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Levels of risk: maternal-, middle childhood-, and neighborhood-level predictors of adolescent disinhibitory behaviors from a longitudinal birth cohort in the United States

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Abstract

Objective—Disruptive behavior in adolescence may indicate a broad vulnerability to disinhibition, which begins in childhood and culminates in adult externalizing psychopathology. We utilized prospective birth cohort data to assess childhood predictors of adolescent disinhibition. We also examined the effect of pre-adolescent fluctuation in cognitive ability.

Methods—Data were drawn from the Child Health and Development Study cohort, born 1961–1963; we used the subsample who participated in follow-up through adolescence ($n=1752$). Six indicators of behavioral disinhibition (BD), reported in adolescence, were analyzed as a count outcome. Predictor variables were drawn from several waves of data collection and included individual-, maternal-, and neighborhood-level measures. Cognitive ability was assessed with the Peabody Picture Vocabulary Test at two time points. Neighborhood characteristics were assessed using census data from 1970.

Results—Number of BD indicators was predicted by maternal characteristics at prenatal assessment (maternal age and alcohol consumption) and age-10 assessment (maternal smoking, education, and separation from father). Characteristics of the child that predicted BD included birth order and conduct problems in middle childhood. Neighborhood poverty did not predict BD. Regardless of initial cognitive ability score, movement to a higher quartile by adolescence was associated with lower BD, while movement to a lower quartile was associated with higher BD.

Conclusion—Risk for adolescent BD exists prenatally and extends through middle childhood. Change in cognitive ability during pre-adolescence emerged as a potentially important factor that merits further investigation. A greater focus on the life course can aid in comprehensively understanding disruptive behavior emergence in adolescence.

Keywords

Risk Levels; Maternal/Pregnancy; Child Health; Disinhibitory behavior; Adolescence

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Introduction

The disruptive behaviors that mark adolescent conduct problems rarely occur in isolation (Jessor & Jessor, 1977). That is, adolescents who use alcohol also tend to use tobacco, underachieve at school, engage in precocious sexual behavior, and get into trouble with the law (Lacono et al., 1999; Lacono et al., 2008; Young et al., 2000). The communalities characteristically found among these multiple indicators have led researchers to posit the existence of a broad vulnerability to engage in disinhibited behavior, distinguished by undersocialized conduct and low levels of dispositional constraint (Krueger et al., 2002; Young et al., 2000). Findings from longitudinal birth cohort studies have shown that this general vulnerability is related to an array of highly comorbid adult outcomes that share a common association with under-constrained behavior, specifically substance disorders and antisocial conduct (Keyes et al., 2007; Lacono et al., 2008; McGue et al., 2001; McGue & Lacono, 2005). These adverse adult outcomes are hypothesized to occur more frequently in those with a childhood onset of impulsive, hyperactive, or aggressive behavior than in those who make time-limited adolescent forays into deviant activity (Moffitt, 1993).

Thus, the identification of pathways to disinhibited behavior in adolescence has important implications for both etiology and public health. For etiology, a consideration of the full life course of exposure experience can illuminate mediators of adult psychopathology. For public health, the identification of children at increased risk of adolescent behavioral disinhibition can foster prevention and intervention efforts. Prenatal events (Buschgens et al., 2009; Chilcoat & Breslau, 2002; Fergusson et al., 1998; Nigg & Breslau, 2007), socio-demographic factors (Fergusson et al., 2005a; Fergusson et al., 2005b; Fischer, 1984; Lynam & Moffitt, 1993), family instability (Burt et al., 2008; Fergusson et al. 1994), neighborhood context (Ingoldsby & Shaw, 2002; Leventhal & Brooks-Gunn, 2000), cognitive ability (Fergusson et al., 2005a; Hinshaw, 1992; Lynam & Moffitt, 1993), and early conduct problems (Block et al., 1988; Fergusson et al., 2005b; Lynskey & Fergusson, 1995) have all been shown to predict adolescent disinhibited behavior; however, the evaluation of this literature is complicated by the frequent co-occurrence of these indicators. For example, Wakschlag & Hans (2002) reported that the association between maternal age and offspring risk for conduct disorder was reduced when other maternal risk factors were considered. Similarly, Fergusson et al. (2005a), demonstrated that while cognitive ability was inversely associated with the later adverse outcomes, this association was largely confounded by childhood conduct problems and family social circumstances. In this study, however, cognitive ability was measured at a single time point; it remains unclear whether the study of dynamic changes in cognitive ability (Moffitt et al., 1993) may illuminate associations that are obscured by single time measurement.

The present study uses data from a population-based longitudinal study of children born 1961–63 in the East Bay Area of California (van den Berg et al., 1988) to accomplish two primary aims. The first aim is to examine, both individually and in combination, the prognostic significance of prospectively-measured maternal, child, and neighborhood characteristics hypothesized to predict disinhibited behavior in adolescence. These measures can be classified into the following domains: the pre- and perinatal environment (prenatal exposure to maternal smoking and alcohol consumption, birth weight, and birth order); the childhood family environment (family income, maternal education and cognitive ability, maternal smoking, and parental separation); the neighborhood environment, measured by the percentage of individuals in the neighborhood living below the poverty line; as well as childhood conduct problems and cognitive ability. The second aim of the present study is to integrate a more dynamic life course perspective, by examining the effect of pre-adolescent fluctuation in cognitive ability on adolescent behavioral disinhibition. Using the Peabody

Picture Vocabulary test, we examine whether change in cognitive functioning from middle childhood to adolescence is predictive of disinhibited behavior in adolescence.

Methods

Study population and design

Data are drawn from four waves of the Child Health and Development Study (CHDS – van den Berg et al., 1988), one of the largest pregnancy cohorts ever assembled in the United States. The CHDS included pregnant women participating in the Kaiser Permanente Health Plan and residing in the East Bay Area of California. Almost 100% of women receiving prenatal care from late 1959 to the fall of 1966 participated in the study ($n=20,754$).

Membership in the Kaiser Permanente Health Plan typically required employment; thus, this sample has a slightly higher baseline socioeconomic status than a randomly selected cross-section of the population. Nevertheless, a broad range of backgrounds is represented in the Kaiser Health Plan, creating substantial socioeconomic variation within the sample that is similar to the general population, save the extremes (Krieger, 1992). Further, the ethnic diversity of the sample closely approximates that of the East Bay Area at the time of first data collection in 1959–66: 65% White, 23% Black, 4% Asian, and 3% Latino, with the remainder reporting multiple races or unclassified.

While data collection has remained ongoing for over forty years, the present study focuses on those children who participated in follow-up studies into adolescence; we draw on data in the pregnancy, age 9–11 years, and age 15–18 year interviews (CHDS-A, $n=1,752$). Inclusion in the 15–18 year adolescent sample was contingent on having participated in the first two waves. Compared to the original CHDS cohort, the CHDS-A included a greater proportion of subjects whose mothers were married and living with a husband at the original intake, who were white, and who were high school graduates; it also includes a smaller proportion of first-born offspring. Detailed information on the derivation of this sample can be found elsewhere (van den Berg, 1984). Table 1 describes the demographic, behavioral, and neighborhood characteristics of children and mothers in the CHDS-A (details of these measures below).

Measures

Outcome: Behavioral Disinhibition (BD)—Behavioral disinhibition (BD) was assessed by six child-report items gathered at the age 15–18 interview. Items were chosen to reflect substance involvement, academic underachievement, sexually permissive peers, and disregard for the law. These items, shown in Table 2, loaded on one factor with good overall fit statistics (eigenvalue=2.87, CFI=0.99, TLI=0.98, RMSEA=0.03, SRMR=0.04). Chronbach's alpha for the six-item scale was 0.62. These items were all defined dichotomously and included: regularly (once per month or more) gets high or tight from drinks (36.5%); most or all of friends have had sex (28.2%); considers self a fair or poor student (26.7%); ever been a regular (at least one per day for a significant period of time) smoker (25.1%); frequently absent from school for reasons other than illness, work, or family (17.7%); and feels obeying the law is not important (11.5%).

Predictors: prospectively-assessed characteristics of children, mothers, and neighborhoods

Pregnancy interviews—Characteristics of the child at birth (including sex and birth weight) were collected via maternal reports, medical records, and other research assessments. Birth weight was dichotomized (≤ 2500 grams versus >2500 grams) based on previous literature typically defining low birth weight as ≤ 2500 grams (Chilcoat & Breslau,

2002; Breslau & Chilcoat, 2000; Buschgens et al., 2009; Nigg & Breslau, 2007) and preliminary data analysis indicating that mean behavioral disinhibition did not vary among respondents with birth weights >2500 grams. For the purpose of this analysis, the child's race was recorded as the mother's self-reported race at the pregnancy interview. Maternal age at the time of birth was also considered. Finally, mothers also reported on specific behaviors at the time of pregnancy including cigarette smoking and alcohol consumption.

Age nine to eleven interviews—Measures at age nine to eleven included the Peabody Picture Vocabulary Test (PPVT), which was administered to both the mother and the child. Mothers reported on current cigarette smoking, highest level of education achieved, parental separation, and family income. Birth order of the respondent and family size were also measured at the nine to eleven interview, and were highly correlated ($r=0.81$). Because family size was not significantly associated with the outcome at the bivariable level ($p\text{-value}=0.14$), only birth order was retained in the present analysis.

Neighborhood of residence was determined by geocoding participants' home addresses at the age 9–11 interview using ArcGIS 9.3 (ESRI, Redlands, CA), and locating each within the appropriate 1970 Census tract using the National Historic Geographic Information System (NHGIS). Tract-level data from the 1970 Census were then linked to each child. Percent of families living below the poverty line for an average family of four in 1970 in the neighborhood of each respondent was assessed and analyzed as a potential predictor of BD in adolescence. Percent living below the 1970 median income in both California and the United States was also considered. Results were not dependent on the neighborhood income measure chosen.

Conduct problems at the age 9–11 interview were culled from a 100-item battery of child characteristics, administered to the mother. Details on the origin and development of these questions can be found elsewhere (Tuddenham et al., 1974). Items assessing childhood conduct problems included: stays away from home without permission, takes things that do not belong to him/her, flares up over nothing, has temper explosions, bullies others, and starts fights as well as truthful, and dependable, both reverse coded. Items exhibited unidimensionality in an exploratory analytic framework (eigenvalue=4.78, CFI=0.95, TLI=0.94, RMSEA=0.07, SRMR=0.05), and item severity and discrimination were subsequently estimated using a two-parameter Item Response Theory (IRT) analysis. Severity score on the latent factor were used in these analyses. 'Bullying others' evidenced the highest severity among the eight items analyzed [6.74]; 'taking things that do not belong to him/her' evidence the lowest severity [2.18]. Item discrimination ranged from 3.55 ('bullying others') to 1.59 ('stays away from home without permission').

Age fifteen to seventeen interviews—The PPVT was administered to the child a second time at the age 15–18 interview. We also included child age at the adolescent interview, which ranged from 15 ($n=139$) to 18 ($n=9$), with a majority of adolescents aged 17 ($n=1149$). We combined 17 and 18 year old participants due to the small number of 18 year olds.

Statistical analysis

For categorical and ordinal predictors, the mean number of BD indicators at each level of the predictor was calculated. For continuous predictors, the correlation between each predictor and the total number of BD indicators was calculated. Formal bivariable and multivariable analyses were performed using Poisson regression models due to the right-skewness of the outcome variable (Mean=1.42 [SD=1.5]). Regression diagnostics indicated that Poisson models were appropriate (e.g., models did not demonstrate over dispersion, and

residuals appeared normally distributed for all models presented). Multivariable analysis was conducted with all covariates of interest entered simultaneously using SAS software version 9.1.

The trajectory of childhood cognitive ability was determined by dividing children into quartiles (<25th percentile, 25th to 49th percentile, 50th to 74th percentile, and ≥75th percentile) based on their PPVT scores at the age 9–11 and age 15–18 interviews. Percentiles were calculated within each age. Those who remained in the same quartile at both time points were categorized as “Stable”. Those who moved to a higher or lower quartile were categorized as “Moved up” and “Moved down,” respectively. These groups were then used to predict BD, both in bivariable and multivariable analysis.

Four variables evidenced substantial missing data: maternal alcohol consumption [missing $n=470$], maternal cognitive ability [missing $n=351$], child conduct disorder symptoms [missing $n=250$], and family income [missing $n=173$]. In order to maximize the information available, we conducted multiple imputation using full information maximum likelihood methods through the EM algorithm (Dempster et al., 1977; Little & Rubin, 2002). Information was provided by the full set of variables included in the analysis.

Results

Predictors of adolescent behavioral disinhibition

Table 3 provides the mean number of BD indicators at each level of the predictor for all categorical and ordinal predictors as well as the correlation with the total number of BD indicators for all continuous predictors. For all variables, crude and adjusted Poisson regression beta estimates are provided.

Based on the adjusted regression model, there were no differences between White and Black children on BD; however, those who reported “other” race (Asian [$n=49$], Hispanic/Latino [$n=87$], and unreported [$n=1$]) had significantly fewer mean BD indicators compared to Whites ($\beta=-0.27$, $SE=0.1$, $p=0.004$). Gender was not associated with BD ($p=0.93$). Younger respondents had lower BD than older respondents. Adolescents with low birth weight had significantly fewer mean BD indicators ($\beta=-0.25$, $SE=0.11$, $p=0.03$). Other characteristics of the child predictive of BD included birth order and conduct problems assessed at age 9–11 ($\beta=0.16$, $SE=0.03$, $p<0.001$). Cognitive ability assessed at the age 9–11 interview was not significantly related to BD ($p=0.24$).

Characteristics of the mother predictive of BD included maternal alcohol consumption in pregnancy ($\beta=0.26$, $SE=0.06$, $p<0.001$) and smoking reported at the age 9–11 interview ($\beta=0.17$, $SE=0.06$, $p=0.003$). Maternal smoking during pregnancy was predictive of BD in a bivariable regression model ($\beta=0.30$, $SE=0.04$, $p<0.001$), but this association was not significant after controlling for mother’s smoking at the age 9–11 interview. The interaction between mother smoking during pregnancy and mother smoking at age 9–11 was not statistically significant ($\beta=0.06$, $SE=0.12$, $p=0.59$). Several additional maternal factors significantly predicted adolescent BD in the adjusted analysis, including maternal age at child’s birth ($\beta=-0.03$, $SE=0.00$, $p<0.001$), as well as highest level of education ($\beta=0.23$, $SE=0.08$, $p=0.002$) and separation from the child’s father ($\beta=0.23$, $SE=0.05$, $p<0.001$) by the age 9–11 interview. Neither family income ($p=0.42$) nor maternal cognitive ability ($p=0.47$) predicted adolescent BD in the adjusted analysis.

Per cent living below the U.S. poverty line for a family of four in 1970 was not significantly predictive of BD (Table 3). Per cent living below the median California income ($\beta=-0.0011$, $SE=0.0$, $p=0.33$) and per cent living below the median U.S. income ($\beta=-0.0014$, $SE=0.0$,

$p=0.22$) were also not statistically significant in bivariable or adjusted models. Examination of these characteristics as continuous and dichotomous variables also did not reveal any significant bivariable or multivariable associations (results not shown).

Cognitive change from age 9–11 to adolescence

Table 4 presents mean number of BD indicators according to the trajectory of cognitive ability across two time points. Relative to adolescents who remained in the same ability quartile from age 9–11 to age 15–18 (regardless of initial score), adolescents whose cognitive ability increased had significantly fewer mean BD indicators ($\beta = -0.20$, $SE=0.1$, $p<0.001$), while adolescents whose cognitive ability decreased had significantly more mean BD indicators ($\beta=0.19$, $SE=0.0$, $p<0.001$).

Individuals who remained in the highest quartile at both time points had the lowest mean number of BD indicators in adolescence (mean=1.01, $SD=1.3$, $N=191$); the highest mean number of BD indicators were found for the group who moved from the 50–75th percentile at age 9–11 to below the 50th percentile at age 15–18 (mean=1.83, $SD=1.5$, $N=166$). The mean score for these individuals was not significantly different than those who were below the 25th percentile at both time points (mean=1.78, $SD=1.5$, $N=294$). Full detailed results and pair-wise comparisons are available upon request.

Discussion

The present study demonstrates that behavioral disinhibition (BD) in adolescence represents an array of disruptive behaviors that are psychometrically and conceptually cohesive. This is consistent with previous research indicating that adolescent disruptive behaviors are indicative of a broad vulnerability to more general disinhibition that begins in childhood and culminates in adult externalizing psychopathology (Keyes et al., 2007; Lacono et al., 1999; McGue & Lacono, 2005). We have demonstrated that BD can be predicted by factors as early as the prenatal environment; maternal alcohol consumption at pregnancy significantly predicts BD more than fifteen years later, controlling for a broad array of potential confounding factors including socioeconomic status of the family, maternal cognitive ability, and neighborhood context. Factors associated with the rearing environment at age 9–11 were also associated with BD, including maternal smoking, maternal education, and parental separation. Further, we show that conduct problems, prospectively assessed at age 9–11, are independent predictors of BD in adolescence. Finally, while childhood cognitive ability measured at age 9–11 is not a significant predictor of BD, we document that change in cognitive ability from middle childhood to adolescence is strongly related to BD. Specifically, moving from a higher quartile on cognitive score to a lower quartile predicts increased BD symptoms relative to individuals whose cognitive ability remains stable, while moving from a low to a higher score is associated with lower BD symptoms.

Taken together, these findings indicate that risk for adolescent behavioral disinhibition exists at birth and extends through middle childhood. While our results are broadly consistent with previous literature documenting predictive validity for such factors as family socioeconomic environment (Fergusson et al., 2005a; Lynam et al., 1993), family instability (Fergusson et al., 1994), and conduct problems (Block et al., 1988; Fergusson et al., 2005b; Lynskey & Fergusson, 1995), we extend this work in two important domains. First, we include a broad array of prospectively assessed potential risk factors including maternal alcohol consumption during pregnancy and maternal smoking at two time points. Second, we consider these comprehensive factors conjointly in controlled regression. This allowed us to determine, for example, that when controlling for maternal smoking closer to the onset of adolescence, maternal smoking in pregnancy is not a risk factor for BD. These findings

suggest that a greater focus on life course processes can aid in comprehensively understanding the processes through which disruptive behavior emerges in adolescence.

Two findings from these analyses are inconsistent with previous literature. First, we did not find that gender is predictive of adolescent behavioral disinhibition. Epidemiologic studies traditionally document that adolescent boys are more likely than adolescent girls to engage in substance use and other externalizing behaviors (Hinshaw, 1992). Analysis of item-specific gender differences indicated that boys were slightly although statistically significantly more likely than girls to binge drink ($\chi^2=9.4$, $df=1$, $p=0.002$), consider themselves fair or poor students ($\chi^2=4.5$, $df=1$, $p=0.03$), and report that obeying the law is not important ($\chi^2=9.7$, $df=1$, $p=0.002$). Girls were substantially more likely than boys to smoke ($\chi^2=30.8$, $df=1$, $p<0.001$) and slightly more likely to report that most or all of friends have had sex ($\chi^2=5.7$, $df=1$, $p=0.02$). There was no gender difference in school absence ($\chi^2=0.32$, $df=1$, $p=0.57$). Thus, the lack of a gender difference is, to some extent, due to compensatory gender differences among the disinhibitory behaviors. More importantly, research is accumulating to suggest evidence of a cohort effect on gender differences in substance use and other externalizing behaviors (Keyes et al., 2008; Johnson & Gerstein, 1998). That is, among cohorts born approximately after 1950, a converging gender gap in the incidence and prevalence of substance use and other problem behaviors has emerged. The lack of strong gender differences at the item level may be indicative of this cohort effect. Second, while a previous study from a prospective community sample in Michigan found that low birth weight increases risk of BD in adolescence (Breslau & Chilcoat, 2000; Chilcoat & Breslau, 2002; Nigg & Breslau, 2007) we find fewer mean BD symptoms among adolescents with low birth weight. Assessment of BD in the Michigan sample was at age 11 years; the differences in these results may be attributable to differing ages of BD assessment. We examined differences in conduct disorder symptoms assessed at the 9–11 interview, but did not find associations with low birth weight ($t=1.02$, $p=0.31$). Further analysis of the role of low birth weight on adolescent outcomes is necessary to elucidate these relationships.

The role of cognitive ability in the development of disruptive behavior has been a source of debate in the literature (Fergusson et al., 2005a; Lynam et al., 1993; Schonfeld et al., 1988), with research primarily attempting to tease apart the directionality of effect. Our results are consistent with those of Fergusson et al. (2005a) from a New Zealand cohort study, indicating that the association between cognitive ability measured in middle childhood and adolescent disruptive behavior is explained by other factors, for example, conduct problems in middle childhood. However, the results from the present study suggest another methodological consideration: cognitive ability is not necessarily a fixed characteristic (Ceci, 1996). Regardless of the initial score, individuals who evidenced a decrease in cognitive ability from middle childhood to adolescence had significantly more BD indicators relative to individuals with stable cognitive ability scores. Similarly, individuals who increased in cognitive ability had significantly fewer BD indicators in adolescence. Rather than cognitive ability measured at one point in time as a risk factor for behavioral disinhibition, we suggest that change in cognitive ability prior to adolescence is a potentially important etiologic factor. Previous (although not all – see Moffit et al., 1993) research has indicated that change in cognitive ability can be substantial during mid-childhood (Breslau et al., 2001; Ceci, 1996), and that socio-economic disadvantage may contribute to a decline (Breslau et al., 2001). Our findings contribute to this growing research, suggesting that changes in cognitive ability are not rare and that decreases in cognitive ability are meaningful predictors of behavioral disinhibition. Of course, temporality issues may still impact the validity of this finding; while early conduct problems were included as a control variable, it is possible that early problem behavior unmeasured in this study preceded a change in cognitive ability. As heritability estimates of cognitive ability increase with age (McGue et al., 1993; Plomin, 1999), shared genetic factors between changes in cognitive

ability and changes in behavioral disinhibition may also contribute to the observed association. Future research with genetically informative data is important to further elucidate this association, and a greater focus on the dynamics of cognitive ability may prove a fruitful area of study.

Poverty of the neighborhood at age 9 to 11 did not significantly predict BD in adolescence. The research on neighborhood income effects in alcohol and drug epidemiology generally have been mixed: some studies find an effect of living in a socially disadvantaged neighborhood (Browning et al., 2004; Ennett et al., 1997; Lambert et al., 2004; Leventhal & Brooks-Gunn, 2000), while other studies do not (Allison et al., 1999; Brook et al., 1989; Clarke et al., 2008; Dupere et al., 2008). Thus, our results are not particularly inconsistent with the existing literature. Differences in measurement, including the limitations of using census tracts as proxies for neighborhoods (Diez Roux, 2007), could introduce noise into effect estimates across these studies, including the present study. While census tracts may not perfectly correlate with more qualitatively designated neighborhood boundaries, census tracts have been shown to provide reasonable approximations of neighborhoods (Sampson, 1997). Further investigation of the role of neighborhood on behavioral disinhibition in samples with greater racial and income diversity should remain an important future direction.

While a prospective design, large sample, and comprehensive measurement are significant strengths of the present study, limitations should be noted. This study began in the early 1960's; thus, the respondents in this study were adolescents in the mid- to late-1970s. Nationally representative surveys of adolescents have documented substantial temporal trends in substance use as well as other disinhibitory behaviors across time (Johnston et al., 2007). Further, important predictor variables in this study such as maternal smoking and alcohol consumption have changed in population prevalence across time as well (Substance Abuse and mental health Services Administration, 2003). However, the patterns of interrelationships between the predictors and outcomes included in this study may not have changed substantially. Replication of these results using data collected more recently would be beneficial to examine the implications of this study for current and future adolescents. Secondly, the majority of data included in this report rely on self-report of the children and the mothers. For example, we relied on mother's report of childhood conduct symptoms at the age 9 to 11 interview, and child's report of behavioral disinhibition in adolescence. Measurement error in these reports is an inevitable consequence of conducting behavioral research of this nature. Thirdly, there were nontrivial amounts of missing data on several key variables used in these analyses. We assumed that data was missing at random and used full-information maximum likelihood methods to conduct multiple imputation (MI). As a sensitivity analysis, we estimated all models using list-wise deletion (LD) for respondents with missing data (results available as an online addendum to this report). Three variables significant in Table 3 were non-significant using list-wise deletion, but the direction and magnitude of the effects were similar across the two approaches; age 16 vs. 17–18 (LD $\beta = -0.11$, MI $\beta = -0.13$), maternal smoking at the 9–11 interview (LD $\beta = 0.10$, MI $\beta = 0.17$) and birth weight (LD $\beta = -0.22$, MI $\beta = -0.25$). One variable (Black race) was significant using list-wise deletion but insignificant using multiple imputation (LD $\beta = -0.29$, MI $\beta = -0.27$). All other results did not materially change depending on missing data approach used. Because limited information was available on the fathers and siblings of CHDS children, we focused on characteristics of the mothers and children only. Father and sibling behavior may be additionally important determinants of adolescent behavioral disinhibition (East & Felice, 1992; Fagan & Najman, 2003; Malone et al., 2002) and future studies with more comprehensive measures of first-degree family members would be helpful to more fully elucidate risk factors.

In summary, the present study begins to unravel the mechanisms through which behavioral disinhibition arises in adolescence. These pathways are important to disentangle, as identification of and early intervention with children vulnerable to disinhibition may aid in the prevention of adult substance disorders and antisocial conduct. Risk for adolescent disinhibited behavior begins during the prenatal period and extends into middle childhood. Downward fluctuation in cognitive ability from middle-childhood to adolescence may be a risk factor for adolescent behavioral disinhibition; future research should examine the characteristics of children who exhibit downward change in order to be fully understand, this potentially vulnerable group. Future research should formally examine the association between these early indicators and adult externalizing psychopathology incorporating behavioral disinhibition as a potential mediator, providing a more comprehensive delineation of psychopathology across the life course.

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Table 1

Characteristics of mothers and children measured across four waves of data collection in the adolescent Child Health and Development Study (CHDS-A)

Race		N	%
	White	1275	72.3
	Black	340	19.4
	Other	137	7.8
Gender			
	Male	885	50.5
	Female	867	49.5
Age at adolescent interview			
	15	139	7.9
	16	456	26.0
	17–18*	1157	66.0
Maternal smoking during pregnancy			
	Current	586	33.6
	Never/Former	1157	66.4
Maternal weekly alcohol consumption at time of pregnancy			
	>2 drinks	418	25.5
	1–2 drinks	747	42.6
	0 drinks	559	31.9
Maternal smoking at age 9–11 interview			
	Current	543	31.5
	Never/Former	1180	68.5
Mother's highest education by age 9–11 interview			
	Less than high school	168	9.8
	High school	659	38.3
	More than high school	896	52.0
Mother reported separation from father by age 9–11 interview			
	Yes	413	23.8
	No	1325	76.2
Percent below the poverty line** in the ages 9–11 neighborhood			
	1st Quartile (>3.0%)	421	24.0
	2nd Quartile	420	24.0
	3rd Quartile	437	24.9
	4th Quartile (<1.0%)	474	27.1
Birth order			
	1 st	429	24.8
	2 nd	470	27.1

Race		N	%
	3 rd	370	21.4
	4 th	234	13.5
	≥5 th	229	13.2
Birth weight			
	≤2500 grams	83	4.7
	>2500 grams	1669	95.3
		Mean (SD)	Range
Maternal age at child's birth		28.73 (5.9)	(15 to 47)
Child cognitive ability *** at age 9–11 interview		82.52 (11.5)	(53 to 138)
Child conduct problems at age 9–11 interview (score on latent trait) +		0.00 (0.68)	(−0.535 to 2.74)
Family income at age 9–11 interview		13,348 (7203.9)	(0 to 98,000)
Maternal cognitive ability ** at age 9–11 interview		127.00 (17.1)	(60 to 161)

* Age 17, N=1148, Age 18 N=9

** The percentage of the neighborhood living below the poverty line for a family of four in 1970

*** As measured with the Peabody Picture Vocabulary Test (PPVT)

+ As determined using 2-parameter Item Response Theory of eight conduct disorder symptoms, extracting factor severity scores on underlying latent trait.

Table 2

Description of behavioral disinhibition items measured in adolescence used to create outcome variable*

		N	%
Regularly (once a month or more) gets high or tight from drinks			
	Yes	619	36.5
	No	1075	63.5
Most or all friends have had sex			
	Yes	441	28.2
	No	1124	71.8
Consider yourself a fair or poor student			
	Yes	443	26.7
	No	1218	73.3
Ever been a regular smoker (at least cigarette one per day for a significant period of time)			
	Yes	434	25.1
	No	1296	74.9
Frequently absent from school for reasons other than illness, work, or family			
	Yes	305	17.7
	No	1414	82.3
Obeys the law is not important			
	Yes	195	11.5
	No	1508	88.5
Count of disinhibitory behaviors			
	0	613	36.2
	1	410	24.2
	2	278	16.4
	3	217	12.8
	4	115	6.8
	5	48	2.8
	6	14	0.8

* Factor analysis indicated that the six items loaded on one factor with good overall fit statistics (eigenvalue=2.87, CFI=0.99, TLI=0.98, RMSEA=0.03, SRMR=0.04). Chronbach's alpha for the six-item scale was 0.62.

Table 3

Predictors of adolescent disinhibitory behaviors: results of a longitudinal birth cohort in the U.S. (N=1,752)

		Mean behavioral disinhibition (SD)	Unadjusted Beta [SE]	p- value	Adjusted ⁺ Beta [SE]	p-value
Race						
	White	1.45 (1.5)	REF		REF	
	Black	1.48 (1.4)	0.02 [0.05]	0.71	-0.09 [0.07]	0.24
	Other	0.97 (1.2)	-0.40 [0.09]	<0.001	-0.27 [0.10]	0.004
Gender						
	Male	1.41 (1.4)	-0.00 [0.04]	0.91	0.004 [0.04]	0.93
	Female	1.42 (1.5)	REF			
Age at adolescent interview						
	15	0.82 (1.2)	-0.62 [0.10]	<0.001	-0.64 [0.10]	<0.001
	16	1.35 (1.4)	-0.12 [0.05]	0.01	-0.13 [0.05]	0.01
	17-18	1.52 (1.5)	REF		REF	
Maternal smoking during pregnancy						
	Current	1.71 (1.6)	0.30 [0.04]	<0.001	0.07 [0.06]	0.20
	Never/Former	1.26 (1.4)	REF			
Maternal weekly alcohol consumption at time of pregnancy						
	>2 drinks	1.47 (1.5)	0.22 [0.05]	<0.001	0.26 [0.06]	<0.001
	1-2 drinks	1.38 (1.4)	0.06 [0.05]	0.23	0.03 [0.05]	0.54
	0 drinks	1.30 (1.4)	REF		REF	
Maternal smoking at age 9-11 interview						
	Current	1.76 (1.6)	0.34 [0.04]	<0.001	0.17 [0.06]	0.004
	Never/Former	1.24 (1.4)	REF		REF	
Mother's highest education by age 9-11 interview						
	Less than high school	1.64 (1.5)	0.25 [0.07]	<0.001	0.19 [0.08]	0.01
	High school	1.53 (1.5)	0.18 [0.04]	<0.001	0.13 [0.05]	0.05
	More than high school	1.27 (1.4)	REF		REF	

		Mean behavioral disinhibition (SD)	Unadjusted Beta [SE]	p- value	Adjusted ⁺ Beta [SE]	p-value
Mother reported separation from father by age 9–11 interview						
	Yes	1.78 (1.5)	0.31 [0.04]	<0.001	0.23 [0.05]	<0.001
	No	1.29 (1.4)	REF			
Percent below the poverty line* in the ages 9–11 neighborhood						
	1st Quartile (>3.0%)	1.46 (1.4)	0.01 [0.06]	0.80	0.02 [0.06]	0.72
	2nd Quartile	1.41 (1.5)	−0.02 [0.06]	0.74	0.02 [0.06]	0.70
	3rd Quartile	1.35 (1.5)	−0.06 [0.06]	0.32	−0.00 [0.06]	0.99
	4th Quartile (<1.0%)	1.44 (1.5)	REF		REF	
Birth order						
	1 st	1.24 (1.4)	−0.25 [0.07]	<0.001	−0.50 [0.08]	<0.001
	2 nd	1.37 (1.4)	−0.16 [0.07]	0.02	−0.34 [0.07]	<0.001
	3 rd	1.56 (1.5)	−0.04 [0.07]	0.58	−0.21 [0.07]	0.003
	4 th	1.35 (1.4)	−0.18 [0.08]	0.02	−0.30 [0.08]	<0.001
	≥5 th	1.62 (1.5)	REF		REF	
Birth weight						
	≤2500 grams	1.12 (1.4)	−0.23 [0.11]	0.03	−0.25 [0.11]	0.02
	>2500 grams	1.43 (1.5)	REF		REF	
		Correlation Coefficient	Unadjusted B	p-value	Adjusted ⁺ B	p-value
Child cognitive ability** at age 9–11 interview		−0.05	−0.00 [0.00]	0.008	−0.00 [0.00]	0.24
Child conduct problems at age 9–11 interview		0.13	0.21 [0.03]	<0.001	0.16 [0.03]	<0.001
Family income at age 9–11 interview		−0.05	−0.03 [0.01]	0.009	0.01 [0.01]	0.42
Maternal age at child's birth		−0.09	−0.02 [0.00]	<0.001	−0.03 [0.00]	<0.001
Maternal cognitive ability*** at age 9–11 interview		−0.04	−0.00 [0.00]	0.04	0.00 [0.00]	0.47

* Percentage of individuals living below the poverty line for a family of four in 1970

** As measured by the Peabody Picture Vocabulary Test

⁺ Adjusted for race, gender, age at adolescent interview, maternal smoking during pregnancy, maternal weekly alcohol consumption at the time of pregnancy, maternal smoking at age 9–11 interview, mother's highest education by age 9–11 interview, mothers separation from father by age 9–11 interview, percent in the neighborhood below the poverty line, birth weight, birth order, child conduct problems at age 9 to 11 interview, family income at age 9 to 11 interview, maternal age at child's birth, and maternal cognitive ability at age 9–11 interview.

Change in cognitive ability between age 9–11 and age 15–18 as a predictor of behavioral disinhibition at age 15–18: results of a longitudinal birth cohort in the United States (N=1,752)

Table 4

	N	Mean behavioral disinhibition	Unadjusted beta	P-value	Adjusted ⁺ beta	P-value
Moved down *	456	1.73 (1.5)	0.20 [0.0]	<0.001	0.19 [0.1]	<0.001
Moved up *	437	1.12 (1.4)	-0.24 [0.1]	<0.001	-0.20 [0.1]	<0.001
Stable *	859	1.41 (1.4)	REF		REF	

* Groups were defined by the within-sample age-standardized percentile on the Peabody Picture Vocabulary Test. Individuals were defined as stable if they remained in the same quartile of cognitive ability from age 9–11 to adolescence. Individuals were defined as moving up if their quartile on cognitive ability increased from age 9–11 to adolescence. Individuals were defined as moving down if their quartile on cognitive ability decreased from age 9–11 to adolescence.

⁺ Adjusted for race, gender, age at adolescent interview, maternal smoking during pregnancy, maternal weekly alcohol consumption at the time of pregnancy, maternal smoking at child's age 9–11, mother's highest education by age 9–11, mother's reported separation from father by age 9–11 interview, percent in the neighborhood below the poverty line, birth weight, birth order, child conduct disorder symptoms at age 9 to 11, family income, maternal age, and maternal cognitive ability.