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EARLY LIFE RISKS, ANTISOCIAL TENDENCIES, AND PRETEEN DELINQUENCY*

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Abstract

Early age-of-onset delinquency and substance use confer a major risk for continued criminality, alcohol and drug abuse, and other serious difficulties throughout the life course. Our objective is to examine the developmental roots of preteen delinquency and substance use. Using nationally representative longitudinal data from the UK Millennium Cohort Study ($n = 13,221$), we examine the influence of early childhood developmental and family risks on latent pathways of antisocial tendencies from ages 3 to 7, and the influence of those pathways on property crime and substance use by age 11. We identified a normative, non-antisocial pathway; a pathway marked by oppositional behavior and fighting; a pathway marked by impulsivity and inattention; and a rare pathway characterized by a wide range of antisocial tendencies. Children with developmental and family risks that emerged by age 3—specifically difficult infant temperament, low cognitive ability, weak parental closeness, and disadvantaged family background—face increased odds of antisocial tendencies. There is minimal overlap between the risk factors for early antisocial tendencies and those for preteen delinquency. Children on an antisocial pathway are more likely to engage in preteen delinquency and substance use by age 11, even after accounting for early life risk factors.

Social scientists have long shown that some youth first engage in delinquency and substance use before the age of 13 years, and such early age-of-onset antisocial behavior increases the risk of long-duration, high-frequency involvement in crime and substance abuse (Blumstein

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et al., 1986; Bosick, Bersani, and Farrington, 2015; Ellickson, Tucker, and Klein, 2003; Thornberry and Krohn, 2003). Research has also identified childhood “antisocial tendencies” (hereafter called AST), such as physical aggression, disobedience, impulsivity, and inattention, that heighten the risk of preteen delinquency and substance use (Farrington, 2003; Tremblay, 2014). Prominent theories in criminology and developmental psychopathology suggest that preteen delinquency and substance use originate in early childhood developmental and family-related disadvantages that increase the risk of AST (Gottfredson and Hirschi, 1990; Moffitt, 1993). Our objective in this study is to test whether childhood AST do in fact link early developmental and family disadvantages with preteen delinquency and substance use.

We use nationally representative prospective data from the UK Millennium Cohort Study (MCS). The MCS collected information on a recent birth cohort of 9-month-olds and their parent(s)/caregiver(s) at multiple points throughout childhood. It includes over a decade’s worth of longitudinal data on a large ($n = 13,221$), contemporary, nationally representative sample of British children born at the start of the millennium. Drawing from criminological theories of early-onset delinquency, we first examine whether developmental and family factors assessed from age 9 months to 3 years predict latent AST pathways from ages 3 to 7 years. We then place these AST pathways as intermediate steps linking early developmental and family risks with delinquency and substance use at age 11.

CRIMINOLOGICAL THEORIES EMPHASIZING EARLY AGE OF ONSET

Past theoretical work has highlighted two main potential sources of early-onset delinquency and substance use (Farrington, 2003; Tremblay, 2014). The first—most directly stated by Moffitt (1993)—stems from neuropsychological deficits that emerge *in utero* or shortly after birth and cause persistent adjustment problems. The second—most directly stated by Gottfredson and Hirschi (1990)—comes from family-related risks that prevent very young children from acquiring the skills and resources needed to inhibit later antisocial impulses. In accordance with such perspectives, evidence indicates that children at risk for preteen delinquency exhibit early warning signs, such as physical aggression, oppositional behavior, inattention, and impulsivity, that continue past the toddler years (Caspi et al., 1995; Loeber and Farrington, 2000). Brought about by developmental and family risks in childhood, these behavioral tendencies emerge as “prodromes” of early-onset delinquency. In this section, we begin by reviewing the evidence for longitudinal linkages between childhood AST and subsequent delinquency. We then review evidence on the developmental precursors of early AST, and the extent to which they overlap with the correlates of subsequent delinquency.

THE ANTISOCIAL TENDENCIES OF EARLY-ONSET DELINQUENTS

It is well known that childhood physical aggression and opposition-defiance are among the most consistent predictors of adolescent, as well as adult, offending and criminal justice system involvement (Gottfredson and Hirschi, 1990; Moffitt, 1993). White and colleagues (1990) showed that children who are aggressive and disobedient at age 3 are at increased risk of antisocial behavior at age 11 as well as delinquency and police contact at age 15. Maughan and colleagues (2000) observed that youth who were physically aggressive (e.g., who fought and bullied) during early adolescence were more likely to be arrested by age 16.

Using longitudinal data from the British Cohort Study (born 1970), Murray and colleagues (2010) found that age 5 conduct problems predicted both conduct problems at age 10 and adult criminal convictions. Another study similarly found that conduct problems at age 8 (e.g., fighting, disobedience) predicted whether an adolescent had been convicted of a felony by age 20 (Savolainen et al., 2010). Though childhood physical aggression and opposition are robust predictors of problem behaviors at older ages, for most people the prevalence of aggressive and oppositional behaviors reaches its peak very early in childhood and then normatively declines (Brame, Nagin, and Tremblay, 2001; Broidy et al., 2003; NICHD Early Child Care Research Network, 2004; Tremblay et al., 2004; van Lier et al., 2009).

There is also evidence linking childhood impulsivity and inattention to early-onset delinquency (for reviews see Farrington, Loeber, and van Kammen, 1990; Hawkins et al., 2000). For instance, behavioral impulsivity (i.e., a disinhibited behavioral response) at age 3 predicts antisocial behavior during late childhood and conduct disorders during adolescence (Caspi et al., 1995). Similarly, children suffering from attention problems (e.g., lack of concentration, restlessness, tendency to daydream) at age 5 were more likely to engage in delinquency at age 14 (Bor, McGee, and Fagan, 2004). In a study by Farrington (1989), boys ages 8 to 10 who showed signs of high impulsivity on psychomotor tests or who were rated by teachers as lacking concentration or being restless had heightened levels of aggression in adolescence, and were more likely to be convicted of a violent crime by age 32.

Children who exhibit multiple forms of AST (i.e., combinations of aggression, disobedience, impulsivity, and inattention) appear to possess a wide-ranging capacity for antisocial behavior, which increases the risk for delinquency and substance use in adolescence (Loeber and Schmaleng, 1985a, 1985b). For instance, six-year-olds rated by kindergarten teachers as oppositional *and* hyperactive are more likely to persist in violent behavior through age 15 (Nagin and Tremblay, 2001). In addition, although an aggressive temperament, lying to one's parents, and being impulsive are all independent predictors of later delinquency (Loeber and Farrington, 2000), in concert, they further increase a child's risk for antisocial behavior in adolescence and into young adulthood (Donker et al., 2003).

EARLY LIFE RISK FACTORS FOR ENDURING ANTISOCIAL TENDENCIES

Criminological theories that emphasize age of onset point to both developmental and family-related disadvantages as the central risk factors for both childhood AST and early-onset delinquency. For instance, children who suffer from neuropsychological deficits—subclinical levels of brain damage stemming from congenital disabilities, birth defects, heavy fetal alcohol exposure, severe maltreatment, or accidental trauma—are often irritable, inattentive, and impulsive (Moffitt, 1993). Impaired executive functioning has also been linked to problems with learning, verbal ability, memory, literacy, attention, and hyperactivity. Children who have these deficits begin life “on the wrong foot” (Moffitt, 1993: 682) by straining their relations with their primary caregivers. These effects can be compounded when high-risk children are raised in adverse home environments (Moffitt et al., 2011). Theoretically, then, children with neuropsychological deficits are subject to cumulative disadvantage in the realms of school, friendships, and work, embarking on a

pathway fraught with such intractable difficulty that a life course filled with persistent antisocial behavior is a more likely outcome.

Empirically, several studies have linked childhood factors such as cognitive ability and temperament with later juvenile delinquency and criminal involvement in adulthood (for a review, see Farrington and Welsh, 2007; Portnoy et al., 2013). For example, Piquero and colleagues (2007: 196) found that low verbal intelligence at age 7 was associated with a higher arrest frequency by age 33. Similarly, Savolainen and colleagues (2010) found that a combination of low verbal intelligence at age 8 and impulsivity at age 15 amplified the probability that an adolescent would experience a felony conviction by age 20. Infants with difficult temperaments are also at increased risk of long-term antisocial behavior (Moffitt et al., 2002; White et al., 1990; see also Farrington and Welsh, 2007: 46). For instance, Caspi (2000) found that children with an “undercontrolled” temperament at age 3 were more likely to have criminal convictions between ages 18 and 21.

Similar to Moffitt’s (1993) theory, Gottfredson and Hirschi’s (1990) general theory of crime locates the etiology of preteen delinquency in early life disadvantages – specifically family disadvantages. According to their theory, when parents fail to monitor, recognize, and correct evidence of early antisocial behavior, children are not properly socialized to resist the temptations of the present moment. Thus, childhood impulsivity is a key antisocial tendency that increases the risk of early-onset delinquency (Caspi et al., 1995; Moffitt et al., 2011; Savolainen et al., 2010). Consistent with the contention that parental socialization establishes self-control, parent-child interaction variables (i.e., closeness, supervision, involvement, and discipline) are robust predictors of early-onset antisocial behavior (Farrington and Welsh, 2007).

Past work has identified additional family risk factors that can also predispose children to childhood AST and subsequent early-onset delinquency. Regarding AST, Tremblay and colleagues (2004) used parental and family characteristics measured soon after birth to predict the child’s likelihood of engaging in physical aggression from ages 5 months to 42 months. Their results indicated that children born to mothers who had previously engaged in antisocial behavior during high school exhibited significantly higher rates of physically aggressive behavior. Research has also found that single-parent family structure and being born to teenage parents both increase the risk of early childhood AST (Nagin and Tremblay, 2001; Tremblay et al., 2004). In addition, parental substance use has been identified as a risk factor for offspring inattention, impulsivity, hyperactivity problems, poor social skills, and eventually delinquency and substance use (Gibson, Piquero, and Tibbetts, 2000; Ware et al., 2014).

Research also has linked early family risk factors with later delinquency and justice system contacts. Dodge, Greenberg, and Malone (2008) documented that children of parents with low educational attainment and occupational status were at heightened risk for violent behavior during late adolescence. Similarly, Maughan and colleagues (2000) found that youth raised in low-income families tended to report higher levels of physical aggression throughout adolescence. Bor and colleagues (2001) examined how a child’s family characteristics at age 5 were associated with antisocial behavior and police contact at age 14.

They found that low maternal education, family poverty, and single-parent family structure in early childhood were predictive of delinquency and criminal justice contact during adolescence. Thus both parental characteristics and broader socioeconomic risk may contribute jointly to antisocial behavior over time (Shaw, Bell, and Gilliom, 2000).

TESTING THE LONGITUDINAL PATHWAY

The prospective links between (1) early disadvantages and childhood AST, (2) childhood AST and preteen delinquency, and (3) early disadvantages and adolescent delinquency provide suggestive evidence for a major theoretical proposition: that early risk factors lead to preteen delinquency and substance use by triggering (or preventing the inhibition of) AST in early childhood. Still, research has not yet tested the entire pathway leading from childhood disadvantages (i.e., developmental and family disadvantages) to AST, and then from AST to early-onset delinquency. As scholars have noted, there appears to be overlap between the known risk factors for early aggressive, disruptive, and impulsive behavior and those for later adolescent delinquency (Loeber, Burke, and Pardini, 2009; Shaw and Gross, 2008). Yet not only does early antisocial behavior overpredict teenage antisocial behavior (Shaw and Gross, 2008), but also the limited available evidence indicates that early risk factors may be independent predictors of later delinquency net of early antisocial behavior. For example, Heron and colleagues (2013) found that early conduct problems, maternal substance use, and socioeconomic disadvantage all independently accounted for substantial proportions of youths' risk for later problem marijuana use. In addition, Murray and colleagues (2010) found that conduct problems at age 5 did not explain the associations between maternal smoking, family structure/size, or neighborhood poverty and conduct problems at age 10.

The theorized longitudinal pathway from early disadvantages to AST to preteen delinquency has two main implications. First, if the theory is correct, then AST and preteen delinquency should share the same early risk factors. If there is minimal overlap between the sets of risk factors, then early disadvantages cannot operate on preteen delinquency via AST. Second, assuming that AST and early-onset delinquency do have common risk factors, if the theory is correct then AST should account for the association between those risk factors and preteen delinquency. If AST tendencies do not explain this association, this would suggest that these early risk factors do not cause preteen delinquency by causing intractable and self-reinforcing behavior problems in childhood.

In this study, we begin by examining how early life developmental and family factors influence latent pathways of AST across early to middle childhood. We operationalize early childhood disadvantages as developmental factors (i.e., birth complications, difficult infant temperament at 9 months, and low cognitive ability at age 3) and family factors (i.e., parental substance use, family structure, parental closeness, and socioeconomic background, all at 3 years). Using multilevel latent class models, we operationalize AST as configurations of misconduct, temper problems, impulsivity, inattention, and low perseverance from ages 3 to 7. Instead of using cross-sectional "snapshots" of early childhood behavior, our examination of AST pathways helps us to capture more serious and persistent behavioral tendencies that are likely to precede delinquency, rather than transient

problems (Campbell, Shaw, and Gilliom, 2000). Our first contribution is to test whether the same risk factors predict childhood AST as well as preteen property crime and substance use. The latter two outcomes encompass two forms of delinquency that are rare for 10–11 year olds, which enables us to capture their early onset (Tremblay, 2014). Our second contribution is to examine whether childhood AST account for the association of early risk factors with these two forms of preteen delinquency.

METHOD

DATA

The ongoing MCS recruited all nine-month-old children born between September 1, 2000 and January 11, 2002 who were alive and residing in a sample of electoral wards from England, Northern Ireland, Scotland, and Wales at nine months of age. The MCS oversampled electoral wards with high child poverty, high concentrations of Asian (i.e., Indian, Pakistani, and Bangladeshi) and Black (i.e., Black African, Black Caribbean, and Black other) families, and those in Northern Ireland, Scotland, and Wales (Hansen, 2014). The sampling frame of children from these wards was compiled from lists from the Child Benefit register (which covers virtually all UK children, apart from new immigrants) supplemented by Health Visitors lists (which include information on new immigrants). Approximately 2.8% of families were excluded from the lists because they were sensitive cases (e.g., recent child death in the family; child taken into protective care); these families were more likely to be from disadvantaged areas. An additional 6.9% of families opted out of the study; these families were more likely to be from socioeconomically advantaged areas. In total, parents of 18,552 nine-month old children participated (approximately 91% of all eligible children; for more details see Plewis, 2007).

Follow-up parent surveys were completed when children were ages 3, 5, 7, and 11 years. In addition, data were collected from children during their final year in primary school (modal age 11). By this most recent wave, 13,287 children had been retained, including 13,221 children who had not yet transitioned to secondary school¹ (69% of all MCS families and 81% of eligible children whose families had not emigrated, who had not permanently withdrawn, and who were living; Gallop et al., 2013). Retention was similar among the four UK countries, though statistically significant ($p < .05$) differences were found between respondents and non-respondents by age 11 across a range of background characteristics. Using a logit model, Mostafa (2014) showed that age 11 retention was more likely among girls, Pakistani and Bangladeshi children, and youth whose mothers were older (i.e., age 30–40 at first birth), attempted breastfeeding, or had a mortgage. The MCS dataset includes a weight that accounts for nonrandom sample attrition by age 11 (based on the inverse of the predicted probabilities from the logit model predicting age 11 retention), as well as MCS families having unequal probabilities of sample inclusion (due to the stratified design and to non-response) when the child was 9 months old.

¹The 66 MCS students who had transitioned to secondary school at the time of interview were significantly older (mean age 11.5) than the 13,221 students who were still in primary school at the time of the most recent survey administration. We excluded the small number of secondary students to focus our analyses on delinquency and substance use that occurs before the end of primary school (i.e., year 5 in the UK).

We use data from the 13,221 children who had completed surveys while still in primary school. To handle item-missing data, we relied on the “mi” command in Stata 13 to impute 10 datasets using chained regressions, where in a series of steps: 1) each item with missing data is first regressed on all of the other variables that appear in the models; 2) item-missing data is replaced with the predicted values from each equation; and 3) the items with predicted values are used in successive equations (Johnson and Young 2011). Following Rubin’s (1987) rules, we used the “mi estimate” command to combine results across the 10 datasets and adjust standard errors and significance tests. The percentage of missing values that were imputed for each variable ranged from 0% (for gender) to 18% (for mother reports of mother-child closeness when the child was age 3); the mean percentage was 8.7. Table 1 shows weighted descriptive statistics for our outcome and predictor variables. We used the “svy” command to adjust these descriptive statistics for the complex survey design (clustering by electoral ward, which required adjustments to the standard errors, and the oversampling of high poverty and minority electoral wards, which in combination with non-response required the calculation of weighted point estimates).

FOCAL OUTCOMES: PRETEEN DELINQUENCY

During their final year of primary school (age 11), children completed confidential self-report surveys regarding whether they had ever committed at least one of the following *property crimes*: “taken something from a shop without paying for it”; “written things or sprayed paint on a building, fence or train or anywhere else where you shouldn’t have”; or “on purpose damaged anything in a public place that didn’t belong to you, for example by burning, smashing or breaking things like cars, bus shelters and rubbish bins.” As shown in Table 1, approximately 9% of youth had committed at least one of these offenses by age 11. We also created a *substance use* measure indicating whether or not the youth had previously tried a cigarette (even if it was only a single puff) or had an alcoholic drink (more than a few sips). About 15% of children had used one or both of these substances by age 11.

FOCAL PREDICTORS: EARLY CHILDHOOD DEVELOPMENTAL AND FAMILY RISK FACTORS

Early childhood developmental factors—We examined five parent-reported and investigator-assessed measures. *Easy infant temperament* was mother-reported when children were 9 months old using an established fourteen-item scale tapping children’s positive mood, adaptability to new situations, and regularity (Carey, Fox, and McDevitt, 1977). Scores were standardized to increase interpretability, and high scores indicated that mothers felt that their infant did not have a difficult temperament. *Cognitive ability* was investigator-assessed (Connelly, 2013) at age 3 years using the Bracken School Readiness Composite (Bracken, 2002), which assesses children’s knowledge of colors, letters, numbers, and shapes, and the naming vocabulary subtest of the British Ability Scales II (Elliott, Smith, and McCulloch, 1997), which assesses children’s expressive verbal ability. These age-adjusted scores were averaged ($r = .59$) and then standardized, where higher scores indicated a greater understanding of basic concepts, readiness for school, and verbal ability. *Heavy fetal alcohol exposure* was based on mothers’ retrospective reports when children were infants of whether during pregnancy they drank 7 or more units of alcohol per week or 6 or more units per occasion, similar to Kelly and colleagues (2008). We also

included a dichotomous measure of whether the child had a *low birthweight* (1 = under 2.5kg at birth).

Early childhood family factors—Parent(s) reported on their own past-year *illicit drug use* (1 = neither reported illicit drug use), *alcohol use* (1 = neither reported drinking alcohol), and their *smoking* (1 = neither was a current smoker). We also included measures of whether the child was *born when the mother was less than 20 years of age* (1 = yes) and of *family structure* (measured as a set of dummy variables for married, cohabiting, and single parents). *Mother-child closeness* was assessed via maternal reports using Pianta's (1992) short-form Relationship Scale, which measures mother-child comfort, praise, information sharing, and affection. Scores were standardized, with higher scores indicating greater mother-child closeness. Finally, two indicators of family socioeconomic background were used: *Parent(s') highest educational level* was based on national vocational qualifications ranging from 0 (i.e., no educational diploma) to level 5 qualifications (i.e., postsecondary academic credentials); and *parent(s') highest occupational status* was measured as a set of six mutually exclusive dummy variables indicating employment: 1) in a semi-routine or routine job; 2) a low supervisory or technical job; 3) a small employer or self-employed; 4) an intermediate level job; 5) a managerial/professional job; or 6) not working. With the exception of young mother (measured at age 9 months), all family factors were measured at age 3.

FOCAL MEDIATORS: CHILDHOOD ANTISOCIAL TENDENCIES

To assess AST in childhood, at ages 3, 5, and 7, parents used the “Strengths and Difficulties” questionnaire (Goodman, 2001) to describe their child's behavior. Using a three-point scale of “not true,” “somewhat true,” and “certainly true,” parents were asked to rate the degree to which during the past six months their child: (1) fought with or bullied other children; (2) often had temper tantrums; (3) was generally disobedient; (4) was easily distracted; (5) was impulsive (i.e., did not stop and think before acting); and (6) was not perseverant (i.e., did not see tasks through to the end). The items were recoded to indicate whether each behavior was “certainly” true of the child (coded 1) versus not true or somewhat true (coded 0). Missing values for each item were coded 2.

We used multilevel latent class models estimated in Latent Gold (Vermunt, 2003; Vermunt and Magidson, 2005) to classify how these six indicators of childhood antisocial tendencies were associated at each age as well as over time. These models used children's scores on the behavioral measures to identify typical and qualitatively distinct latent behavioral patterns (classes) based on the observed indicators. In the first level, latent classes, or probabilistic combinations, of the six behavior items were estimated when the child was age 3, 5, and 7. In the second level, a set of latent variables captured the patterns of movement between the latent classes over time. The model thus identified latent pathways of early antisocial tendencies from age 3 to 7.

The model estimation and selection process involves 1) the selection and coding of observed indicators of the latent classes, 2) the estimation of a set of several different models that vary in the number of classes formed, and 3) the selection of the best-fitting model from among

that set. As described above, we used categorical indicators of six behaviors. Following Vuolo and colleagues (2012), to identify these pathways we used information from all families who provided data when the child was age 3, 5, or 7, even if the family had missing data on one or more of the survey items, or they did not participate in the study in one or more of the waves or at age 11.² We then estimated a series of models with up to 9 latent classes at level 1 (within-age behavioral patterns) and up to 8 pathways at level 2 (across-age behavioral change patterns). The Bayesian information criterion (BIC) statistic was used to guide model selection (Macmillan and Eliason, 2003; Vuolo et al., 2012). Appendix A shows a series of BIC statistics for the full series of multilevel latent class analyses of AST, which indicated that the model with the lowest BIC (in italics) had seven latent classes at each age of observation (level 1) and seven latent pathways (level 2). Four of those seven pathways identified four substantively distinct latent patterns of children's AST; we describe those patterns in detail below. The remaining three pathways flagged families with high probabilities of missing data at age 3, at ages 5 and 7, or at all three waves. The 13% of our sample with high probabilities of membership in one of these pathways were set to missing and then assigned to one of the substantive pathways (via multiple imputation) in subsequent analyses.³

In Figure 1, we show how the configurations of these behaviors are nested within patterns of movement over time. In each of the four latent pathways, each line represents the predicted probability of a parent reporting "certainly true" for each item at ages 3, 5, and 7. Children in latent pathway 1—representing approximately 75% of children in this sample—had low probabilities of fighting, having temper tantrums, disobedience, lacking perseverance, or being inattentive or impulsive. The probability of fighting in this pathway was essentially zero from ages 3 to 7. The probability of temper tantrums and impulsiveness was highest at age 3 (at 10% and 12%, respectively) and declined substantially by age 5 (to 2.4% for temper tantrums and 4% for impulsiveness).

Compared to latent pathway 1, children in the remaining three pathways showed significantly higher probabilities of conduct problems, inattention, and impulsivity, albeit in different profiles. Children in latent pathway 2—comprising only 5% of the sample—had high probabilities of parents reporting that all six of the survey items were "certainly true" of their children. Furthermore, the predicted probabilities remained elevated over time. These children had the highest predicted probabilities of all items at all ages. Latent pathway 3—accounting for about 10% of children—was distinguished by a relatively high probability of temper tantrums that declined from ages 3 to 7 (from 64% to 48%). Compared to pathways 1 and 4, the probability of disobedience and fighting was also relatively high, though these behaviors were not as likely as among children in pathway 2. Finally, latent pathway 4—comprising about 10% of children—included children who had relatively high probabilities of inattention, impulsiveness, and lack of perseverance, but low and declining probabilities

²To check whether missing data was influencing the estimation of the AST pathways, in unlisted models we estimated the multilevel latent class models using data only from families who completed surveys at all three waves. In both sets of estimations (i.e., models based upon data using listwise deletion versus those that include missing data), the four substantive AST pathways were almost exactly the same.

³An alternative strategy would be to include a separate dummy variable for the missing AST pathways in our regression models. We found no differences in the overall pattern of results when we used this strategy compared to the findings we show in Tables 2 and 3.

of fighting, temper tantrums, and disobedience. Given these behavioral profiles, we named the latent pathways (1) *normative*, (2) *mixed conduct and attention problems*, (3) *temper problems*, and (4) *attention problems*. Each child was assigned to a latent pathway based upon their modal probability of belonging to these pathways.

CONTROL VARIABLES

In all regression models, we included controls for the child's *male* gender (1 = yes) and race/ethnicity, coded as a set of dummy variables for White British, Black British (Black Caribbean, Black African and Black other), Asian British (Indian, Pakistani, and Bangladeshi), and other. Following Hansen and colleagues (2015), we also included the stratum design variable as a set of dummy variables in all regression models⁴, and we adjusted all standard errors for the clustering of respondents in primary sampling units (electoral wards).

STRATEGY OF ANALYSIS

Our analysis proceeded in four stages. First, as described previously, we used multilevel latent class models to group children into different AST pathways. Second, we used multinomial logistic regression to examine the extent to which early childhood developmental and family risk factors predicted membership into these AST pathways. Third, we used logistic regressions to investigate the effects of early childhood risk factors on self-reported property crime or substance use by age 11. Finally, we re-estimated the models from stage 3, this time introducing the AST pathways as predictors. This allowed us to assess both the independent effects of early risk factors and childhood AST on preteen delinquency, as well as to evaluate the degree to which AST mediated the relationship between childhood risk factors and early-onset delinquency.

RESULTS

To test whether the same developmental and family risk factors predict childhood AST as well as preteen property crime and substance use, we first estimated a multinomial regression model with early childhood risk factors as predictors of the latent AST pathways. The normative pathway served as the reference category against which the other pathways were compared. Table 2 shows that the developmental and family risk factors for membership in the *mixed conduct and attention problems* pathway – characterized by fighting, tantrums, disobedience, distractibility, impulsivity, and low perseverance – and the *temper problems* pathway – characterized by temper tantrums, disobedience, and fighting – were similar. Relative to members of the *normative* pathway, children in these pathways had lower cognitive ability and more difficult temperaments. They also had lower levels of mother-child closeness, had parents with lower levels of education, and were less likely to live in two-parent families, with parents in higher status occupations, and with non-smoking parents. In addition, children in the temper problems pathway, relative to the normative pathway, were also more likely to have had a low birth weight. Children in the mixed

⁴We present unweighted regression estimates in Tables 2 and 3 because many of the measures used to create the attrition weight already appear as covariates in our regression models. It is important to note, however, that the results remain very similar regardless of whether we use weighted or unweighted data.

conduct and attention problems pathway were more likely to be male and less likely to be Asian British or Black British than White British, relative to children in the normative pathway.

The risk factors for membership in the *attention problems* pathway – characterized by impulsivity, inattention, and lack of perseverance – resembled those for membership in the mixed problems and temper problems pathways, with some notable exceptions. Unlike members of those two pathways, children in the attention problems pathway were no less likely to live with parents who did not smoke, and they had similar socioeconomic backgrounds as children in the normative pathway. They were, however, more likely to live in single-parent than married or cohabiting families, they had lower cognitive scores, more difficult temperaments, lower birth weights, and lower mother-child closeness, and they were more likely to be male and less likely to be Black British. Heavy fetal alcohol exposure and parental alcohol and illicit drug use did not distinguish any of the three antisocial pathways from the normative pathway. Notably, protective effects of socioeconomic background were limited to the mixed problems and temper problems pathways, and were most evident at the higher levels of job status. Likelihood of membership in the attention problems pathway did not differ by parental education or occupational status.

Do these AST pathways explain longer-term associations of early childhood disadvantages with preteen delinquency? In Table 3, Model 1 shows odds ratios and confidence intervals from logistic regressions predicting preteen property crime and substance use from early childhood factors. Model 2 then includes the childhood AST pathways.

As shown in Model 1 of the first column, preteen property crime shared some risk factors with the AST pathways, namely cognitive ability, parental smoking, family structure, and parental socioeconomic background. However, infant temperament and mother-child closeness did not predict property crime. In addition, one unique predictor of property crime emerged: The odds of property crime were reduced by a factor of .69 among children whose parent(s) did not use illicit drugs. When the latent pathways were included (see Model 2), the significant effects of the risk factors observed in Model 1 were reduced slightly; however, with the exception of cognitive ability, they remained statistically significant. Compared to the normative pathway, the odds of a property crime were 2.02, 1.65, and 1.46 times greater for the mixed conduct and attention problems, temper problems, and attention problems pathways, respectively.

The right half of Table 3 shows results predicting preteen substance use, before (Model 1) and after (Model 2) the inclusion of the AST pathways. Of the significant predictors of the AST pathways, only families with non-smoking parents, married/cohabiting parents, or closer mother-child relationships had lower odds of substance use. Children had significantly lower odds of having had a drink and/or smoked a cigarette if their parent(s) did not use drugs (odds ratio=.63), or drink alcohol (odds ratio=.63), neither of which significantly predicted AST pathway membership. When the AST pathways were added in Model 2, children in the mixed conduct and attention problems pathway, temper problems pathway, and attention problems pathway had significantly elevated odds of substance use (by factors of 1.57, 1.25, and 1.33, respectively), in comparison to the normative pathway.

As in the property crime model, the addition of the indicators of the AST pathways appeared to have little impact on the coefficients for the early childhood risk factors, although the coefficient for mother-child closeness was reduced to non-significance.

As a supplemental test of our hypothesis, in additional models we used Karlson, Holm, and Breen's (2012) test for indirect effects to assess whether the AST pathways mediated the relationship between the early childhood risk factors and preteen delinquency. In unlisted models, we found that for both property crime and substance use, none of the coefficients for the childhood risk factors showed statistically significant changes (i.e., p for all changes $> .05$) after the AST pathways were included (i.e. when comparing Models 1 and 2 in Table 3).

In final supplemental models we also considered whether the pattern of findings was different for girls and boys. To assess whether gender was a moderator, in unlisted analyses we estimated Model 2 of Table 3 separately for boys and girls, and then used z-tests to compare the equality of the estimates (Clogg, Petkova, and Haritou, 1995). In only 1 of 46 comparisons (23 predictors of each outcome) did the estimates vary significantly by gender (i.e., p for difference $< .05$), and that estimate was for a variable that did not significantly predict the outcome. Importantly, though the rate of easy infant temperament and levels of cognitive ability and mother-child closeness were lower for boys than girls, and though boys had a heightened risk relative to girls of 2 of the 3 AST pathways as well as of property crime and substance use, the association between preteen delinquency and the childhood risk factors and AST pathways did not differ by gender.

DISCUSSION

While past research has independently examined (1) the effect of early risk factors on childhood problem behavior, (2) the effect of childhood problem behavior on early onset delinquency, and (3) the effect of early risk factors on later delinquency, it has remained unclear whether all three factors are part of a single longitudinal pathway. When combined, the constituent propositions of this pathway coalesce into a broader theoretical statement on the developmental antecedents of early-onset delinquency: Early childhood developmental and family disadvantages precipitate childhood antisocial tendencies, which in turn lead to preteen delinquency. If this theorized pathway is accurate, one would expect substantial overlap between the early risk factors for preteen delinquency and childhood AST. That is, if developmental and family problems during infancy and toddlerhood serve as the wellspring for a cascade leading first to childhood AST and then to preteen delinquency, then this latter outcome should be traceable back to the same origin. In addition, the observed association between early risk factors and preteen delinquency should be mediated by childhood AST.

In the current study, we found partial support for this theorized pathway. There was moderate overlap between the risk factors for childhood AST and for preteen delinquency, including cognitive ability and several family factors (mother-child closeness, family structure, socioeconomic background, and parental smoking). At the same time, one of the most consistent risk factors for preteen delinquency – parental substance use during early

childhood (i.e., parental drug use and drinking at age 3) – was not predictive of childhood AST. Furthermore, one of the most consistent predictors of childhood AST – infant temperament – did not predict preteen delinquency, nor did low birthweight. Even more importantly, AST did not mediate the effects of early risk factors on delinquency. Thus, although our findings confirm the previously established associations between early developmental and family disadvantages, early childhood aggression and impulsivity, and delinquency, they do not provide support for the assertion that childhood AST, as operationalized here, represent the major link between early risk factors and preteen delinquency and substance use.

These results appear to contrast with the elaborative models of problem behavior set forth in theories of early-onset delinquency (Gottfredson and Hirschi, 1990; Moffitt, 1993). Under such models, children's low capacities to control their behavior and disadvantaged child-rearing environments that are likely to reinforce early expressions of aggression, temper, and impulsivity represent key risk factors. Theoretically, as children develop under these circumstances, their problem behaviors escalate unchecked in a continuous process that eventually results in early adolescent law violation. Our findings indicate that early risk factors also influence early-onset offending independently from their influences on parent-reported antisocial tendencies assessed repeatedly across ages 3 to 7 years. This finding suggests that early childhood risk factors do not solely have their effects by sparking sequences of gradually escalating problem behaviors, at least as presently assessed and modeled. Instead, this suggests that there may be multiple avenues through which early risks can lead to delinquency (Richters, 1997). Some risk factors may manifest early as childhood AST, which then act as independent predictors of preteen delinquency. Other early life risk factors or disadvantages may emerge later as "sleepers" effects on delinquency as the child enters adolescence (see also Heron et al., 2013; Loeber and Stouthamer-Loeber, 1986; Murray et al., 2010).

These findings may pose challenges for intervention. Because early antisocial behaviors predict later delinquency, behavioral interventions during childhood could have beneficial effects on preteen behavior. Yet since early childhood risk factors are independent predictors of preteen behavior, such interventions would not be enough to erase the family-linked gaps in the eventual onset of preteen delinquency and substance use. Although some of these gaps – for example, those related to parental substance use – might be addressed by directly targeting the risk factors themselves, risk factors such as family structure and socioeconomic background may be less modifiable. It thus is important to continue to study the mechanisms by which early disadvantages impact early-onset delinquency, and perhaps to focus on mechanisms that could potentially be targeted by prevention or intervention programs.

Despite this, the results of the current analysis do substantiate a classic theoretical claim about the origins of delinquency: As theorized by both Moffitt (1993) and Gottfredson and Hirschi (1990), precursors to antisocial behavior emerge long before children enter grade school. Our analysis showed that factors established by age 3 years, and even by age 9 months, predicted distinct patterns or trajectories of misconduct throughout childhood, which in turn predicted both property crime and substance use by age 11. Though our data cannot yet explore whether these misconduct trajectories will lead to offending later in

adolescence and adulthood, past studies have shown that the individual problem behaviors that comprise these trajectories are risk factors for more serious criminal behavior. Previous scholars have argued that persistent antisocial tendencies for very young children often manifest as “whining, pushing, and shoving” (Hirschi and Gottfredson, 1993: 53) or “biting and hitting” (Moffitt, 1993: 679). Early in the life course, children who show signs of physical aggression (Maughan et al., 2000; Savolainen et al., 2010), opposition-defiance (Murray et al., 2010), difficult temperament (Caspi, 2000), or impulsivity and inattention (Farrington, 1989; Hawkins et al., 2000) are at greater risk for arrest and conviction during late adolescence and into adulthood. The roots of offending thus appear to extend back into the earliest period of development.

In addition, we also found that impulsive and inattentive children are more likely to engage in early-onset delinquency. Theoretically, childhood impulsivity is the key predictor of subsequent delinquency and substance use in Gottfredson and Hirschi’s (1990) self-control theory, and one of several indicators of cognitive deficits among Moffitt’s (1993) life course persistent offenders. In particular, the subset of children – approximately 6% in our sample – who showed a co-occurrence of impulsivity/inattention *and* early misconduct may possess both neuropsychological deficits and low self-control, which may work in concert to increase the risk of later antisocial behavior (Farrington and Welsh, 2007; Loeber and Keenan, 1994; Savolainen et al., 2010). More generally, our results are consistent with the idea that children who show problem behaviors across multiple domains (impulsivity, inattention, conduct problems, etc.) have a high risk for early initiation into delinquency and substance use (Loeber and Farrington, 2000).

The current study benefited from a number of relatively unique features of the nationally-representative ongoing MCS data which has followed children from infancy through age 11 years. For instance, a variety of conduct problems (e.g., fighting other children, having frequent temper tantrums, and being disobedient) were assessed on three occasions across early to middle childhood. This prospective design allowed us to both observe how these problem behavior items clustered together at each age, and to document within-person changes in antisocial tendencies over time. Whereas the vast majority of MCS children exhibited low probabilities of antisocial behavior, approximately one-quarter of the sample followed one of three higher-risk latent pathways: (1) temper problems, a pathway marked by oppositional behavior, fighting, and frequent temper tantrums; (2) attention problems, a pathway typified by impulsivity, inattention, and a lack of perseverance; and (3) mixed conduct and attention problems, a pathway characterized by a small number of children who exhibited the highest likelihood of all six antisocial indicators at each wave. As subsequent waves of data are collected in the MCS (age 14 in 2015, and age 17 in 2018), we will be able to chart longer-term sequences linking these antisocial tendencies to delinquency and substance use in adolescence and beyond.

There are, of course, risk factors that are likely to trigger antisocial tendencies and subsequent preteen delinquency that were not included in this study. For instance, research shows that low parental involvement (Bradley and Corwyn, 2007), and low child involvement in family activity (Farrington et al., 2002) have been linked to early onset delinquency. Other risk factors include parents who rely on erratic and inconsistent

discipline, harsh discipline, or—at the extreme end—physically abuse their children (Farrington, Ttofi, and Coid, 2009; Moffitt et al., 2011; Moffitt, 2005; Patterson, DeBaryshe, and Ramsey, 1989). Nervous and withdrawn children tend to avoid delinquency during childhood and early adolescence, but may show an increase in delinquency during the late teenage years (Zara and Farrington, 2009).

The present findings should be considered in light of several strengths and limitations of the data. Whereas much prior work using well-characterized local samples has addressed childhood antecedents of early adolescent delinquency (e.g., Broidy et al., 2003), the present high-quality prospective data from infancy throughout childhood in a large, contemporary nationally-representative sample across the four countries of the UK are unique in both criminological and developmental studies. The measures of early life developmental and family risk factors are strong, and antisocial tendencies are reported at three waves by parents; however, it should be noted that the items we used from the Strengths and Difficulties questionnaire are more commonly combined into scales. Additional items and broader coverage of neuropsychological deficits, parenting, and childhood conduct problems would add considerable value. It is also possible that collecting pregnancy alcohol use data via face-to-face interview may have led to some under-reporting. Finally, while retention is excellent for a study of this scope and duration, it is possible that differential attrition may affect generalizability.

Though we identified several important precursors of preteen delinquency in this prospective sample of British youth, it will be important to continue following these children as they enter the peak ages of offending. In the future, we will be able to address whether preteen delinquency leads to more frequent and/or more serious offending in adolescence. In follow-up surveys it is plausible that at least some of the preteen delinquents in this sample will become life course persistent offenders, but many of these youth will likely show discontinuity and follow non-criminal trajectories. As more waves of data are collected, we will also be able to assess how much of the contemporaneous effects of family, school, and peers on juvenile delinquency are explained by pathways of antisocial tendencies we identified here. Addressing these questions will be an important next step in our future research with the MCS cohort.

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Appendix A

Bayesian Information Criterion Statistic for Multilevel Latent Class Analysis of Childhood Antisocial Tendencies

Level 2 pathways/Level 1 latent classes	BIC statistic
Latent Pathway 1 (level 2)	
1 class (level 1)	454309
2 classes	257731
3 classes	240653
4 classes	237469
5 classes	236586
6 classes	236031
7 classes	235721
8 classes	235702
9 classes	235831
Latent Pathway 2 (level 2)	
1 class (level 1)	454320
2 classes	254673
3 classes	234818
4 classes	231726
5 classes	230687

Level 2 pathways/Level 1 latent classes	BIC statistic
6 classes	229904
7 classes	229558
8 classes	229509
9 classes	229532
Latent Pathway 3 (level 2)	
1 class (level 1)	454331
2 classes	254638
3 classes	232703
4 classes	229544
5 classes	228537
6 classes	227811
7 classes	227401
8 classes	227361
9 classes	227504
Latent Pathway 4 (level 2)	
1 class (level 1)	454342
2 classes	254681
3 classes	232584
4 classes	229023
5 classes	227943
6 classes	227333
7 classes	226693
8 classes	226815
Latent Pathway 5 (level 2)	
1 class (level 1)	454353
2 classes	254725
3 classes	232648
4 classes	228932
5 classes	227658
6 classes	226952
7 classes	226309
8 classes	226725
Latent Pathway 6 (level 2)	
1 class (level 1)	454364
2 classes	254768
3 classes	232724
4 classes	229018
5 classes	227497
6 classes	226487
7 classes	225997
8 classes	226474

Level 2 pathways/Level 1 latent classes	BIC statistic
Latent Pathway 7 (level 2)	
1 class (level 1)	454375
2 classes	254811
3 classes	232800
4 classes	229120
5 classes	227599
6 classes	226440
7 classes	225980
8 classes	226062
Latent Pathway 8 (level 2)	
1 class (level 1)	454385
2 classes	254855
3 classes	232876
4 classes	229234
5 classes	227736
6 classes	226579
7 classes	225991
8 classes	226118

NOTE: Lowest BIC in italics

ABBREVIATION: BIC = Bayesian information criterion

Figure 1.
Latent pathways of early childhood antisocial tendencies

Table 1

Weighted Descriptive Statistics (N = 13,221)

Variables	Weighted Mean or %	SE	Age of Child when Measured
Focal Outcome Variables			10–11 years
Property crime	9.11%		
Substance use	14.88%		
Childhood Antisocial Tendencies (AST) Latent Pathways			3–7 years
Normative	74.91%		
Mixed conduct and attention problems	5.57%		
Temper problems	10.18%		
Attention problems	9.34%		
Early Childhood Developmental Factors			
Easy infant temperament	-.006	.016	9 months
Cognitive ability (standardized)	-.008	.023	3 years
Heavy fetal alcohol exposure	2.17%		9 months
Low birth weight	7.21%		9 months
Early Childhood Family Factors			
Parents do not use illicit drugs	88.16%		3 years
Parents do not smoke	57.45%		3 years
Parents do not drink	13.82%		
Teenage mother (< 20 years at child birth)	9.96%		9 months
Family structure			3 years
Single parent	19.89%		
Married parents	60.95%		
Cohabiting parents	19.16%		
Mother-child closeness (standardized)	-.038	.014	3 years
Parent education (highest)	2.75	.034	3 years
Parent occupational status (highest)			3 years
Not working	26.02%		
Semi-routine, routine	14.69%		
Low supervisory, technical	6.67%		
Small employer, self-employed	8.00%		
Intermediate	9.68%		
Managerial, professional	34.95%		
Background Variables			
Boy	51.62%		9 months
Race/ethnicity			9 months
White British	84.46%		
Asian British	7.08%		
Black British	3.47%		
Other	4.99%		

ABBREVIATION: SE = standard error (omitted for dichotomous variables)

Source: UK Millennium Cohort Study

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Table 2

Odds Ratios and Confidence Intervals from Multinomial Regressions Predicting Childhood Antisocial Tendencies (AST) Pathways (versus Normative / Low Latent Pathway), N = 13,221

Predictors	Mixed Conduct and Attention Problems		Temper Problems		Attention Problems	
	OR	[95% CI]	OR	[95% CI]	OR	[95% CI]
Early Childhood Developmental Factors						
Easy infant temperament (standardized)	.78***	[.70,.87]	.80***	[.74,.88]	.86**	[.78,.95]
Cognitive ability (standardized)	.52***	[.46,.59]	.83***	[.76,.91]	.62***	[.57,.68]
Heavy fetal alcohol exposure	.95	[.52,1.72]	1.10	[.70,1.74]	1.10	[.68,1.78]
Low birth weight	1.36	[.97,1.89]	1.33*	[1.07,1.65]	1.82***	[1.45,227]
Early Childhood Family Factors						
Parents do not use illicit drugs	.91	[.70,1.20]	.93	[.74,1.16]	.86	[.69,1.07]
Parents do not smoke	.75*	[.60,.95]	.81**	[.70,.93]	.95	[.82,1.10]
Parents do not drink	1.20	[.86,1.67]	.79	[.61,1.02]	.77	[.54,1.09]
Teenage mother	.76	[.55,1.06]	.93	[.72,1.20]	1.29	[.97,1.70]
Family structure (vs single parent)						
Married parents	.43***	[.31,.59]	.74*	[.56,.97]	.79*	[.63,.98]
Cohabiting parents	.69*	[.51,.93]	.86	[.65,1.13]	.76*	[.58,.98]
Mother-child closeness (standardized)	.62***	[.57,.68]	.80***	[.74,.87]	.76***	[.70,.82]
Parent education (highest)	.88**	[.80,.96]	.88***	[.83,.94]	.94	[.88,1.01]
Parent occupational status (highest) (vs not working)						
Semi-routine, routine	.74	[.52,1.04]	.91	[.73,1.13]	1.08	[.84,1.38]
Low supervisory, technical	.96	[.66,1.40]	.88	[.66,1.18]	1.04	[.76,1.42]
Small employer, self-employed	.65	[.39,1.08]	.78	[.56,1.08]	.72	[.53,.99]
Intermediate	.75	[.49,1.14]	.65**	[.49,.87]	1.11	[.82,1.49]
Managerial, professional	.62*	[.41,.94]	.57***	[.45,.72]	.97	[.76,1.23]
Background Variables						
Boy	2.34***	[1.84,299]	1.10	[.94,1.27]	1.82***	[1.60,2.07]
Race/ethnicity (vs White British)						

Predictors	Mixed Conduct and Attention Problems		Temper Problems		Attention Problems	
	OR	[95% CI]	OR	[95% CI]	OR	[95% CI]
Asian British	.35***	[.22,.56]	1.16	[.76,1.76]	.90	[.61,1.32]
Black British	.26***	[.12,.57]	.76	[.47,1.23]	.52*	[.31,.86]
Other British	.77	[.46,1.28]	1.08	[.75,1.56]	.88	[.58,1.32]

NOTE. Model included fixed effects for stratum (omitted from table).

ABBREVIATION: OR = odds ratio; CI = Confidence Intervals

Source: UK Millennium Cohort Study

p < .001;
**
p < .01;
*
p < .05

Table 3

Odds Ratios and Confidence Intervals from Logistic Regressions Predicting Preteen Property Crime and Substance Use from Early Childhood Factors and Antisocial Tendencies (AST) Pathways (N = 13,221)

Predictors	Property Crime			Substance Use		
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
	OR	[95% CI]	OR	[95% CI]	OR	[95% CI]
AST Pathways (vs Normative)						
Mixed conduct and attention problems		2.02***	[1.46,2.79]		1.57***	[1.24,2.01]
Temper problems		1.65***	[1.32,2.06]		1.25*	[1.03,1.52]
Attention problems		1.46**	[1.14,1.88]		1.33**	[1.09,1.63]
Early Childhood Developmental Factors						
Easy infant temperament (standardized)	.96	[.89,1.04]	.98	[.91,1.06]	.96	[.91,1.02]
Cognitive ability (standardized)	.88**	[.81,.97]	.92	[.84,1.01]	.97	[.90,1.04]
Heavy fetal alcohol exposure	1.25	[.86,1.82]	1.26	[.87,1.83]	1.33	[.95,1.85]
Low birth weight	1.17	[.91,1.50]	1.13	[.88,1.46]	1.04	[.83,1.30]
Early Childhood Family Factors						
Parents do not use illicit drugs	.69***	[.57,.85]	.70***	[.57,.85]	.63***	[.54,.74]
Parents do not smoke	.67***	[.58,.78]	.68***	[.59,.80]	.78***	[.69,.89]
Parents do not drink	.93	[.72,1.20]	.94	[.73,1.21]	.63***	[.49,.82]
Teenage mother	.81	[.63,1.03]	.81	[.64,1.03]	.95	[.77,1.19]
Family structure (vs single parent)						
Married parents	.68**	[.53,.87]	.71**	[.55,.91]	.67***	[.55,.82]
Cohabiting parents	.82	[.65,1.04]	.85	[.67,1.07]	.78*	[.63,.96]
Mother-child closeness (standardized)	.95	[.88,1.02]	.98	[.91,1.06]	.94*	[.89,.99]
Parent education (highest)	.93*	[.87,.99]	.94	[.88,1.00]	.95	[.91,1.00]
Parent occupational status (highest) (vs not working)						
Semi-routine, routine	.77*	[.63,.95]	.79*	[.64,.97]	1.13	[.94,1.37]
Low supervisory, technical	.77	[.56,1.07]	.78	[.57,1.08]	1.02	[.79,1.33]
					1.15	[.95,1.39]
					1.04	[.79,1.35]

Predictors	Property Crime				Substance Use			
	Model 1		Model 2		Model 1		Model 2	
	OR	[95% CI]	OR	[95% CI]	OR	[95% CI]	OR	[95% CI]
Small employer, self-employed	.68*	[.49,.94]	.70*	[.51,.98]	1.12	[.87,1.43]	1.15	[.90,1.47]
Intermediate	.80	[.61,1.05]	.82	[.62,1.09]	1.09	[.87,1.37]	1.12	[.89,1.40]
Managerial, professional	.70**	[.55,.90]	.73*	[.57,.93]	.92	[.77,1.11]	.94	[.78,1.13]
Background Variables								
Boy	1.68***	[1.46,1.94]	1.62***	[1.41,1.86]	1.66*	[1.49,1.86]	1.62***	[1.46,1.81]
Race/ethnicity (vs White British)								
Asian British	.76	[.51,1.12]	.78	[.53,1.17]	.27***	[.18,.41]	.28***	[.18,.42]
Black British	1.01	[.71,1.42]	1.08	[.77,1.53]	.65*	[.45,.95]	.68*	[.47,.99]
Other British	.70*	[.50,.99]	.70*	[.50,1.00]	.56***	[.40,.79]	.57***	[.40,.79]

NOTE. Models include fixed effects for stratum (omitted from table);

ABBREVIATIONS: AST=Antisocial Tendencies; OR = odds ratio; CI = Confidence Intervals;

p < .001;
**
p < .01;
*
p < .05;

Source: UK Millennium Cohort Study