified by sex across the four waves. Relative to female participants, male participants scored significantly higher on Rule-Breaking (Delinquency) at Waves 1, 2 and 4, and significantly higher on Aggression at Waves 1 and 3.

Phenotypic cross-trait correlations across the four assessments of Aggressive Behavior and Delinquent Behavior are reported in Table 2. Correlations within the same assessment wave are moderate-to-high in magnitude (ranging from 0.57 to 0.68). The time-lagged correlations are also positive, but somewhat lower in magnitude (0.28–0.47). Cross-trait, within-time correlations between Aggressive Behavior and Delinquent Behavior were stronger in magnitude than any of

**Table 1** Descriptive statistics for rule-breaking (delinquent) and aggressive behavior

	Males Mean (SD) n	Females Mean (SD) n
Rule-breaking (delinquent), 9–10 years**	1.53 (1.86) n=589	1.08 (1.46) n=624
Rule-breaking (delinquent), 11–13 years*	1.43 (1.91) n=414	1.12 (1.72) n=454
Rule-breaking (delinquent), 14–15 years	1.73 (2.43) n=565	1.49 (2.56) n=589
Rule-breaking (delinquent), 16–18 years*	1.61 (2.19) n=457	1.31 (1.90) n=476
Aggressive behavior, 9–10 years**	6.37 (5.35) n=589	5.28 (5.21) n=624
Aggressive behavior, 11–13 years	5.12 (5.19) n=414	4.75 (5.14) n=454
Aggressive behavior, 14–15 years*	5.36 (5.67) n=565	4.63 (5.30) n=589
Aggressive behavior, 16–18 years	3.72 (4.31) n=457	3.89 (4.02) n=476

Significant mean differences across sex are flagged as \*  $p < 0.05$  or \*\*  $p < 0.001$

**Table 2** Phenotypic correlations for rule-breaking (delinquent) and aggressive behavior

	Delinquent behavior			
	Ages 9–10	Ages 11–13	Ages 14–15	Ages 16–18
<i>Aggressive behavior</i>				
Ages 9–10	0.63	0.41	0.35	0.28
Ages 11–13	0.43	0.68	0.42	0.35
Ages 14–15	0.37	0.47	0.67	0.38
Ages 16–18	0.31	0.36	0.37	0.57

All correlations are significant at  $p < 0.01$

**Table 3** Twin correlations for rule-breaking (delinquent) and aggressive behavior

	Aggressive behavior				Delinquent behavior			
	Ages 9–10	Ages 11–13	Ages 14–15	Ages 16–18	Ages 9–10	Ages 11–13	Ages 14–15	Ages 16–18
MZ males	0.62	0.72	0.74	0.52	0.63	0.76	0.85	0.65
MZ females	0.53	0.71	0.72	0.41	0.63	0.68	0.88	0.70
DZ males	0.38	0.23 <sup>a</sup>	0.11 <sup>a</sup>	0.10 <sup>a</sup>	0.42	0.47	0.46	0.38
DZ females	0.44	0.38	0.43	0.30	0.51	0.65	0.48	0.45
DZ OS	0.41	0.52	0.51	0.29	0.30	0.48	0.56	0.41

DZ dizygotic, MZ monozygotic, OS opposite sex

<sup>a</sup> $p > 0.01$

the twelve within-trait, cross-time correlations (for the sake of brevity, the latter are not displayed). This underscores the fact that aggression and delinquency are part of the same antisocial construct.

Within-trait correlations between twins are presented in Table 3. Higher correlations for MZ twin pairs relative to DZ pairs indicate the presence of genetic influences on the variance of a trait. This pattern was observed for all eight indices. However, it is possible that the heritability of antisocial behavior differs according to the developmental trajectory of aggression/delinquency.

### Latent Class Growth Analyses Across Waves 1–4

#### Rule-Breaking (Delinquent) Behavior

A latent growth curve model without any mixture assumption was first fit to the data. The slope mean was significantly greater than zero, as were the random effects (i.e., variances around the intercept and slope). However, there was minimal covariance between the intercept and slope factors ( $p = 0.42$ ); i.e., individual differences in baseline delinquent behavior did not predict subsequent growth. To guard against model misspecification, we also examined growth using a nonlinear weighting strategy. Specifically, we introduced a quadratic factor to the growth model in addition to modeling the linear change (slope). (The slope loadings were fixed at 0, 1, 2, and 3 at the four respective waves to permit model identification.) Incorporating quadratic growth led to a poorer-fitting model, with neither the mean nor variance of the quadratic parameter reaching statistical significance. As a result, we proceeded with the LCGA using a linear-only model of growth.

Fit and comparison statistics for the LCGA class solutions are reported in Table 4. Neither the AIC nor SABIC reached a trough, but rather showed progressive improvement as the number of classes increased. Nonetheless, improvement considerably slowed once the number of extracted classes reached three or four. Based on the LMR likelihood ratio



**Table 4** Model fitting of delinquent behavior, Waves 1–4

# of latent groups	# of free P.	LRT p-value	AIC	SABIC	Class breakdown N (%)
None	4		15,854.27	15,862.91	
Two groups	7	0.0000	13,226.92	13,242.03	Class 1: 361 (23.5) Class 2: 1172 (76.5)
Three groups	10	0.0000	12,771.45	12,793.02	Class 1: 152 (9.9) Class 2: 711 (46.4) Class 3: 670 (43.7)
Four groups	13	0.2696	12,690.59	12,718.65	Class 1: 60 (3.9) Class 2: 668 (43.6) Class 3: 667 (43.5) Class 4: 138 (9.0)
Five groups	16	0.3500	12,661.43	12,695.96	Class 1: 417 (27.2) Class 2: 13 (7.4) Class 3: 346 (22.6) Class 4: 67 (4.4) Class 5: 590 (38.5)
Six groups	19	0.3761	12,634.81	12,675.82	Class 1: 346 (22.6) Class 2: 85 (5.5) Class 3: 14 (0.9) Class 4: 73 (4.8) Class 5: 391 (25.5) Class 6: 624 (40.7)

P model parameters, LRT Lo–Mendell–Rubin likelihood ratio test, AIC Akaike Information Criterion, SABIC sample-size adjusted Bayesian Information Criterion

test, a three-class solution was tenable. This is indicated by the non-significant p-value of the neighboring 4-class model ( $p = 0.27$ ).

The three subgroups that emerged can be described as *Low-Level Delinquent* (low but stable rule-breaking across development), *Non-Delinquent* (abstainers who refrain from all antisocial activity), and *Persistent Delinquent* (childhood onset with adolescent escalation). The latter unsurprisingly had the smallest class size (comprising 9.9% of individuals). Developmental trajectories based on expected class membership from this 3-class model are plotted in Fig. 1. The distribution of delinquent subgroups demonstrated significant sex differences ( $\chi^2 = 25.8$ ,  $df = 2$ ,  $p < 0.001$ ). Specifically, there were nearly twice as many males as females in the Persistent Delinquent group (66% vs. 34%).

One may question our characterization of the elevated class as “persistent”, given that levels of delinquency nearly doubled for this subgroup between preadolescence and age 14–15. Due to the theoretical importance of an adolescent-onset trajectory, we probed more complex solutions for evidence of such a class. Inspection of the four-class solution revealed a putative *Late Onset* (adolescent onset/transitory) subgroup that showed a similar intercept as “low-level” delinquents. However, this “late onset” class was marked by a slope that ran parallel to the slope of the now-reduced “persistent delinquent” class. The “persistent” subgroup exhibited twice as much delinquent behavior as the supposed “late onset” class at age 14–15. Because the latter is essentially a

milder (lower-intercept) variant of *Persistent Delinquent*, it did not seem advisable to select a four-class solution. Moreover, the *Persistent Delinquent* class size would be rendered unrealistically small (3.9%) if we had sought to isolate a pure adolescent-onset subgroup in the 4-class solution.

### Aggressive Behavior

A baseline latent growth model was fit to the four waves of data. The slope mean was significantly negative, indicating that Aggressive Behavior decreases with age. Unlike for delinquent behavior, there was a strong inverse relationship between the intercept and slope factors ( $p = 0.001$ ). Children with higher baseline levels of aggression tended to show steeper decreases over time. A growth curve model specifying both linear and quadratic change was untenable.

Results from the LCGA model-fitting are detailed in Table 5. The fit criteria (AIC and SABIC) did not show any stabilization in improvement across the range of solutions tested. Rather, the values were in continuous freefall as the number of classes increased (see Table 5). The outcome of the LMR likelihood ratio test, however, suggested that a three-class solution was viable ( $p = 0.29$ ).

These classes could be described as *Non-Aggressive*, *Moderately Aggressive*, and *Highly Aggressive*. The latter subgroup showed a notable decrease in aggressive behavior during late adolescence, although levels remained substantially above those of the other subgroups. Mean aggression

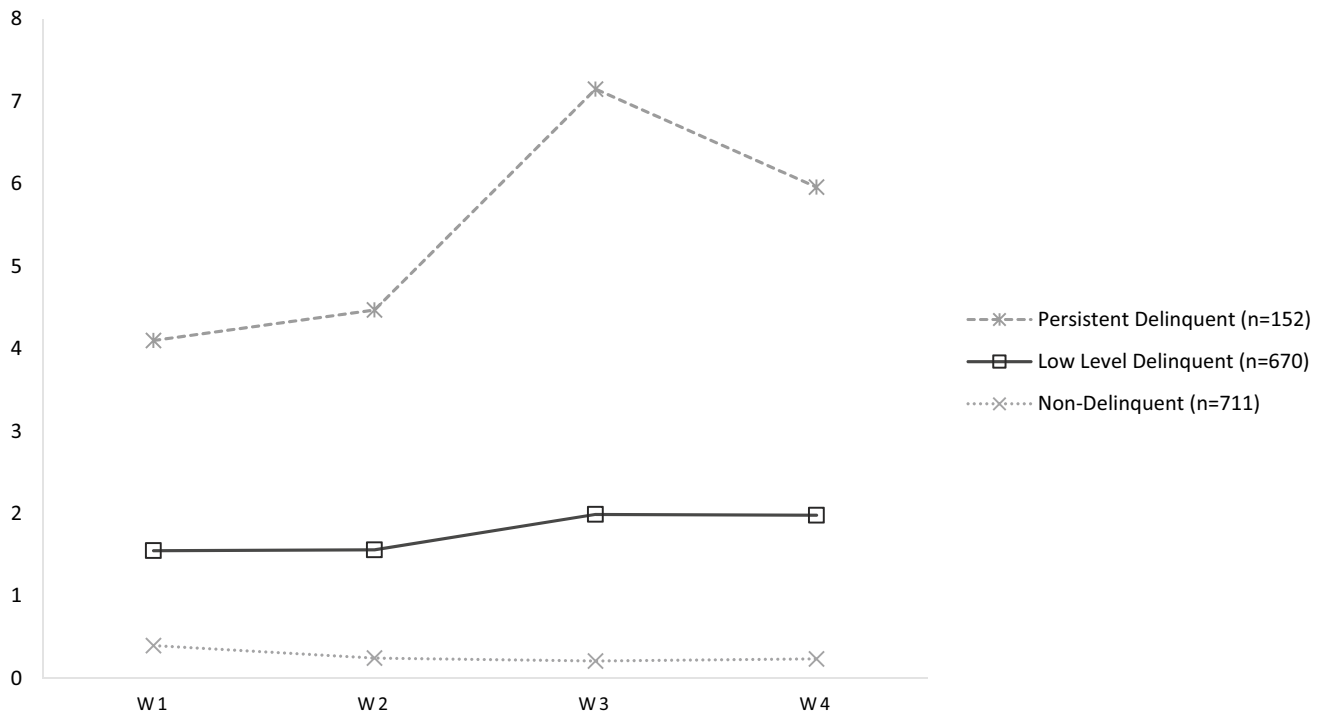


Fig. 1 Expected developmental trajectories of delinquent behavior across ages 9–10 to 16–18 years

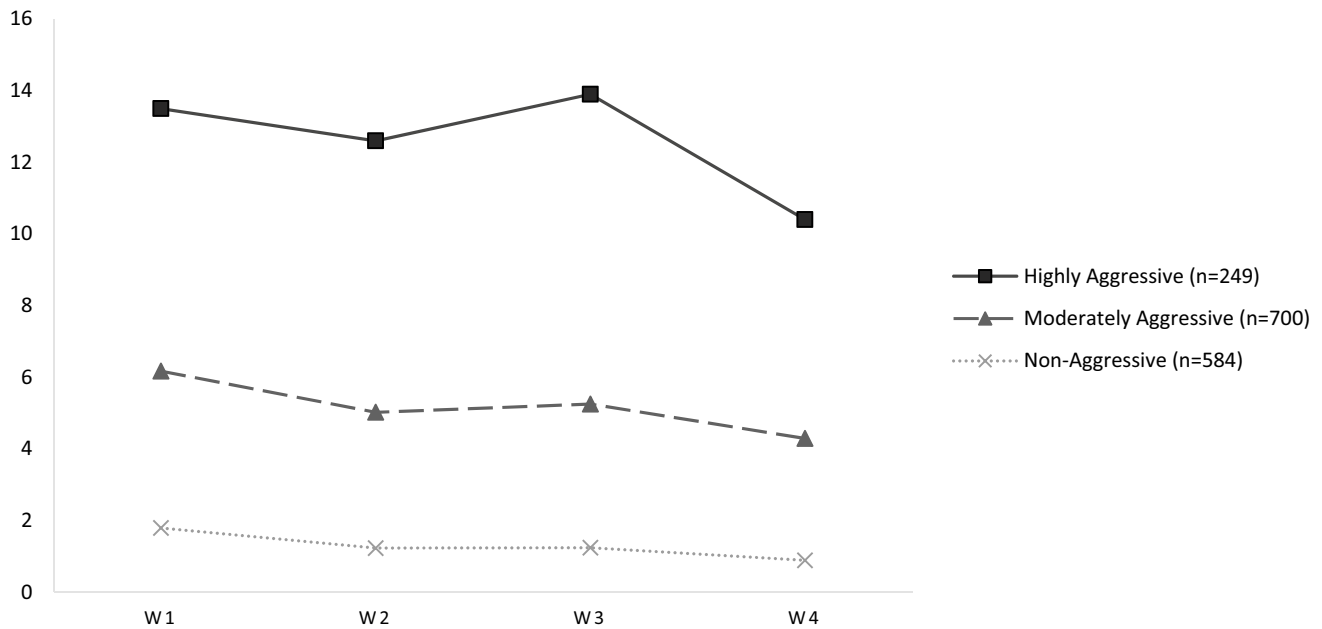
Table 5 Model fitting of aggressive behavior, Waves 1–4

# of latent groups	# of free P.	LRT p-value	AIC	SABIC	Class breakdown N (%)
None	4		31,146.68	31,155.31	
Two groups	7	0.0000	24,183.28	24,198.39	Class 1: 942 (61.4) Class 2: 591 (38.6)
Three groups	10	0.0004	22,730.07	22,751.65	Class 1: 700 (45.7) Class 2: 584 (38.1) Class 3: 249 (16.2)
Four groups	13	0.2909	22,361.71	22,389.76	Class 1: 375 (24.5) Class 2: 433 (28.2) Class 3: 110 (7.2) Class 4: 615 (40.1)
Five groups	16	0.0942	22,084.02	22,118.55	Class 1: 92 (6.0) Class 2: 445 (29.0) Class 3: 351 (22.9) Class 4: 190 (12.4) Class 5: 455 (29.7)
Six groups	19	0.1415	21,901.81	21,942.82	Class 1: 19 (1.2) Class 2: 217 (14.2) Class 3: 483 (31.5) Class 4: 383 (25.0) Class 5: 144 (9.4) Class 6: 287 (18.7)

P model parameters, LRT Lo–Mendell–Rubin likelihood ratio test, AIC Akaike Information Criterion, SABIC sample-size adjusted Bayesian Information Criterion

scores are plotted according to individuals’ most likely class membership in Fig. 2. The distribution of aggressive subgroups demonstrated significant sex differences ( $\chi^2 = 7.3$ ,

$df = 2, p = 0.03$ ), mainly on account of the larger male membership in the *Highly Aggressive* class (57% vs. 43% female).



**Fig. 2** Expected developmental trajectories of aggressive behavior across ages 9–10 to 16–18 years

Inspection of the four-class model revealed a very uninteresting pattern of trajectories. The subgroups simply differed with respect to the intercept (baseline); there was little separation in terms of the slope. With further probing, a *Desisting* (decreasing) subgroup emerged in the five-class solution. Members of this class possessed a moderately high aggression baseline, with levels eventually approaching those of the *Non-Aggressive* subgroup by late adolescence. However, the five-class solution led to a fuzzier classification of individuals into classes (i.e., entropy values were substantially lower than for the three-class and four-class solutions). The inclusion of more classes only led to ever-finer divisions of the low/moderate/high aggressive groups.

### Demographic Covariates

Gender is not the only demographic characteristic known to correlate with antisocial offending. Prior literature indicates that socioeconomic disadvantage as well as belonging to a Hispanic or Black ethnic/racial group pose an increased risk for juvenile delinquency. Thus, there is reason to hypothesize that low socioeconomic status and an ethnic minority background are disproportionately represented among Low-Level and Persistent Delinquents. The present sample is ideally suited to evaluate these variables, given the pronounced sociocultural diversity of communities in Southern California.

Family socioeconomic status (SES) was based on a composite of both parents' educational levels and current occupational status [55] as well as household income. Details

regarding the computation of this SES factor are provided elsewhere [56]. Ethnic/racial background of twins was determined through the race and Hispanic status of the children's biological parents, as reported by the primary caregiver. If the mother and father belonged to different racial groups (or were multiracial themselves), then the twin offspring were coded as 'Mixed'.

Demographic characteristics of the subgroups extracted from the optimal three-class solution are presented in Table 6. The various ethnic/racial groups were not equally distributed across the three subgroups of Delinquent Behavior ( $\chi^2 = 37.6$ ,  $df = 8$ ,  $p < 0.001$ ). Notably, white non-Hispanic participants were overrepresented among Non-Delinquents, while Hispanic participants were underrepresented among this subgroup.<sup>1</sup> On the other hand, ethnic/racial status was not significantly associated with Aggressive Behavior trajectories ( $\chi^2 = 12.2$ ,  $df = 8$ ,  $p = 0.14$ ). Socioeconomic status appeared to differentiate the Non-Delinquent and Non-Aggressive classes from all other classes. Accordingly, we computed two dummy variables for each antisocial construct, in which the low-level/persistent delinquent subgroups and moderately/highly aggressive subgroups were compared to their respective (non-antisocial) reference

<sup>1</sup> The validity of this analysis hinges on the assumption that each ethnic/racial group contains a similar (~50%) proportion of males. In practice, Asian-American participants were skewed towards males (67%), whereas the other groups were 47–51% male. Despite this sex imbalance, Asian-American participants were especially unlikely to be classified as Persistent Delinquents.



**Table 6** Demographic composition of delinquent and aggressive behavior subgroups

Class	Male (%)	White (%)	Hispanic (%)	Black (%)	Asian (%)	Mixed (%)	SES <i>M (SD)</i>
<b>Delinquent behavior</b>							
Non-delinquent	44.2	33.8	29.4	13.8	4.6	18.3	0.20 (1.02)
Low level	51.9	25.6	40.3	11.1	4.4	18.6	- 0.16 (0.97)
Persistent	65.8	23.7	37.5	22.4	1.3	15.1	- 0.14 (0.88)
<b>Aggressive behavior</b>							
Non-aggressive	46.4	31.7	30.1	14.3	5.2	18.8	0.19 (1.02)
Moderately	50.0	28.2	37.5	12.5	3.7	18.1	- 0.07 (0.99)
Highly	56.6	26.2	39.1	14.5	3.2	16.9	- 0.16 (0.92)

SES family socioeconomic status (z-score)

**Table 7** Tetrachoric twins correlations and heritability estimates

<b>Rule-breaking (delinquent) behavior</b>			
	Non-delinquent	Low-level	Persistent
MZ twins (n = 333 pairs)	0.77 ± 0.05	0.62 ± 0.06	0.85 ± 0.06
DZ twins (n = 433 pairs)	0.76 ± 0.04	0.54 ± 0.06	0.51 ± 0.10
Heritability	3% [0%, 27%]	16% [0%, 50%]	67% [23%, 100%]
<b>Aggressive behavior</b>			
	Non-aggressive	Moderate	High
MZ twins (n = 333 pairs)	0.62 ± 0.06	0.46 ± 0.07	0.69 ± 0.08
DZ twins (n = 433 pairs)	0.43 ± 0.07	0.23 ± 0.03	0.46 ± 0.08
Heritability	39% [3%, 75%]	46% [33%, 59%]	45% [1%, 89%]

Standard errors are listed after the plus-minus signs; 95% confidence intervals around the heritability estimates are enclosed in brackets

group. The regression of SES on these dummy variables was significantly negative ( $ps < 0.001$ ), indicating lower SES in the higher-risk antisocial classes.

### Shared Twin Liability for Antisocial Trajectories

The genetic and environmental etiologies of the various classes were next examined. Calculations of heritability are based on the tetrachoric correlations, i.e., correlations of liability for latent group membership between Twin 1 and Twin 2. Tetrachoric correlations and the attendant heritability coefficients are reported in Table 7. Correlations of liability for Persistent Delinquency were markedly higher in MZ twins than DZ twins (0.85 vs. 0.51). (The lower DZ correlation is not due to within-pair heterogeneity in gender; the tetrachoric correlation for opposite-sex twin pairs was in fact higher than the correlation for DZ same-sex pairs.) For the low-risk delinquent subgroups, the tetrachoric correlations were almost equally high between MZ and DZ twins. Substantial heritability (67%) was thus only found for Persistent Delinquency, whereas genetic influences were

non-significant for the other two delinquency subgroups (heritability ranged from 3 to 16%). Family-wide environmental factors are accordingly protective against delinquency and implicated for low-level delinquency.

We decomposed the tetrachoric correlations into genetic and environmental sources for the three subgroups of Aggressive Behavior. In all cases, the MZ tetrachoric correlation was significantly higher than that for DZ pairs (see Table 7), yielding moderate heritability coefficients (39–46%). This supports the idea that genetic influences span the entire continuum of risk for aggressive behavior.

## Discussion

A primary goal of the present study was to uncover some of the commonly theorized trajectories of delinquent and aggressive behavior from previous work. We used a developmentally rich dataset of caregiver observations from the Child Behavior Checklist. Measures of antisocial behavior were available on up to four occasions, when twin offspring

were between ages 9–10 and 16–18. We used latent class growth analysis (LCGA) to identify distinct antisocial subgroups.

Results revealed that the developmental course of aggressive behavior may not be amenable to a qualitative conceptualization. Based on various fit criteria and inspection of class solutions, our LCGA uncovered a generic pattern of high, moderate, and low subgroups. For example, we did not find compelling evidence for a desisting/childhood-limited subtype of aggression. Furthermore, it was difficult to detect a pure adolescent-onset subtype of delinquency. While some of our class solutions presumably captured these trajectories, there was weak empirical justification for them based on class size considerations and interpretability.

A second goal of this investigation was to estimate the genetic liability for the various trajectories. We found that delinquent and aggressive subgroups appear to have different genetic and environmental etiologies. It reinforces the notion that antisocial behavior is a complex heterogeneous phenomenon. Below we discuss key findings with respect to the latent class structure of delinquent and aggressive behavior.

### Delinquent Behavior

For delinquent behavior, three trajectories were identified from ages 9–10 to 16–18: Low Level Delinquent, Non-Delinquent, and Persistent Delinquent. Non-delinquents represented the largest class and were characterized by higher socioeconomic status and an increased likelihood of having a non-Hispanic white background. The Persistent Delinquent class was predictably in the minority, comprising approximately 10% of participants. Its members were already antisocial at baseline and showed an escalating pattern of delinquent behavior during adolescence. An adolescent-onset subgroup was explored but appeared to be a milder variant of the Persistent Delinquent trajectory. That is, the two subgroups were characterized by the same increasing slope but with very different intercepts. It was not possible to explore a “late bloomer” class [15], as our data did not extend beyond ages 16–18.

A prior analysis of antisocial behavior from ages 7 to 32 years identified four developmental trajectory groups, including life-course persistent, adolescent-onset, low level, and childhood limited groups [24]. The authors operationalized antisocial conduct using a combination of aggressive and non-aggressive items, which limits direct comparison with our results. At any rate, Odgers et al. [24] identified a decreasing (childhood-limited) group, whereas we could not identify such a trajectory. Our findings are partially in line with Moffitt’s developmental taxonomy, which predicts a Persistent Delinquent group [14] and, more recently, Low Level Delinquent and Non-Delinquent groups [25, 29]. Differences between the present findings and prior

research—notably our lack of an “adolescent-onset” class—could be due to the compressed age range of the present sample as well as dependence on caregiver-reported data. Previous studies, where such a trajectory has often borne out, typically incorporated self-reports of antisocial behavior in adolescence.

### Aggressive Behavior

The various classes of aggressive behavior that emerged were distinguishable by different baseline levels rather than distinct slopes/trajectories. Three trajectories were identified: Non-Aggressive, Moderately Aggressive, and Highly Aggressive. The ethnic/racial breakdown did not significantly differ across the three subgroups, although membership in the Non-Aggressive subgroup was associated with higher socioeconomic status. We did not find evidence of an Increasing (adolescent onset/transitory) subgroup for aggressive behavior, which accords with previous research [28, 34, 35]. Rather, we observed a decreasing trend in aggression over time, such that all classes were characterized by lower scores at age 16–18 than at baseline. This is consistent with children’s typical decline in aggressive behavior as they mature [22, 35, 57, 58].

Similar to previous work, we identified Low Aggressive and Moderately Aggressive subgroups [34, 58]. Di Giunta et al. [34] identified four aggressive behavior trajectory groups from self-reports (low stable, moderate-low declining, moderate declining, and high stable), and three trajectories from mothers’ ratings (low declining, moderate declining, high stable). Furthermore, Côté et al. [58] found three groups based on data collected from the child’s primary caregiver: low desisters, moderate desisters, and high stable.

Notwithstanding these prior findings, we are not convinced that the developmental course of aggression is optimally represented by distinct subgroups/trajectories. This opinion resonates with the conclusions of Walters [31], who surmised that antisocial behavior exists on a continuum, in which individuals differ in degree rather than kind. The fact that a similar degree of genetic influence was present across the continuum of risk for aggressive behavior supports the idea that aggression conforms to a dimensional rather than qualitative structure.

### Genetic and Environmental Etiologies

Previous research has found that delinquent and aggressive behaviors have different underlying genetic and environmental etiologies. Delinquent behavior is influenced by genetic factors as well as family-wide environmental effects, whereas aggressive behavior seems to primarily be influenced by genetic factors [18, 43]. Results from our liability threshold twin models confirmed these observations,

but with an important twist. The magnitude of genetic and environmental influence apparently differs depending on the delinquent subgroup. Genetic influences were significant for Persistent Delinquency but were negligible for the Low-Level Delinquent and Non-Delinquent subgroups. Concordance for the latter was mainly attributable to family-wide environmental effects.

Liability for persistent delinquency was highly heritable. Due to its relatively small class size, the heritability coefficient (67%) was statistically indistinguishable from 100%. The finding that Persistent Delinquency is especially influenced by genes is consistent with earlier work (e.g., Lyons et al. [40]). By contrast, twin modeling indicated that genetic effects were uniform in magnitude across all aggressive behavior subgroups; heritability coefficients ranged from 39 to 46%.

### Limitations

A few limitations of the present study must be considered when interpreting our findings. First, we had patterns of attrition across the four waves related to our aggression variable. Specifically, our analyses indicated that those who ceased participating in our study exhibited initially higher levels of aggression, but not delinquent behavior. Second, the Aggressive Behavior subscale is overly broad. It measures other aspects of negative emotionality, not just aggression. Perhaps this explains why we get muted sex differences. Third, we relied on parent-report data, as youth self-report data only became available starting at Wave 3 (14–15 years old). Although use of parent-report data is able to paint a more complete developmental picture, parents may have limited information regarding their children's antisocial activities, specifically regarding delinquent behavior [59]. Parents rated both of their twin offspring at the same time, which may have led to inflated estimates of familial aggregation of antisocial behavior. Finally, our participants were 16–18 years old at the last assessment. Thus, it is possible that the low-level/non-delinquent subgroups contain individuals who have yet to reach their “potential” as antisocial late-bloomers [15, 60].

### Summary

Genetically informative designs can uniquely clarify the etiology of various subtypes and trajectories of antisocial behavior. Prior studies have typically been unable to characterize the broad genetic and environmental structure of these developmental trajectories. In a community sample of 1532 twin youth, we used growth mixture modeling to uncover distinct classes of aggressive and delinquent behavior from ages 9–10 to 16–18. An optimal three-class solution emerged for both aggressive behavior and delinquent

behavior. Threshold models indicated that genetic contributions to liability to the Aggressive Behavior subgroups—Non-Aggressive, Moderate, and High—were consistently moderate in magnitude.

There was greater etiological heterogeneity among the Delinquent Behavior subgroups: Non-Delinquent, Low-Level, and Persistent. Twin concordance for the two lower-risk subgroups was not significantly attributable to genetic factors. However, it appears that persistent delinquency represents an etiologically distinct class of rule-breaking with strong genetic roots (heritability = 67%). The ‘persistent delinquents’ that we identified may be on a similar developmental track as the life-course persistent offenders and “continuous antisocials” described by Moffitt [14] and DiLalla and Gottesman [15]. This has important implications for understanding the nature of severe, pervasive patterns of rule-breaking behavior. Temperamental factors and other inherited vulnerabilities may contribute to persistent delinquency, whereas family-wide environmental factors (e.g., parental monitoring) may confer general protection against delinquency.

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### Compliance with Ethical Standards

**Conflict of interest** This study was supported by a grant to Laura Baker from the National Institute of Mental Health (R01 MH58354). Adrian Raine was supported by the National Institute of Mental Health (Independent Scientist Award K02 MH01114-08). Catherine Tuvblad was funded by the Swedish Research Council (2018-01041).

**Ethical Approval** All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

**Informed Consent** Informed consent was obtained from all individual [caregiver] participants included in the study. Furthermore, assent was obtained from minor offspring to participate in the study and provide salivary DNA.

### References

1. Berkowitz L (1993) *Aggression: its causes, consequences, and control*. Temple University Press, Philadelphia
2. Dodge KA, Coie JD, Lynam D (2006) Aggression and antisocial behavior in youth. In: Eisenberg N, Damon W, Lerner RM (eds) *Handbook of child psychology. Social, emotional, and personality development*, vol 3. Wiley, New York

3. Webster GD, Dewall CN, Pond RSJ, Deckman T, Jonason PK, Le BM et al (2014) The brief aggression questionnaire: psychometric and behavioral evidence for an efficient measure of trait aggression. *Aggress Behav* 40:120–139. <https://doi.org/10.1002/ab.21507>
4. Achenbach TM (2009) The achenbach system of empirically based assesemnt (ASEBA): development, findings, theory, and applications. University of Vermont Research Center for Children, Youth and Families, Burlington
5. Robins LN (1966) Deviant children grown up, a sociological and psychiatric study of sociopathic personality. Williams & Wilkins, Baltimore
6. Kim-Cohen J, Caspi A, Moffitt TE, Harrington H, Milne BJ, Poulton R (2003) Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort. *Arch Gen Psychiatry* 60:709–717. <https://doi.org/10.1001/archpsyc.60.7.709>
7. Rutter M, Giller H, Hagell A (1998) Antisocial behavior by young people. Cambridge University Press, Cambridge
8. American Psychiatric Association (2013) Diagnostic and statistical manual of mental disorders, vol 5. American Psychiatric Publishing, Arlington
9. McCollister KE, French MT, Fang H (2010) The cost of crime to society: new crime-specific estimates for policy and program evaluation. *Drug Alcohol Depend* 108:98–109. <https://doi.org/10.1016/j.drugalcdep.2009.12.002>
10. Piquero AR, Farrington DP, Blumstein A (2003) The criminal career paradigm. In: Tonry M (ed) *Crime and justice: a review of research*, vol 30. University of Chicago Press, Chicago
11. Piquero AR, Farrington DP, Blumstein A (2007) Key issues in criminal careers research: new analysis from the Cambridge study in delinquent development. Cambridge University Press, Cambridge
12. Farrington DP (1986) Age and crime. In: Tonry M, Morris N (eds) *Crime and justice: an annual review of research*, vol 7. University of Chicago Press, Chicago
13. Farrington DP, Piquero AR, Jennings WG (2013) Offending from childhood to late middle age: recent results from the Cambridge study in delinquent development. Springer, New York
14. Moffitt TE (1993) Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychol Rev* 100:674–701. <https://doi.org/10.1037/0033-295X.100.4.674>
15. DiLalla LF, Gottesman I (1989) Heterogeneity of causes for delinquency and criminality: lifespan perspectives. *Dev Psychopathol* 1:339–349. <https://doi.org/10.1017/S0954579400000511>
16. Frick PJ, Lahey BB, Loeber R, Tannenbaum L, Van Horn Y, Christ MAG et al (1993) Oppositional defiant disorder and conduct disorder: a meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clin Psychol Rev* 13:319–340. [https://doi.org/10.1016/0272-7358\(93\)90016-F](https://doi.org/10.1016/0272-7358(93)90016-F)
17. Patterson GR, DeBaryshe BD, Ramsey E (1989) A developmental perspective on antisocial behavior. *Am Psychol* 44:329–335. <https://doi.org/10.1037/0003-066X.44.2.329>
18. Burt SA, Donnellan MB, Iacono WG, McGue M (2011) Age-of-onset or behavioral sub-types? A prospective comparison of two approaches to characterizing the heterogeneity within antisocial behavior. *J Abnorm Child Psychol* 39:633–644
19. Eley TC, Lichtenstein P, Moffitt TE (2003) A longitudinal behavioral genetic analysis of the etiology of aggressive and non-aggressive antisocial behavior. *Dev Psychopathol* 15:383–402. <https://doi.org/10.1017/S095457940300021X>
20. Loeber R, Hay D (1997) Key issues in the development of aggression and violence from childhood to early adulthood. *Annu Rev Psychol* 48:371–410. <https://doi.org/10.1146/annurev.psych.48.1.371>
21. van Lier PAC, Vitaro F, Barker ED, Koot HM, Tremblay R (2009) Developmental links between trajectories of physical violence, vandalism, theft, and alcohol-drug use from childhood to adolescence. *J Abnorm Child Psychol* 37:481–492. <https://doi.org/10.1007/s10802-008-9289-6>
22. Brody LM, Tremblay RE, Brame B, Fergusson D, Horwood JL, Laird R et al (2003) Developmental trajectories of childhood disruptive behaviors and adolescent delinquency: a six-site, cross-national study. *Dev Psychol* 39:222–245. <https://doi.org/10.1037/0012-1649.39.2.222>
23. Xie H, Drabick DA, Chen D (2011) Developmental trajectories of aggression from late childhood through adolescence: similarities and differences across gender. *Aggress Behav* 37:387–404. <https://doi.org/10.1002/ab.20404>
24. Odgers CL, Moffitt TE, Broadbent JM, Dickson N, Hancox RJ, Harrington H et al (2008) Female and male antisocial trajectories: from childhood origins to adult outcomes. *Dev Psychopathol* 20:673–716. <https://doi.org/10.1017/S0954579408000333>
25. Piquero AP, Moffitt TE (2005) Explaining the facts of crime: how the developmental taxonomy replies to Farrington's invitation. In: Farrington DP (ed) *Integrated developmental and life-course theories of offending*. Transaction Publisher, Rutgers, Piscataway
26. D'Unger AV, Land KC, McCall PL, Nagin DS (1998) How many latent classes of delinquent/criminal careers? Results from mixed Poisson regression analyses. *Am J Sociol* 103:1593–1630. <https://doi.org/10.1086/231402>
27. Miller S, Malone PS, Dodge KA (2010) Developmental trajectories of boys' and girls' delinquency: sex differences and links to later adolescent outcomes. *J Abnorm Child Psychol* 38:1021–1032. <https://doi.org/10.1007/s10802-010-9430-1>
28. Nagin D, Tremblay RE (1999) Trajectories of boys' physical aggression, opposition, and hyperactivity on the path to physically violent and nonviolent juvenile delinquency. *Child Dev* 70:1181–1196. <https://doi.org/10.1111/1467-8624.00086>
29. Moffitt TE (2006) Life-course persistent versus adolescence-limited antisocial behavior. In: Cicchetti D, Cohen DJ (eds) *Developmental psychopathology: risk, disorder, and adaptation*, vol 3. Wiley, New York
30. Pepler DJ, Jiang D, Craig WM, Connolly J (2010) Developmental trajectories of girls' and boys' delinquency and associated problems. *J Abnorm Child Psychol* 38:1033–1044. <https://doi.org/10.1007/s10802-010-9389-y>
31. Walters GD (2010) The latent structure of life-course persistent antisocial behavior: is Moffitt's developmental taxonomy a true taxonomy? *J Consult Clin Psychol* 79:96–105. <https://doi.org/10.1037/a0021519>
32. Silverthorn P, Frick PJ (1999) Development pathways to antisocial behavior: the delayed-onset pathway in girls. *Dev Psychopathol* 11:101–126
33. Fontaine N, Carbonneau R, Vitaro F, Barker ED, Tremblay RE (2009) Research review: a critical review of studies on the developmental trajectories of antisocial behavior in females. *J Child Psychol Psychiatry* 50:363–385. <https://doi.org/10.1111/j.1469-7610.2008.01949.x>
34. Di Giunta L, Pastorelli C, Eisenberg N, Gerbino M, Castellani V, Bombi AS (2010) Developmental trajectories of physical aggression: prediction of overt and covert antisocial behaviors from self- and mothers' reports. *Eur Child Adolesc Psychiatry* 19:873–882. <https://doi.org/10.1007/s00787-010-0134-4>
35. Brame B, Nagin DS, Tremblay RE (2001) Developmental trajectories of physical aggression from school entry to late adolescence. *J Child Psychol Psychiatry* 42:503–512. <https://doi.org/10.1111/1469-7610.00744>



36. Pardini D, Frick PJ (2013) Multiple developmental pathways to conduct disorder: current conceptualizations and clinical implications. *J Can Acad Child Adolesc Psychiatry* 22:20–25
37. Pulkkinen L, Lyyra A-L, Kokko K (2009) Life success of males on nonoffender, adolescence-limited, persistent, and adult-onset antisocial pathways: follow-up from age 8 to 42. *Aggress Behav* 35:117–135. <https://doi.org/10.1002/ab.20297>
38. Pedersen W, Hart RK, Moffitt TE, von Soest T (2020) Delinquency abstainers in adolescence and educational and labor market outcomes in midlife: a population-based 25-year longitudinal study. *Dev Psychol*. <https://doi.org/10.1037/dev0001117>
39. Vaughn MG, Fu Q, Wernet SJ, DeLisi M, Beaver KM, Perron BE et al (2011) Characteristics of abstainers from substance use and antisocial behavior in the united states. *J Crim Justice* 39:212–217. <https://doi.org/10.1016/j.jcrimjus.2011.02.003>
40. Lyons MJ, True WR, Eisen SA, Goldberg J, Meyer JM, Faraone SV et al (1995) Differential heritability of adult and juvenile antisocial traits. *Arch Gen Psychiatry* 52:906–915. <https://doi.org/10.1001/archpsyc.1995.03950230020005>
41. Silberg JL, Rutter M, Tracy K, Maes HH, Eaves L (2007) Etiological heterogeneity in the development of antisocial behavior: the Virginia twin study of adolescent behavioral development and the young adult follow-up. *Psychol Med* 37:1193–1202. <https://doi.org/10.1017/S0033291707000293>
42. Taylor J, Iacono WG, McGue M (2000) Evidence for a genetic etiology of early-onset delinquency. *J Abnorm Psychol* 109:634–643. <https://doi.org/10.1037/0021-843X.109.4.634>
43. Burt AS (2009) Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. *Clin Psychol Rev* 29:163–178. <https://doi.org/10.1016/j.cpr.2008.12.004>
44. Burt SSA (2012) How do we optimally conceptualize the heterogeneity within antisocial behavior? An argument for aggressive versus non-aggressive behavioral dimensions. *Clin Psychol Rev* 32:263–279. <https://doi.org/10.1016/j.cpr.2012.02.006>
45. Tremblay RE (2010) Developmental origins of disruptive behaviour problems: the ‘original sin’ hypothesis, epigenetics and their consequences for prevention. *J Child Psychol Psychiatry* 51:341–367. <https://doi.org/10.1111/j.1469-7610.2010.02211.x>
46. Baker LA, Barton M, Lozano DI, Raine A, Fowler JH (2006) The Southern California twin register at the University of Southern California: II. *Twin Res Hum Genet* 9:933–940. <https://doi.org/10.1375/183242706779462912>
47. Baker L, Tuvblad C, Wang P, Gomez K, Bezdjian S, Niv S et al (2013) The Southern California twin register at the University of Southern California: III. *Twin Res Hum Genet* 16:336–343. <https://doi.org/10.1017/thg.2012.127>
48. Achenbach TM, Rescorla LA (2001) Manual for ASEBA school-age forms & profiles. University of Vermont, Research Center for Children, Youth, & Families, Burlington, VT
49. Achenbach TM (1991) Manual for the child behavior checklist/4-18 and 1991 profile. University of Vermont Department of Psychiatry, Burlington, VT
50. Heath AC, Madden PA, Martin NG (1998) Assessing the effects of cooperation bias and attrition in behavioral genetic research using data-weighting. *Behav Genet* 28:415–427
51. Jung T, Wickram KAS (2008) An introduction to latent class growth analysis and growth mixture modeling. *Soc Personal Psychol Compass* 2(1):302–317. <https://doi.org/10.1111/j.1751-9004.2007.00054.x>
52. Muthén LK, Muthén BO (1998–2012) Mplus user’s guide, 7th edn. Muthén & Muthén, Los Angeles
53. Lo Y, Mendell NR, Rubin DB (2001) Testing the number of components in a normal mixture. *Biometrika* 88:767–778
54. Neale MC, Cardon LR (1992) Methodology for genetic studies of twins and families. Kluwer, Dordrecht
55. Hollingshead AB (1975) Four factor index of social status. Yale University, Department of Sociology, New Haven
56. Baker LA, Jacobson KC, Raine A, Lozano DI, Bezdjian S (2007) Genetic and environmental bases of childhood antisocial behavior: a multi-informant twin study. *J Abnorm Psychol* 116:219
57. Tremblay RE, Nagin DS, Séguin JR, Zoccolillo M, Zelazo PD, Boivin M et al (2004) Physical aggression during early childhood: trajectories and predictors. *Pediatrics* 114:e43–e50
58. Côté S, Vaillancourt T, LeBlanc J, Nagin DS, Tremblay RE (2006) The development of physical aggression from toddlerhood to pre-adolescence: a nationwide longitudinal study. *J Abnorm Child Psychol Assess*. <https://doi.org/10.1007/s10802-005-9001-z>
59. Bartels M, Hudziak JJ, van den Oord EJCG, van Beijsterveldt CEM, Rietveld MJH, Boomsma D (2003) Co-occurrence of aggressive behavior and rule-breaking behavior at age 12: multi-rater analyses. *Behav Genet* 33:607–621. <https://doi.org/10.1023/A:1025787019702>
60. Beckley AL, Caspia A, Harringtona H, Houtsa RM, Mcgeee TR, Morganf N et al (2016) Adult-onset offenders: is a tailored theory warranted? *J Crim Justice* 46:64–81. <https://doi.org/10.1016/j.jcrimjus.2016.03.001>

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