Do polygenic influences on educational attainment predict crime? Findings from two birth cohorts

Background: Recent genome-wide association studies (GWAS) have uncovered genetic variants associated with educational attainment. Polygenic scores derived from these GWAS were shown to predict a suite of psychological characteristics, including cognitive ability, self-control, and interpersonal skill. Deficits in these capacities are associated with greater risk for antisocial behavior. This raises the possibility that molecular-genetic predictors of attainment also forecast antisocial behavior, including crime. We tested this hypothesis in two birth cohorts of more than 3,000 individuals born 20 years and 20,000 km apart.

Method: We studied 920 individuals followed from birth to age 38 years in the Dunedin Longitudinal Study and 2,198 individuals followed from age 2 to age 18 in the E-Risk Longitudinal Study. Based on the most recent GWAS for education, we computed a polygenic score for each participant, reflecting the effects of thousands of genetic variants on attainment. Information on participants' records of crime was obtained by a review of government electronic crime databases. Childhood cognitive ability and self-control, as well as conduct problems across the life-course were assessed in clinical interviews.

Results: Study participants with lower polygenic scores for educational attainment were more likely to have a criminal record as young adults. They were also at greater risk to display a life-course-persistent phenotype of antisocial behavior, characterized by onset in childhood and persistence across life. Individuals' antisocial behavior developed in the context of gene-environment correlations: those with lower polygenic scores were more likely to grew up in poverty and to have a family history of antisocial behavior, but this alone did not explain their greater involvement in crime. Participants convicted of a crime had displayed lower cognitive ability and self-control and poorer academic achievement as children. These characteristics partially mediated genetic associations with criminal offending later in life.

Conclusion: Genetic discoveries made for educational attainment are related to antisocial behaviors, including crime. Part of this association appears to be mediated by personal characteristics that emerge early during childhood, including lower cognitive ability and lower self-control. Genetic associations with the development of antisocial behavior unfold against a backdrop of environmental risk. Our findings show how findings from genomic science can be leveraged for the study of antisocial behavior and crime.

Methods

Sample description

Dunedin cohort

Participants in the first cohort were members of the Dunedin Multidisciplinary Health and Development Study, a longitudinal investigation of health and behavior in a birth cohort. Dunedin Study members (N = 1,037; 91% of eligible births; 52% male) were all individuals born between April 1972 and March 1973 in Dunedin, New Zealand, who were eligible on the basis of residence in the province and who participated in the first assessment at age 3. The cohort represented the full range of socioeconomic status (SES) in the general population of New Zealand's South Island. On adult health, the cohort matches the New Zealand National Health and Nutrition Survey on key health indicators (e.g. body mass index, smoking, visits to the doctor; Poulton, Moffitt, & Silva, 2015). The cohort is primarily white; fewer than 7% self-identify as having non-Caucasian ancestry, matching the demographics of the South Island (Poulton et al., 2015).

Assessments were carried out at birth and ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, and, most recently, 38 years, when 95% of the 1,007 study members still alive took part. At each assessment wave, Study members are brought to the Dunedin research unit for a full day of interviews and examinations. These data are supplemented by searches of official records and by questionnaires that are mailed, as developmentally appropriate, to parents, teachers, and peers nominated by the Study members themselves. The Otago Ethics Committee approved each phase of the study and informed consent was obtained from all study members.

E-Risk cohort

Participants in the second cohort were members of the Environmental Risk (E-Risk) Longitudinal Twin Study, which tracks the development of a birth cohort of 2,232 British children. The sample was drawn from a larger birth register of twins born in England and Wales in 1994-1995 (Trouton, Spinath & Plomin, 2002). Full details about the sample are reported elsewhere (Moffitt & E-Risk Study Team, 2002). Briefly, the E-Risk sample was constructed in 1999-2000, when 1,116 families (93% of those eligible) with same-sex 5-year-old twins participated in home-visit assessments. This sample comprised 55% monozygotic (MZ) and 45% dizygotic (DZ) twin pairs; sex was evenly distributed within zygosity (49% male). Families were recruited to represent the UK population of families with newborns in the 1990s, on the basis of residential location throughout England and Wales and mother's age. Teenaged mothers with twins were over-selected to replace high-risk families who were selectively lost to the register through non-response. Older mothers having twins via assisted reproduction were under-selected to avoid an excess of well-educated older mothers.

At follow-up, the study sample represents the full range of socioeconomic conditions in the UK, as reflected in the families' distribution on a neighbourhood-level socioeconomic index (ACORN [A Classification of Residential Neighbourhoods], developed by CACI Inc. for commercial use) (Odgers, Caspi, Bates, Sampson & Moffitt, 2012; Odgers, Caspi, Russell, Sampson, Arseneault & Moffitt, 2012). Specifically, E-Risk families' ACORN distribution matches that of households nation-wide: 25.6% of E-Risk families live in "wealthy achiever" neighbourhoods compared to 25.3% nationwide; 5.3% vs. 11.6% live in "urban prosperity" neighbourhoods; 29.6% vs. 26.9% live in "comfortably off" neighbourhoods; 13.4% vs. 13.9% live in "moderate means" neighbourhoods, and 26.1% vs. 20.7% live in "hard-pressed" neighbourhoods. E-Risk underrepresents "urban prosperity" neighbourhoods because such houses are likely to be childless.

Follow-up home visits were conducted when the children were aged 7, 10, 12 and, most recently, 18 years, when 93% of the study members took part. Home visits at ages 5, 7, 10, and 12 years included assessments with participants as well as their mother (or primary caretaker); the home visit at age 18 included interviews only with the participants. Each twin was assessed by a different interviewer. These data are supplemented by searches of official records and by questionnaires that are mailed, as developmentally appropriate, to teachers, and co-informants nominated by the Study members themselves. The Joint South London and Maudsley and the Institute of Psychiatry Research Ethics Committee approved each phase of the study. Parents gave informed consent and twins gave assent between 5-12 years and then informed consent at age 18.

Genotyping and imputation

We used Illumina HumanOmni Express 12 BeadChip arrays (Version 1.1; Illumina, Hayward, CA) to assay common single-nucleotide polymorphism (SNP) variation in the genomes of cohort members. We imputed additional SNPs using the IMPUTE2 software (Version 2.3.1; https://mathgen.stats.ox.ac.uk/impute/impute_v2.html; (Howie, Donnelly, & Marchini, 2009)) and the 1000 Genomes Phase 3 reference panel (1000 Genomes Project Consortium et al., 2012). Imputation was conducted on autosomal SNPs appearing in dbSNP (Version 140; http://www.ncbi .nlm.nih.gov/SNP/; (Sherry et al., 2001)) that were "called" in more than 98% of the samples. Invariant SNPs were excluded. The E-Risk cohort contains monozygotic twins, who are genetically identical; we therefore processed genotypes of one randomly-selected twin per pair and used this data to impute their co-twin's genotype. Prephasing and imputation were conducted using a 50-million-base-pair sliding window. The resulting genotype databases included genotyped SNPs and SNPs imputed with 90% probability of a specific genotype among the non-Maori (white) members of the Dunedin

cohort (n=918) and European-descent (white) members of the E-Risk cohort (n=1,999 children in 1,011 families). We analyzed SNPs in Hardy- Weinberg equilibrium ($p > .01$).

Polygenic Scoring

Polygenic scoring was conducted following the method described by Dudbridge (Dudbridge, 2013) using the PRSice software (Euesden, Lewis, & O'Reilly, 2015). Briefly, SNPs reported in the GWAS results released by the Social Science Genetic Association Consortium (Okbay et al., 2016) were matched with SNPs in the Dunedin Study and E-Risk databases. For each SNP, the count of education-associated alleles was weighted according to the effect estimated in the GWAS. Weighted counts were summed across SNPs to compute polygenic scores. We used all matched SNPs to compute polygenic scores irrespective of nominal significance for their association with educational attainment.

Differences in polygenic scores between those who engage in greater or fewer criminal behaviors may be spurious if these individuals come from populations of different genetic ancestry. Because there may be subtle differences in genetic ancestry even within the European-descent participants of the Dunedin and E-Risk samples, we conducted principal components analysis (Price, Zaitlen, Reich, & Patterson, 2010). Analyses were conducted separately in the Dunedin and E-Risk databases. We computed principal components from the genome-wide SNP data with the EIGENSOFT smartPCA tool (Version 5.0.2; http://www .hsph.harvard.edu/alkes-price/software/; (Price et al., 2006)). In the E-Risk database, one twin was selected at random from each family for principal components analysis. SNP-loadings for principal components were applied to co-twin genetic data to compute principal component values for the full sample. Within each database, we residualized polygenic scores for the first ten principal components estimated from the genome-wide SNP data (9) and standardized residuals to have mean=0, SD=1 for analysis.

Measures

All our measures across the two cohorts are summarised in Table 1.

Crime

In the Dunedin cohort, information on criminal offending was obtained by searching the central computer system of the New Zealand Police, which provides details of all New Zealand convictions and sentences and Australian convictions communicated to the New Zealand Police. We conducted searches following the completion of each assessment at ages 21, 26, 32, and 38 (search completed in 2013). Official records of criminal conviction were available from 14 years of age onwards, the age from which criminal conviction was permissible. We computed a binary measure, capturing criminal behavior up until young adulthood. We coded everyone who was convicted of a crime for the first time after age 19 as a non-offender, to keep this group comparable across both cohorts (because E-Risk participants are younger than Dunedin study members, those presently categorised as non-offenders include individuals who may commit offences at later ages).

In the E-Risk cohort, criminal offending was assessed through UK Police National Computer (PNC) records searched by the UK Ministry of Justice (MoJ). The PNC includes complete history of cautions and convictions after age 10 years for individuals cautioned or convicted in the UK from the year 2000 until February 2016. Because E-Risk cohort members were aged ca. 6 years when the PNC records began, the PNC comprises their complete offending histories from age 10 years forward.

Potential confounding variables

In the Dunedin cohort, families' SES was measured using a 6-point scale that assessed parents' occupational statuses, defined based on average income and educational levels derived from the New Zealand Census. Parents' occupational statuses were assessed when Study members were born and again at subsequent assessments up to age-15 years. The highest occupational status of either parent was averaged across the childhood assessments (9). We divided the measure into groups of low SES (groups 6 and 5—eg, oyster canner, car painter; 19.7%), medium SES (groups 4 and 3—eg, butcher, secretary; 63.4%), or high SES (groups 2 and 1—eg, architect, dentist; 16.9%). Family history was assessed as the proportion of a study member's parents and grandparents with conduct disorder or antisocial personality disorder (Odgers et al., 2007). Diagnoses were ascertained using 8 items from the Family History Screen (Weissman et al., 2000) and the Diagnostic Interview Schedule (Robins et al., 1995) that three informants per each family member reported on (e.g., the participant and both of their parents). We standardized the variable to M=0, SD=1 for our analyses.

In the E-risk cohort, families' SES was defined at age 5 using a standardized composite of parents' income, education and social class, which loaded significantly onto one latent factor (Trzesniewski, Moffitt, Caspi, Taylor, & Maughan, 2006). The latent factor was divided into tertiles, thus, 33% of families were characterized as living in a low SES situation during childhood. Fathers' and mothers' lifetime history of antisocial personality disorder was reported by mothers when the children were 5 years old, using the Young Adult Behavior Checklist (Achenbach, 1997), modified to obtain lifetime data and supplemented with questions that assessed the (lifetime) presence of symptoms of antisocial personality disorder (Caspi et al., 2001). We averaged reports about mothers' and fathers' symptoms to obtain a summary measure of parental antisocial behavior and standardized the variable to M=0, SD=1 for our analyses.

Potential mediating variables

In the Dunedin cohort, *truncated education* was assessed using a measure that indicated whether students had sat their English or Maths school certificate exam, a national examination held at about 15 years of age that is the most basic qualification in New Zealand [roughly equivalent to a high school degree in the United States (Kennedy 1981)]. Children's *low self-control* was measured using nine measures of self-control: observational ratings of children's lack of control (ages 3 and 5) and parent, teacher, and self-reports of impulsive aggression, hyperactivity, lack of persistence, inattention, and impulsivity (ages 5, 7, 9, and 11) (Moffitt et al., 2011). The measures were positively and significantly correlated. Based on principal components analysis, the standardized measures were averaged into a single composite score (M = 0, SD = 1), comprising multiple ages and informants, with strong internal reliability $\alpha = 0.86$. Children's *cognitive ability* was assessed at ages 7, 9, and 11 years by the Wechsler Intelligence Scale for Children – Revised (WISC-R)(Wechsler, 1974). IQ scores for the three ages were averaged and standardized. Information on children's academic difficulties was obtained as part of children's psychometric assessment at ages 7 and 9, when mothers were asked about their children's performance reading, printing, arithmetic and spelling. We averaged mothers' responses across ages. Children's *truancy* was assessed at ages 7, 9 and 11 using an item from the Child Behavior Checklist asking mothers whether their child "Skips school, truants, or runs away from school" and an item from the Teacher Report Form asking teachers whether there had been "Truancy or unexplained absence". The response options were "Not true", "Somewhat or sometimes true", "Very true or often true". Truancy was considered present if mother or teacher had endorsed the item as sometimes or often true at age 7, 9 or 11.

In the E-Risk cohort, *truncated education* was assessed based on study members' report of their highest educational achievement at age 18. Study members' education was categorized

as truncated if they did not obtain or scored low (grade D-G) on their General Certificate of Secondary Education (GCSE). GSCEs are a standardized examination taken at the end of compulsory education at age 16. Study members' education was categorized as not truncated if they had obtained a GSCE of grade A*-C, reported A-level qualifications [required for university entrance] or were currently studying to A-level standard. Children's *self-control* during their first decade of life was measured using a multi-occasion/multi-informant strategy, following Moffitt et al. (2011) in the Dunedin Study. Briefly, a self-control factor was estimated via nine measures, including observational ratings of children's lack of control (age 5 years), parent and teacher reports of poor impulse control (ages 5, 7 and 10 years), self-reports of inattentive and impulsive behavior (age 7 years), and interviewer judgments of the personality trait of conscientiousness (age 10 years) (Beckley et al., 2016). Children's *cognitive ability* was individually tested at age 5 using a short form of the Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R; Wechsler, 1990) comprising Vocabulary and Block Design subtests. IQs were prorated (i.e., the full-scale IQ score was estimated from two subscales) following procedures described by Sattler (1992, pp. 998–1004). Children's *academic difficulties* were assessed using teacher reports when the children were 7 and 10 years old. Teachers were asked whether children's current Math and English performance was: 'far below average' (1), 'somewhat below average' (2), 'average' (3), 'somewhat above average' (4) or 'far above average' (5), compared with pupils of the same age. Scores were averaged across academic subjects, averaged again across age 7 and 10 and reverse-coded to derive a global measure of academic problems at primary school. Children's *truancy* was assessed at ages 7 and 10 using an item from the Child Behavior Checklist asking mothers whether their child "Skips school, truants, or runs away from school" and an item from the Teacher Report Form asking teachers whether there had been "Truancy or unexplained absence". The response options were "Not true", "Somewhat or

sometimes true", "Very true or often true". Truancy was considered present if mother or teacher had endorsed the item as sometimes or often true at age 7 or 10.

Life-course trajectories of antisocial behavior

Previous research revealed four life-course trajectories of study members' antisocial behavior in the Dunedin cohort (Odgers et al., 2008). Briefly, antisocial conduct problems were assessed at ages 7, 9, 11, 13, 15, 18, 21, and 26 years through scoring six key symptoms of DSM-IV conduct disorder as being present or absent at each age: physical fighting, bullying others, destroying property, telling lies, truancy, and stealing (American Psychiatric Association, 1994). Other DSMIV CD symptoms were not used because they did not cover the study's age span or had very rare prevalence. Each of the six conduct problem symptoms was operationalized through multiple items collected at each age: the symptom was considered present if any item in the set was endorsed by a reporter. Symptoms were adapted across the age span to ensure that the measures were developmentally appropriate. Growth mixture modeling was used to identify subgroups of individuals that follow unique trajectories in their antisocial behavior over time (Muthen, 2004; Muthen & Shedden, 1999). A series of models assuming different numbers of trajectory groups was fitted to the data. A four-class model represented the best empirical fit to the data according to several indices of model fit and classification accuracy (Odgers et al., 2008). The majority of study members (51%) fell into the "low antisocial behavior" trajectory group, characterised by low levels of antisocial behavior across time. Fewer members displayed antisocial behavior either only in childhood ("childhood-limited" group; 22%) or adolescence ("adolescent-limited" group; 18%). A small number of study members displayed persistently high levels of antisocial behavior across all developmental phases ("life-course persistent" group; 8%).

Statistical analyses

We first examined univariate associations between polygenic scores and confounding and mediating variables using linear regression models. We then tested whether polygenic scores, confounding and mediating variables predicted young adult's criminal convictions using modified Poisson regression models with robust standard errors (Zou, 2004). For all analyses conducted in the E-Risk cohort, we used cluster-adjusted standard errors to correct for clustering due to relatedness between twins. All analyses were conducted in Stata version 12.0 (StataCorp, 2011).

Results

Do children's polygenic scores predict their future involvement in crime?

In both cohorts, children with lower polygenic scores were more likely to have an official record of criminal behavior as young adults (**Table 2**). The effect sizes were nearly identical across the two cohorts (**Figure 1**). The increase in risk was modest: children with a polygenic score 1 standard deviation below the mean were approximately 1.3 times more likely to be convicted of crime by the time they were young adults.

Do children's polygenic scores predict their criminal behavior over and above SES and family history?

In both cohorts, children who grew up in a low-SES household and had relatives who displayed antisocial behaviors were more likely to have a criminal record by young adulthood (**Table 2**). Furthermore, we observed a gene-environment correlation whereby children with lower polygenic scores were more often born into families of low SES and, in the E-Risk cohort, with a familial history of antisocial behavior (**Table 3**). However, even after accounting for SES and family history, children's polygenic scores continued to forecast their involvement in crime (**Table 2**).

Can truncated education explain why polygenic scores for education predict crime?

Polygenic risk for offending is likely explained by intermediate phenotypes, rather than reflecting "genes for crime". Of the possible explanations for why a polygenic score for education predicted crime, perhaps the most obvious one is that it exerts its influence via a person's truncated school career. Indeed, in both cohorts, children with lower polygenic scores were more likely to leave school at the earliest opportunity (**Table 3**) and those who left school early more often had a criminal record by young adulthood (**Table 4, Panel A**).

However, two findings indicate that truncated education alone does not fully explain our results. First, the polygenic score for education continued to predict crime even after we included truncated education in our model (**Table 4, Panel A**). Second, in the E-Risk cohort, cautions and convictions were recorded from age 10 onwards; examining their distribution across ages revealed that the majority of crime occurred before the end of compulsory education. We therefore turned our attention to early childhood (before age 10) and investigated whether behaviors that emerge during this time mediated some of the genetic influence on crime.

Do childhood characteristics mediate the effect of polygenic scores on crime?

Criminological theory postulates three central variables/characteristics that increase a person's risk of engaging in criminal behavior: Low-self control (Gottfredson & Hirschi), neuropsychological abnormalities (mostly Temi I think?) and a lack of commitment to social norms and institutions, such as school (Hirschi, 1969). We examined these characteristics as they emerged in childhood and investigated whether they could account for polygenic influences on crime. In both cohorts, children with lower polygenic scores showed greater levels of all of these risk factors: they performed less well on tests of neuropsychological functioning, displayed lower self-control, had more difficulties in school and truanted more often (**Table 3**). These problems in turn predicted future offending (**Table 4, Panel B**). Including all possible mediators in the same model reduced the prediction of children's polygenic scores on young-adult crime by approximately one third. This finding indicates that part of the reason why children with lower polygenic scores are more likely to get caught for crimes as young adults is that they display a constellation of psychological and behavioural risk factors at a young age.

Are polygenic scores associated with life-course trajectories of antisocial behavior?

To test whether children's polygenic score would predict their antisocial behaviors other than criminal convictions and beyond early adulthood, we next focused our analyses on the members of the Dunedin cohort, who have been followed up into adulthood. Each member of the cohort has previously been assigned to a developmental trajectory group based on the persistence of their antisocial behavior across childhood, adolescence and adulthood (Odgers et al., 2008). We examined whether cohort members who displayed different developmental profiles of antisocial behavior across time differed in their polygenic risk for low educational attainment (**Figure 3**). Our results show a dose-response relationship between the persistence of antisocial behavior over time and study members' polygenic scores for educational attainment. Study members who had never displayed any antisocial behavior had the highest polygenic scores, whereas those who behaved antisocially either only as children or adolescents, had lower polygenic risk scores. Study members who displayed antisocial behavior persistently across childhood, adolescence and adulthood had the lowest polygenic scores. These findings show that the association between children's polygenic scores and antisocial behavior is not confined to criminal convictions in young adulthood but extends to other types of antisocial conduct problems and across the life-course.

Table 1. Measures and descriptive information in Dunedin and E-Risk cohorts.

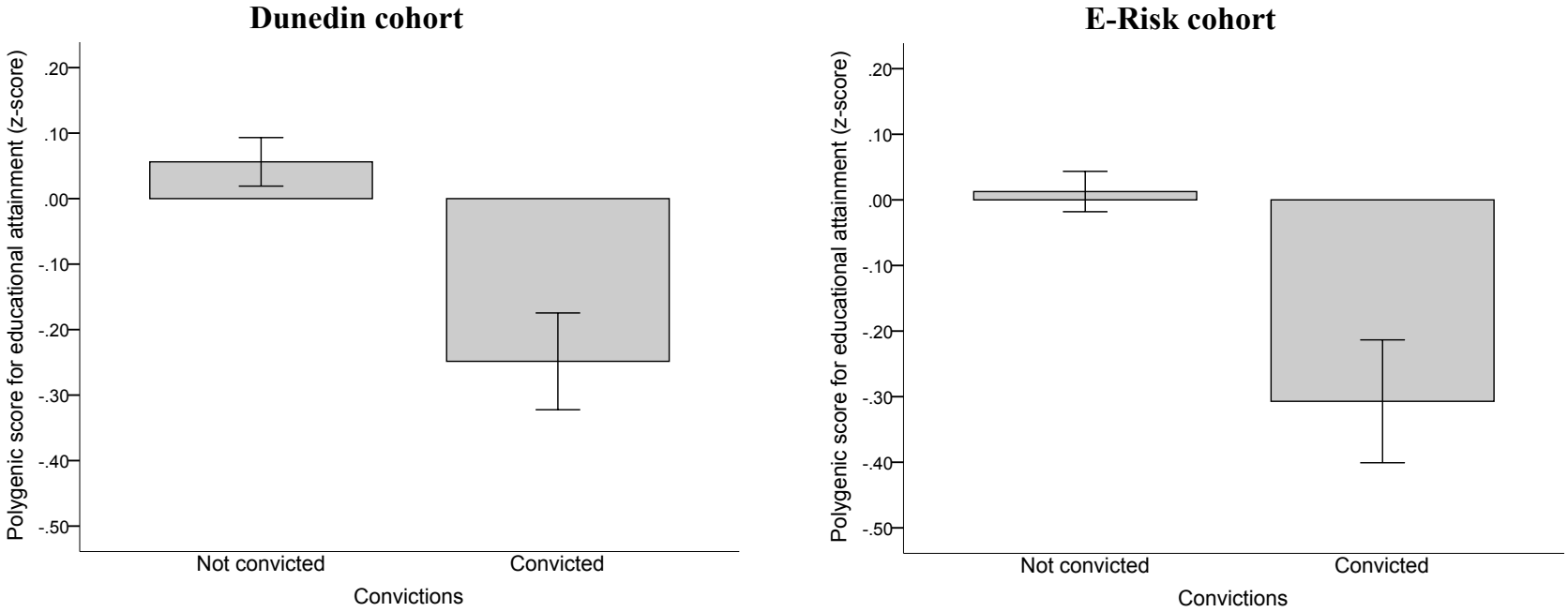
	Dunedin cohort born in 1972/1973 in Dunedin, NZ	E-Risk cohort born in 1994/1995 in the UK
	Measure or data source	Measure or data source
Polygenic score	Polygenic score for educational attainment	Polygenic score for educational attainment
Criminal record in young adulthood	Official records of convictions, age 14 onwards	Official records of cautions/convictions, age 10 onwards
Potential confounding variables		
Family socioeconomic status	Parents' occupational status composite, averaged across children's birth to age 15 divided into low, medium and high SES	Parents' income, education and social class composite, defined at age 5, divided into low, medium and high tertiles
Family history of antisocial behavior	Proportion of parents and grandparents with antisocial personality disorder	Averaged score of mother-reported antisocial personality disorder symptoms in both parents
Potential mediating variables		
Truncated education	Variable indicating whether participants sat the NZ English or Maths school certificate exam at age 15	Variable indicating whether participants obtained no or only a low-graded general certificate of secondary education (GSCE) at age 15/16

Table 1 (continued)

	Measure or data source	Measure or data source
Low self-control	Composite of interviewer, parent, teacher, and self-reports of children's lack of control, hyperactivity, lack of persistence, inattention, and impulsivity, across ages 3-11.	Composite of interviewer, parent, teacher, and self-reports of children's lack of control, hyperactivity, lack of persistence, inattention, and impulsivity, across ages 5-10.
Cognitive ability	Total IQ scores on the Wechsler Intelligence Scale for Children—Revised (WISC-R; Wechsler, 1974), averaged across ages 7-11	Prorated total IQ score on short form of Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R; Wechsler, 1990) at age 5
Low educational attainment	Mother reports of academic performance, averaged across ages 7 and 9	Teacher reports of academic performance, averaged across ages 7-10
Truancy	Any truancy at ages 7, 9 or 11, as reported by mother or teacher	Any truancy at ages 7 or 10, as reported by mother or teacher
Antisocial behavior beyond young adulthood		
	Trajectories of conduct problems across ages 7-26 based on previous analyses (Odgers et al., 2008)	-

Note: NZ = New Zealand, UK = United Kingdom

Figure 1. Mean differences in polygenic scores for educational attainment, between young adults with versus without a criminal record.



Note: Error bars reflect standard errors, with robust standard errors in the E-Risk cohort.

Table 2. Predicting young adults' involvement in crime from polygenic risk, low SES and family history of antisocial behavior.

	Dunedin cohort		E-Risk cohort	
	Univariate	Multivariate	Univariate	Multivariate
Predictor	IRR (95% CI)	IRR (95% CI)	IRR (95% CI)	IRR (95% CI)
Polygenic risk for low educational attainment[1]	1.29 (1.13, 1.47)	1.24 (1.08, 1.42)	1.33 (1.13, 1.55)	1.21 (1.02, 1.43)
Low[1] childhood SES	1.65 (1.32, 2.08)	1.46 (1.15, 1.85)	2.38 (1.92, 2.96)	2.09 (1.63, 2.66)
Family history of antisocial behavior	1.29 (1.15, 1.44)	1.23 (1.09, 1.38)	1.44 (1.28, 1.63)	1.23 (1.06, 1.42)

Note: IRR = Incidence Rate Ratio, CI = Confidence Interval

[1]Polygenic scores and SES were reverse-coded in these models, so that estimates indicate whether children with lower polygenic scores or lower SES had greater rates of criminal involvement as young adults.

Table 3. Univariate associations of polygenic score for educational attainment with potentially confounding and mediating variables.

	Dunedin cohort	E-Risk cohort
	B (95% CI)	B (95% CI)
Low childhood SES	-.25 (-.36, -.15)	-.27 (-.34, -.20)
Family history of antisocial behavior	-.01 (-.05, .08)	-.04 (-.10, .01)
Truncated education	-.35 (-.53, -.17)	-.36 (-.48, -.25)
Low self-control	-.13 (-.20, -.06)	-.06 (-.11, .00)
Neuropsychological problems	-.21 (-.28, -.15)	-.14 (-.19, -.09)
Academic difficulties	-.19 (-.26, -.13)	-.14 (-.20, -.09)
Truancy	-.33 (-.58, -.07)	-.19 (-.46, .08)

Table 4. Mediation of polygenic risk score prediction of crime via truncated education and early childhood risk factors.

Panel A. Mediation of polygenic risk score prediction of crime via truncated education.

	Dunedin cohort		E-Risk cohort	
	Univariate	Multivariate	Univariate	Multivariate
Predictor	IRR (95% CI)	IRR (95% CI)	IRR (95% CI)	IRR (95% CI)
Polygenic risk for low educational attainment[1]	1.29 (1.13, 1.47)	1.20 (1.05, 1.36)	1.33 (1.13, 1.55)	1.19 (1.02, 1.39)
Truncated education	3.23 (2.46, 4.24)	3.04 (2.30, 4.01)	4.27 (3.27, 5.59)	4.02 (3.05, 5.28)

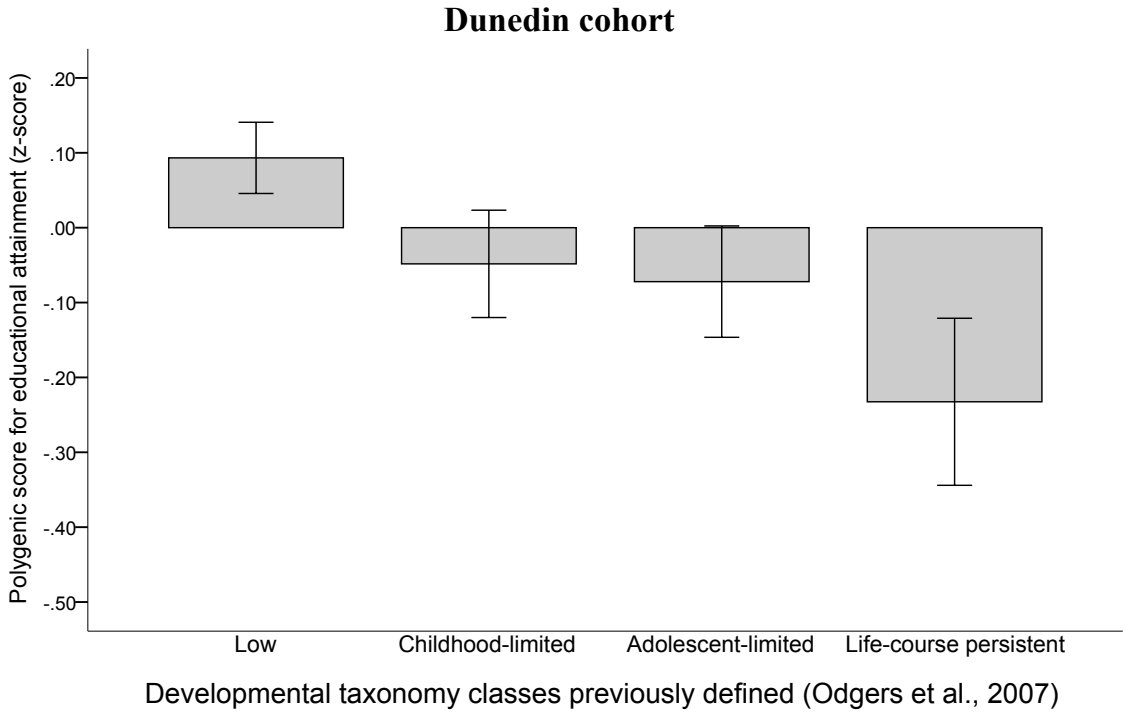
Panel B. Mediation of polygenic risk score prediction of crime via early childhood risk factors.

	Dunedin cohort		E-Risk cohort	
	Univariate	Multivariate	Univariate	Multivariate
Predictor	IRR (95% CI)	IRR (95% CI)	IRR (95% CI)	IRR (95% CI)
Polygenic risk for low educational attainment[1]	1.29 (1.13, 1.47)	1.23 (1.07, 1.42)	1.33 (1.13, 1.55)	1.23 (1.05, 1.43)
Low self-control	1.54 (1.39, 1.71)	1.50 (1.31, 1.73)	2.01 (1.79, 2.26)	1.81 (1.59, 2.06)
Neuropsychological problems	1.18 (1.02, 1.35)	.96 (.80, 1.15)	1.56 (1.34, 1.82)	1.12 (.94, 1.34)
Academic difficulties	1.15 (1.00, 1.33)	.90 (.75, 1.07)	1.80 (1.54, 2.11)	2.34 (1.46, 3.74)
Truancy	2.45 (1.72, 3.50)	1.49 (.97, 2.28)	5.75 (3.78, 8.75)	1.19 (.99, 1.41)

Note: IRR = Incidence Rate Ratio, CI = Confidence Interval

[1]Polygenic score was reverse-coded in these models, so that estimates indicate whether children with lower polygenic scores had greater rates of criminal involvement as young adults.

Figure 3. Mean differences in polygenic scores for educational attainment, for members of developmental trajectories of antisocial behavior



Note: Error bars reflect standard errors.