Comprehensive Psychiatry 92 (2019) 1–6

Contents lists available at ScienceDirect

Comprehensive Psychiatry

journal homepage: www.elsevier.com/locate/comppsych

The etiology of antisocial personality disorder: The differential roles of adverse childhood experiences and childhood psychopathology

Matt DeLisi [a,*], Alan J. Drury [b], Michael J. Elbert [b]

[a] Iowa State University, United States of America
[b] U.S. Probation and Pretrial Services, United States of America

ARTICLE INFO

Keywords:
Antisocial personality disorder
adverse childhood experiences
psychopathology
offending
criminal career

ABSTRACT

Antisocial Personality Disorder (ASPD) is a severe personality disorder with robust associations with crime and violence, but its precise etiology is unknown. Drawing on near-population of federal correctional clients in the Midwestern United States, the current study examined antecedent background factors spanning adverse childhood experiences and childhood psychopathology. Greater adverse childhood experiences were associated with ASPD diagnosis with physical abuse showing associations with ASPD symptoms and sexual abuse with lifetime diagnosis for ASPD. Conduct Disorder was strongly linked to ASPD; however, Oppositional Defiant Disorder and ADHD had null associations. Given the role of environmental factors in the development of ASPD, greater criminological attention should be devoted to understanding how assorted forms of abuse and neglect coupled with childhood psychopathology contribute to ASPD especially given its linkages to severe criminal offending.

© 2019 The Author(s). Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Introduction

Antisocial Personality Disorder (ASPD) is a pervasive pattern of disregard for and violation of the rights of others occurring since age 15 years and including three or more of the following indicators: failure to conform to social norms with respect to lawful behaviors, deceitfulness, impulsivity, irritability and aggressiveness, reckless disregard for safety of self or others, consistent irresponsibility, and lack of remorse. An individual must be at least 18 years of age and have evidence of Conduct Disorder (CD) with onset before age 15 years [1] to meet criteria for a diagnosis. ASPD is the most socially destructive personality disorder given its mutuality with serious conduct problems, violence, and crime [2–4], produces extraordinary societal costs and aggregate social burden [5–7], and is highly prevalent in profiles of the most serious and antisocial criminal offenders [8–14].

The precise etiology of ASPD remains unknown. ASPD is a multifactorial construct with biological and environmental bases, and its heritability has been variously estimated at 38% [15], 50% [16], 56% [17], and 69% [18]. Although there is substantial genetic liability for ASPD, environmental sources also play a role in its developmental course, and the current study focuses on two of these environmental sources: adverse childhood experiences and childhood psychopathology.[1] Adverse childhood experiences, which encompass various forms of abuse, neglect, and impoverishment during childhood are prime examples of the negative socialization experiences that engender antisocial traits and behaviors. Similarly, childhood psychopathology, such as Attention-Deficit/Hyperactivity Disorder (ADHD), Oppositional Defiant Disorder (ODD), and CD frequently serve as stepping stones toward a lifespan of recurrent antisocial behavior as seen in ASPD.

2. Adverse childhood experiences, childhood psychopathology, and ASPD

The adverse childhood experiences framework has recently appeared in criminological research and has consistently shown that greater and more diverse exposure to various forms of abuse, neglect, and childhood adversity are associated with expansive health, mental health, and behavioral problems across the lifespan. Several studies found that not only do criminal offenders experience far greater adverse childhood experiences than those in the general population, but also that accumulating adverse experiences contribute to the most serious,

* Corresponding author at: Iowa State University, 510 Farm House Lane, 203A East Hall, Ames, IA 50011-1070, United States of America.
E-mail address: delisi@iastate.edu (M. DeLisi).

[1] The current study focuses on childhood psychopathology as an external, environmental factor. However, we also recognize there is substantial evidence that ASPD and other antisocial conditions, including childhood psychopathology, have shared genetic variance that is underlain by a common latent factor (see, [19–23]). Unfortunately, we do not have twin data to statistically model these relationships.

https://doi.org/10.1016/j.comppsych.2019.04.001
0010-440X/© 2019 The Author(s). Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

violent, and chronic manifestations of criminal offending [24–28], ones that would be compatible with offenders that evince ASPD.[2]

A variety of studies have examined the associations between adverse childhood experiences and ASPD and produced varying results. Using prospective longitudinal data, Fergusson, Boden, and Horwood [33] examined the association between childhood sexual abuse, childhood physical abuse, and ASPD and found that the prevalence of ASPD at ages 18–21 and 21–25 was two to four times greater among those that had been sexually abused compared to those who had not. Similarly, those who experienced regular physical abuse or severe physical abuse had ASPD at a prevalence that was two to seven times higher than those who were not physically abused. In multivariate models, sexual abuse predicting ASPD; however, physical abuse did not in the fully adjusted model. Douglas et al. [34] studied three adverse childhood experiences, witnessing or experiencing a violent crime, sexual abuse, and physical abuse among a sample of subjects dependent on alcohol, cocaine, or opioids and found adverse childhood experiences increased the odds of ASPD by 1.47 increased odds.

Battle et al.' [35] used data from the Collaborative Longitudinal Personality Disorders Study and studied the effects of emotional abuse, verbal abuse, physical abuse, caretaker sexual abuse, non-caretaker sexual abuse, physical neglect, emotional neglect, inconsistent treatment, denial of feelings, and failure to protect on the development of personality disorders. They found that only verbal abuse and caretaker sexual abuse were associated with increased odds of developing ASPD, and null findings were seen for most forms of abuse and most personality disorders. The exception to this was Borderline Personality Disorder where seven forms of adverse childhood experiences increased the likelihood of it.

Although not formulated as adverse childhood experiences studies per se, other works identified risk factors for ASPD that are congruent with diverse forms of abuse and neglect. Lahey, Loeber, Burke, and Applegate [36] found that offenders whose mother exhibited ASPD symptoms and attendant antisocial conduct were more likely to develop ASPD. Farrington [37] identified several childhood and adolescent risk factors for ASPD using data from the Cambridge Study in Delinquent Development. Although the study mostly focused on socioeconomic, family, and school variables, Farrington also found that two forms of neglect—father uninterested in child and parent uninterested in the child's education—conferred elevated risk for ASPD.

Other research examined various forms of childhood psychopathology and those linkages to the development of ASPD. For instance, Lynam [11] advanced the fledgling psychopathy hypothesis that posited that children and adolescents that have clinical conduct problems coupled with symptoms of hyperactivity, impulsivity, and attention problems are effectively bourgeoning psychopaths that would also likely later meet diagnostic criteria for ASPD. For example, children that exhibit chronically high levels of aggression have been shown to be at nearly 27 times increased odds of developing ASPD in adulthood [38] suggesting that ASPD can represent the culmination of a lifetime of conduct problems and attendant psychopathology.[3]

In clinical terms, youth with severe behavioral problems would disproportionately also likely receive diagnoses for ADHD, ODD, and CD on the way toward an ASPD diagnosis. However, empirical work revealed that childhood psychopathology unevenly predicts subsequent conduct problems and ASPD [47]. Some studies found that CD, ODD, or ADHD were predictive of ASPD whereas others reported null effects for ADHD and ASPD [36][48]. Despite conflicting findings about the precise developmental course and interrelationship between various manifestations of childhood psychopathology, there is little doubt these disorders portend significant impairments in later life, impairments that effectively overlap with ASPD symptoms. To illustrate, Erskine et al. [49] meta-analyzed 98 studies and found that ADHD was predictive of high school dropout, unemployment, substance use disorders, and arrest activity while CD strongly predicted dropout, substance use disorders, and criminal violence.

Indeed, in a research review, Loeber, Burke, Lahey, Winters, and Zera [50] advised there is a general developmental progression from ODD during early childhood to CD during adolescence to ASPD during young adulthood. Moreover, they advised that ADHD during childhood hastened the onset of CD and exacerbated the severity of CD symptoms and conduct problems. But, much remains unclear. According to Loeber et al. [50], "ODD, CD, and later APD [Antisocial Personality Disorder] may be hierarchically and developmentally related. Broad pathways between the disorders as well as more specific symptomatic and conceptual pathways have been tentatively identified and appear to have demonstrated utility…However, further investigation to better elucidate the clinical and prognostic implications of these comorbid conditions remains to be conducted." Following the advisement of Loeber et al. [50], the current study examined antecedent background factors spanning adverse childhood experiences and childhood psychopathology among federal offenders that had ASPD symptomatology or lifetime diagnostic history for ASPD.

3. Method

3.1. Participants and procedures

The current study used retrospective, archival data from the total population of 865 active correctional clients in a federal jurisdiction in the Midwestern United States. Two clients had incomplete data thus the analytical sample is 863. All clients were on supervised release after serving a confinement sentence under the supervision of the Bureau of Prisons. The sample was 84% male, 16% female, 79.4% white, 20.6% African American, 92% non-Hispanic, 8% Hispanic, and the mean age was 44 years. The clients were supervised for a range of offenses and the most prevalent instant conviction offenses were distribution of methamphetamine (35%), felon in possession of firearm (13%), bank fraud, money laundering, and/or identity theft (13%), distribution of cocaine base (12%), possession or manufacturing of child pornography (6.5%), distribution of marijuana (6%), use of firearm during a drug trafficking offense (4.5%), and distribution of cocaine (3.6%). The clients were diverse in terms of their criminal history, offending background, and criminal justice system involvement. The federal criminal history rank employs a 6-point system where I = lowest risk and VI = highest risk in terms of criminal history. In these data, 35.4% were Criminal History Rank I, 13.5% were Criminal History Rank II, 18.7% were Criminal History Rank III, 12.5% were Criminal History Rank IV, 6.7% were Criminal History Rank V, and 13.2% were Criminal History Rank VI. In terms of a broad mix of offenders with heterogeneous antisocial histories, the current data are consistent with prior research (see, [29,51–54]).

We employed two methods of data collection. First, we electronically extracted all data in the client's Probation/Pretrial Services Automated Case Tracking System (PACTS) file and converted the data to an Excel spreadsheet. PACTS is the case management platform used in all 94 federal districts to track federal defendants. This electronic extraction contained information on numerous variables including demographics, case information, conditions, criminal history indices, and other documents relevant to the client's social and criminal history. Second, the senior author manually collected additional data from

[2] Although prior criminological studies of adverse childhood experiences did not focus on ASPD per se, studies have shown that diverse forms of abuse and trauma are significantly associated with offenders that engage in capital offending spanning homicide, rape, kidnapping, and armed robbery [29,30], sexual homicide [31], and contact sexual offenses [32], offender types that are significantly likely to display symptoms of ASPD.

[3] A variety of studies using different data sources have empirically supported Lynam's fledgling psychopathy hypothesis (e.g., [39–44]) although there is also discordant evidence [45]. Lynam's work is not the only prominent theoretical model that cites ODD, CD, ADHD, and ASPD as central to antisocial behavior, these diagnostic conditions are also salient to the neuropsychological deficits seen in Moffitt's [12] developmental taxonomy (see, [46]).

presentence reports (PSR), offender documents from the Bureau of Prisons, local, state, and national criminal histories, psychological and psychiatric reports, treatment reports, and other relevant documents located in PACTS.

During the PSR interview process, defendants self-reported their address and residency history and requests for criminal history were sent to all of those areas. In addition, defendants were questioned about juvenile placements and if the defendant lived in any other location than with their parents, such as foster care, group homes, juvenile homes, state facilities, and others. Based on this information, verification was sent by United States Probation to those facilities. Additional self-reported information on antisocial behavior was gleaned from official mental health and educational records. All variables were coded and entered into the Excel spreadsheet by the senior author and upon completion, the data were transferred into Stata/IC 14.2 for data analyses. Research approval for the study was provided by the Chief District Judge in this federal jurisdiction.

3.2. Measures

3.2.1. Adverse childhood experiences

All adverse childhood experiences were scored on a 3-point ordinal scale (0 = no evidence, 1 = some evidence, 2 = definite evidence) based on information in the client's PACTS file. Evidence for adverse childhood experiences was derived from offender self-reports of the adverse experiences, official record of these experiences from human/social service agencies, official record of these experiences from juvenile justice or out-of-home placement records, and other official documents from the client's PSR. Cases where there were multiple reports of abuse with corroboration from other data sources were coded as 2. Cases were there was some evidence from at least one source were coded as 1. The prevalence/endorsement for the ACEs were: parent exposure to drugs (80.3% no evidence, 8.8% some evidence, 10.9% definite evidence), father neglect (59.6% no evidence, 7.7% some evidence, 32.8% definite evidence), mother neglect (77% no evidence, 5.7% some evidence, 17.3% definite evidence), physical abuse (68.4% no evidence, 2.4% some evidence, 29.2% definite evidence), emotional/verbal abuse (70.3% no evidence, 3.5% some evidence, 26.2% definite evidence), and sexual abuse (86.4% no evidence, 0.4% some evidence, 13.2% definite evidence). A summary measure of adverse childhood experiences was also used (M = 2.88, SD = 3.60, range = 0–12).[4]

3.2.2. Childhood psychopathology

The current study used secondary data for all psychiatric diagnoses including behavioral disorders, personality disorders, and paraphilic disorders. Clients that had a documented DSM-IV lifetime diagnosis in their file were rated as definite evidence (=2). Clients that had documented symptoms of a condition but not enough to warrant a full diagnosis were rated as some evidence (=1). Clients that had no evidence of a condition in their file were rated as no evidence (=0). The current authors did not make any diagnoses. The prevalence for childhood psychopathology was: ADHD (83.78% no evidence, 0.23% some evidence, 15.99% definite evidence), ODD (97.37% no evidence, 0.58% some evidence, 12.05% definite evidence), CD (78.56% no evidence, 1.51% some evidence, 19.93% definite evidence), and ASPD (69.99% no evidence, 5.56% some evidence, 24.45% definite evidence).

3.2.3. Covariates

Several criminal career and behavioral indicators were included to guard against confounding effects. These were age of arrest onset (M = 23.57, SD = 1.46, range = 6–78), total arrest charges (M = 14.31, SD = 14.75, range = 1–97), total convictions (M = 8.51, SD = 8.26, range = 1–67), total prison commitments (M = 2.33, SD = 2.05, range = 1–16), probation and community sentence revocations (M = 0.67, SD = 1.20, range = 0–12), murder arrest charges (M = 0.02, SD = 0.15, range = 0–2), rape/sexual abuse arrest charges (M = 0.14, SD = 0.63, range = 0–7), felony/aggravated assault arrest charges (M = 0.61, SD = 1.54, range = 0–14), other assault charges (M = 0.57, SD = 1.42, range = 0–6), domestic violence arrest charges (M = 0.73, SD = 1.63, range = 0–13), alcohol use onset (M = 14.9, SD = 3.27, range = 4–40), and school expulsion (89.8% no, 10.2% yes).

3.3. Analytical strategy

Multinomial logistic regression with relative risk ratios (RRR) and bootstrapped standard errors with 500 replications modeled the association of the covariates on ASPD symptoms (presented in the first section of the table) and ASPD diagnosis (presented in the second section of the table) with no ASPD evidence as the omitted reference group. One-way analysis of variance (ANOVA) compared clients on the covariates by ASPD status. Omnibus F statistic and effect sizes ($\eta 2$) were provided and can be interpreted as $\eta 2 = 0.02$ is a small effect, $\eta 2 = 0.13$ is a medium effect and $\eta 2 = 0.26$ is a large effect as specified by Cohen [55].

4. Findings

4.1. Multinomial logistic regression model with cumulative adverse childhood experiences

As shown in Table 1, several significant effects emerged. Compared to clients that had no evidence of ASPD, those with CD (RRR = 3.09, z = 3.12, $p < .01$) and those with an early arrest onset (RRR = 0.92, z = −2.44, $p < .05$) were more likely to exhibit symptoms of ASPD. Adverse childhood experiences were not significantly associated with symptoms. In contrast, adverse childhood experiences were significantly associated with a formal diagnosis for ASPD (RRR = 1.09, z = 2.48, $p < .05$). Conduct Disorder (RRR = 11.10, z = 6.92, $p < .001$) and arrest onset (RRR = 0.94, z = −3.06, $p < .01$) remained significant. ADHD, school expulsion, and alcohol onset were not significant in either model.

Table 1
Multinomial logistic regression model for antisocial personality disorder (cumulative adverse childhood experiences).

Variable	RRR	BSE	z
Symptom group			
Adverse childhood experiences	0.99	0.04	−0.22
ADHD	1.01	1.07	0.01
Oppositional defiant disorder	0.32	1.58	−0.23
Conduct disorder	3.09	1.12	3.12**
School expulsion	1.04	0.31	0.14
Arrest onset	0.92	0.03	−2.44*
Alcohol onset	0.94	0.05	−1.13
Diagnostic group			
Adverse childhood experiences	1.09	0.04	2.48*
ADHD	1.01	0.27	0.02
Oppositional defiant disorder	1.28	0.65	0.48
Conduct disorder	11.10	3.86	6.92***
School expulsion	1.22	0.27	0.89
Arrest onset	0.94	0.02	−3.06**
Alcohol onset	0.95	0.06	−0.84
Wald χ^2	283.59***		
Pseudo R^2	0.455		

Note. RRR = relative risk ratio, BSE = bootstrapped standard error, z = z-score,
* $p < .05$.
** $p < .01$.
*** $p < .001$.

[4] Gender differences existed for ACEs. Female clients had significantly higher total scores on the summary measure of ACEs ($Mf_{emale} = 4.97$, $M_{male} = 2.49$, $t = 7.62$, $p < .001$). Much of this was driven by higher prevalence of sexual abuse among women (prevalence 35%) compared to men (prevalence 9%).

Table 2
Multinomial logistic regression model for antisocial personality disorder (individual adverse childhood experiences).

Variable	RRR	BSE	z
Symptom group			
Father neglect	1.08	0.25	0.33
Mother neglect	1.08	0.37	0.22
Physical abuse	1.51	0.28	2.22*
Emotional/verbal abuse	0.62	0.19	−1.55
Sexual abuse	1.33	0.49	0.83
Parent exposure to drugs	0.46	0.21	−1.66
ADHD	1.03	1.49	0.02
Oppositional defiant disorder	0.29	1.52	−0.24
Conduct disorder	3.15	1.64	2.21*
School expulsion	0.99	0.34	−0.01
Arrest onset	0.92	0.05	−2.98**
Alcohol onset	0.93	0.05	−1.30
Diagnostic group			
Father neglect	0.86	0.20	−0.64
Mother neglect	1.18	0.37	0.52
Physical abuse	0.91	0.25	−0.34
Emotional/verbal abuse	0.99	0.31	−0.02
Sexual abuse	1.69	0.44	2.02*
Parent exposure to drugs	1.20	0.36	0.60
ADHD	1.03	0.32	0.08
Oppositional defiant disorder	1.32	0.79	0.46
Conduct disorder	11.46	5.67	4.92***
School expulsion	1.29	0.34	0.99
Arrest onset	0.93	0.02	−3.56***
Alcohol onset	0.95	0.06	−0.85
Wald χ^2	215.54***		
Pseudo R^2	0.467		

Note. RRR = relative risk ratio, BSE = bootstrapped standard error, z = z-score.
* p < .05.
** p < .01.
*** p < .001.

4.2. Multinomial logistic regression model with individual adverse childhood experiences

As shown in Table 2, differential effects for adverse childhood experiences emerged while the significant effects of CD and arrest onset from the prior model remained. Physical abuse (RRR = 1.51, z = 2.22, $p < .05$) was positively associated with symptoms of ASPD, along with CD (RRR = 3.15, z = 2.21, p < .05) and arrest onset (RRR = 0.92, z = −2.98, $p < .01$). Sexual abuse (RRR = 1.69, z = 2.02, p < .05) was significantly associated with a formal diagnosis for ASPD, along with CD (RRR = 11.46, z = 4.92, $p < .001$) and arrest onset (RRR = 0.92, z = −3.56, p < .001).[5]

4.3. One-way ANOVA for criminal career outcomes by antisocial personality disorder status

As shown in Table 3, ASPD has robust associations with numerous measures of the criminal career. Using Cohen's [55] effect size thresholds for η^2 (0.02 small effect, 0.13 medium effect, and 0.26 large effect), most of these differences were medium to large in magnitude. A clear gradient was observed whereby offenders with no evidence of ASPD had the least severe criminal careers followed by those with symptoms and those with formal diagnostic history. Sharp differences were seen for arrest onset ($F[2, 860] = 97.35$, $p = .001$, $\eta^2 = 0.18$), total arrests ($F[2, 860] = 322.09$, $p = .001$, $\eta^2 = 0.43$), total convictions ($F[2, 860] = 303.55$, $\eta^2 = 0.41$), total prison sentences ($F[2, 860) = 181.71$, $p = .001$, $\eta^2 = 0.30$), probation revocations ($F[2, 860] = 44.01$, $p = .001$, $\eta^2 = 0.09$), murder arrests ($F[2, 860] = 11.88$, p = .001, $\eta^2 = 0.03$), rape arrests ($F[2, 860] = 17.68$, p = .001, $\eta^2 = 0.04$), felony assault arrests ($F[2, 860] = 172.73$, p = .001, $\eta^2 = 0.29$), other assault arrests ($F[2, 860] = 87.27$, p = .001, $\eta^2 = 0.17$), and domestic violence arrests ($F[2, 860] = 60.18$, p = .001, $\eta^2 = 0.13$).

Table 3
One-way ANOVA for criminal career outcomes by antisocial personality disorder status.

Variable	None	Symptoms	Diagnosis	F	η^2
Arrest onset	27.02	18.85	14.74	97.35***	0.18
Total arrests	8.42	14.60	31.08	322.09***	0.43
Total convictions	5.22	9.69	17.68	303.55***	0.41
Total prison sentences	1.63	2.73	4.25	181.71***	0.30
Probation revocations	0.44	0.75	1.30	44.01***	0.09
Murder arrests	0.005	0.02	0.06	11.88***	0.03
Rape arrests	0.06	0.48	0.29	17.68***	0.04
Felony assault arrests	0.13	0.31	2.05	172.73***	0.29
Other assault arrests	0.22	0.42	1.59	87.27***	0.17
Domestic violence arrests	0.39	0.65	1.73	60.18***	0.13

*** $p < .001$.

5. Discussion

The adverse childhood experiences framework is an influential one in the social and behavioral sciences [24,56–60] and recent studies examined the differential effects of specific forms of abuse and neglect on specific forms of crime. The current study indicates this is a worthwhile research program as the effects of adverse childhood experiences are variable and dependent on model specification. When summed, adverse childhood experiences did not have an association with symptoms of ASPD, but did have a significant association with diagnostic history of the disorder. The latter finding is important because it withstood controls for powerful covariates relating to childhood psychopathology and the emergence of substance use and the criminal career.

Disaggregating adverse childhood experiences reveals these forms of abuse and neglect do not have straightforward adverse effects on subsequent psychopathology, which is consistent with prior research ([35,61]; Perez, Jennings, & [24]). Physical abuse emerged as a significant predictor of symptoms of ASPD, why might this be the case? Based on the current authors' various conversations with offenders that have incurred physical abuse, we suspect that physical abuse engenders feelings of hostility, contempt, and distrust of adult authority figures, characteristics that set the stage for the personality style that among other symptoms is dominated by irritability, aggression, and the rationalized indifference to others. Unlike emotional and verbal abuse, physical abuse also produces visible injuries that can serve as the impetus for out-of-home placement, which for many offenders with severe conduct problems, is the first step in a long procession of institutional placement.[6]

The significant association between sexual abuse and having a formal diagnosis for ASPD was both unsurprising and surprising. On one hand, sexual abuse is the most severe example of an adverse childhood experience, one that often forecasts the most substantial adjustment problems, and thus it makes intuitive sense that it would be associated with the most severe diagnostic condition. On the other hand, there is evidence that sexual abuse has a disproportionate causal effect on sexual offending (cf., [29,40,59,63–66]), not necessarily on

[5] The multinomial regression models were also executed using dichotomous measures for all adverse childhood experiences and a summary measure of adverse childhood experiences based on the dummy terms. The results were substantively identical. The ACE output for Table 1 was RRR = 0.99, BSE = 0.08, z = −0.11 and RR = 1.14, BSE = 0.07, z = 2.07 and for Table 2, physical abuse was the only ACE that was significantly associated with ASPD symptoms (RRR = 2.18, BSE = 0.79, z = 2.14) and sexual abuse was the only ACE that was significantly associated with ASPD diagnosis (RRR = 2.71, BSE = 1.13, z = 2.39).

[6] Although the current focus is on adverse childhood experiences and thus victimization experiences among these clients, it is also important to recognize that given the significant conduct problems of these offenders, many also perpetrated violence against their caregivers. Recent research [62] on child-to-parent violence perpetrators reported risk profiles including conduct problems, family dysfunction, poor school performance, and substance use—profiles very consistent with the current data.

generalized criminal offending and a globally antisocial disposition such as that embodied by ASPD. Overall, the significant effects for physical abuse and sexual abuse are consistent with recent meta-analytic research that found that sexual abuse and physical abuse among various forms of maltreatment are more strongly linked to aggressive forms of deviance [67].

Adverse childhood experiences evoke the early-life family environments of criminal offenders, and epidemiological research has shown there is considerable heterogeneity in the familial progression of ASPD. Using data from two waves of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), for instance, Vaughn, Salas-Wright, DeLisi, and Qian [14] found that those with an ASPD diagnosis, more than 70% had minimal family history of antisocial behavior, more than 9% had parental and progeny behavioral problems, and more than 20% had multigenerational history of problem behaviors evidenced by widespread criminal versatility and violence. These family background help to contextualize the current findings. In families where there is limited evidence of antisocial conduct and an individual presents with what seems to be de novo ASPD, adverse childhood experiences might be particularly damaging and in fact help to explain why an individual would present with ASPD symptoms when reared in an otherwise prosocial setting. At the other end of the spectrum in multigenerational antisocial families where adverse childhood experiences are endemic [68], the most extreme forms of abuse—sexual abuse—contribute to the development of ASPD.

We stress that adverse childhood experiences are an example of a phenomenon where even null findings are substantially important, and that substantive value potentially even exceeds any statistical effects that are produced. For instance, even if father neglect, mother neglect, emotional/verbal abuse, and parent exposure to drugs had non-significant effects in the models, these experiences nevertheless worsened the overall quality of life of these offenders and played a role in their maladjustment if for no other reason they illustrated a negative, noxious, and unhealthy rearing environment. Given that ACEs can have differential effects, we encourage researchers to measure adverse experiences both individually and collectively as well as potentially create interaction variables between ACEs and other criminological risk factors.

Childhood psychopathology variables produced two clear findings. First, neither ODD nor ADHD were significantly associated with ASPD symptoms or ASPD diagnostic history, findings that are consistent with prior research [36] and discordant with other research [36,48,69]. Second, and in marked contrast, CD exerted strong associations with ASPD with RRR values ranging between 3 and 12. These findings were expected given that CD is a prodrome of ASPD and are consistent with prior research that has shown that approximately 80% to 90% of cases of ASPD also exhibited CD earlier in life [69,70].

However it is engendered, there is no question that ASPD exerts a destructive toll in terms of criminal career severity and violence involvement. A clear gradient from no evidence to symptoms to formal diagnosis illustrated an earlier-starting, more chronic, more violent, and more noncompliant criminal career. One exception to this trend was for rape arrests where clients with ASPD symptoms averaged more arrest charges than those with a formal diagnosis. Nevertheless, the high volume of arrest charges for felony or aggravated assault, other assault, and domestic assault suggest an increased likelihood of continuity in offenders replicated adverse childhood experiences, such as physical abuse in their own home environments.

The ASPD-criminal career gradient effects have straightforward practical value for correctional practitioners. It is common for supervising officers to be cognizant of specific risk factors in their client's criminal career, such as history of use of weapons, history of use of violence against law enforcement and other correctional officials, and occasionally, even specific crimes of interest. A brief review of the client's diagnostic history is also helpful not only for understanding how specific sets of symptoms or diagnosis for ASPD are associated with offending extremity, but also for anticipating responsivity factors that can impede or enhance supervision.

In closing, we cite limitations to the current study that future research should overcome. The current data are archival and cross-sectional, and longitudinal designs are needed not only to specify causal ordering of variables, but also to illuminate precise ways that adverse childhood experiences contribute to the onset of disruptive behaviors during childhood and adolescence. In some of the clients' PSRs, there was a close temporal coincidence between the occurrence of a significant childhood trauma and the emergence of externalizing symptoms, but these anecdotal accounts require empirical study. A perennial limitation of measures of adverse childhood experiences is that most are binary measures whereas most forms of abuse and neglect are ongoing and chronic. We hypothesize that adverse childhood experiences measured with count data could reveal different relationships with ASPD because the most abusive background experiences would appear in the data. Additionally, there are important biosocial and genetic features involved in the development of antisocial personality features and criminal careers [71–73], unfortunately we lacked data to explore these. Given the social burden and voluminous criminal consequences of ASPD, we hope other criminologists expand their focus on this critical personality disorder.

References

[1] American Psychiatric Association. Diagnostic and statistical manual of mental disorders, fifth edition (DSM-5). Arlington, VA: American Psychiatric Publishing; 2013.
[2] Black DW. Bad boys, bad men: confronting antisocial personality disorder. New York, NY: Oxford University Press; 2013.
[3] Martens WH. Antisocial and psychopathic personality disorders: causes, course, and remission—a review article. Int J Offender Ther Comp Criminol 2000;44(4):406–30.
[4] Moran P. The epidemiology of antisocial personality disorder. Soc Psychiatry Psychiatr Epidemiol 1999;34(5):231–42.
[5] Holzer KJ, Vaughn MG. Antisocial personality disorder in older adults: a critical review. J Geriatr Psychiatry Neurol 2017;30(6):291–302.
[6] Scott S, Knapp M, Henderson J, Maughan B. Financial cost of social exclusion: follow up study of antisocial children into adulthood. Br Med J 2001;323(7306):191–5.
[7] Vaughn MG, Fu Q, Beaver D, DeLisi M, Perron B, Howard M. Are personality disorders associated with social welfare burden in the United States? J Pers Disord 2010;24(6):709–20.
[8] Abram KM. The effect of co-occurring disorders on criminal careers: interaction of antisocial personality, alcoholism, and drug disorders. Int J Law Psychiatry 1989;12(2-3):133–48.
[9] DeLisi M. Career criminals in society. Thousand Oaks, CA: Sage; 2005.
[10] DeLisi M, Drury AJ, Caropreso D, Heinrichs T, Tahja KN, Elbert MJ. Antisocial personality disorder with or without antecedent conduct disorder: the differences are psychiatric and paraphilic. Crim Justice Behav 2018;45(6):902–17.
[11] Lynam DR. Early identification of chronic offenders: Who is the fledgling psychopath? Psychol Bull 1996;120(2):209–34.
[12] Moffitt TE. Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. Psychol Rev 1993;100(4):674–701.
[13] Shepherd SM, Campbell RE, Ogloff JR. Psychopathy, antisocial personality disorder, and reconviction in an Australian sample of forensic patients. Int J Offender Ther Comp Criminol 2018;62(3):609–28.
[14] Vaughn MG, Salas-Wright CP, DeLisi M, Qian Z. The antisocial family tree: family histories of behavior problems in antisocial personality in the United States. Soc Psychiatry Psychiatr Epidemiol 2015;50(5):821–31.
[15] Torgersen S, Czajkowski N, Jacobson K, Reichborn-Kjennerud T, Røysamb E, Neale MC, et al. Dimensional representations of DSM-IV cluster B personality disorders in a population-based sample of Norwegian twins: a multivariate study. Psychol Med 2008;38(11):1617–25.
[16] Mason DA, Frick PJ. The heritability of antisocial behavior: a meta-analysis of twin and adoption studies. J Psychopathol Behav Assess 1994;16(4):301–23.
[17] Ferguson CJ. Genetic contributions to antisocial personality and behavior: a meta-analytic review from an evolutionary perspective. J Soc Psychol 2010;150(2):160–80.
[18] Fu Q, Heath AC, Bucholz KK, Nelson E, Goldberg J, Lyons MJ, et al. Shared genetic risk of major depression, alcohol dependence, and marijuana dependence: contribution of antisocial personality disorder in men. Arch Gen Psychiatry 2002;59(12):1125–32.
[19] Krueger RF. The structure of common mental disorders. Arch Gen Psychiatry 1999;56(10):921–6.
[20] Larsson H, Tuvblad C, Rijsdijk FV, Andershed H, Grann M, Lichtenstein P. A common genetic factor explains the association between psychopathic personality and antisocial behavior. Psychol Med 2007;37(1):15–26.
[21] Reichborn-Kjennerud T, Krueger RF, Ystrom E, Torvik FA, Rosenström TH, Aggen SH, et al. Do DSM-5 Section II personality disorders and Section III personality trait domains reflect the same genetic and environmental risk factors? Psychol Med 2017;47(12):2205–15.

[22] Rosenström T, Ystrom E, Torvik FA, Czajkowski NO, Gillespie NA, Aggen SH, et al. Genetic and environmental structure of DSM-IV criteria for antisocial personality disorder: a twin study. Behav Genet 2017;47(3):265–77.
[23] Young SE, Stallings MC, Corley RP, Krauter KS, Hewitt JK. Genetic and environmental influences on behavioral disinhibition. Am J Med Genet 2000;96(5):684–95.
[24] Baglivio MT. On cumulative childhood traumatic exposure and violence/aggression: the implications of adverse childhood experiences (ACE). In: Vazsonyi AT, Flannery DJ, DeLisi M, editors. The Cambridge handbook of violent behavior and aggression. 2nd ed. New York, NY: Cambridge University Press; 2018. p. 467–87.
[25] Baglivio MT, Wolff KT, Piquero AR, Epps N. The relationship between adverse childhood experiences (ACE) and juvenile offending trajectories in a juvenile offender sample. J Crim Just 2015;43(3):229–41.
[26] Craig JM, Piquero AR, Farrington DP, Ttofi MM. A little early risk goes a long bad way: adverse childhood experiences and life-course offending in the Cambridge study. J Crim Just 2017;53:34–45.
[27] Fox BH, Perez N, Cass E, Baglivio MT, Epps N. Trauma changes everything: examining the relationship between adverse childhood experiences and serious, violent and chronic juvenile offenders. Child Abuse Negl 2015;46:163–73.
[28] Wolff KT, Baglivio MT. Adverse childhood experiences, negative emotionality, and pathways to juvenile recidivism. Crime Delinq 2017;63(12):1495–521.
[29] Drury A, Heinrichs T, Elbert M, Tahja K, DeLisi M, Caropreso D. Adverse childhood experiences, paraphilias, and serious criminal violence among federal sex offenders. J Crim Psychol 2017;7(2):105–19.
[30] Trulson CR, Haerle DR, Caudill JW, DeLisi M. Lost causes: blended sentencing, second chances, and the Texas Youth Commission. Austin, TX: University of Texas Press; 2016.
[31] DeLisi M, Beauregard E. Adverse childhood experiences and criminal extremity: new evidence for sexual homicide. J Forensic Sci 2018;63(2):484–9.
[32] Levenson JS, Willis GM, Prescott DS. Adverse childhood experiences in the lives of male sex offenders: Implications for trauma-informed care. Sex Abuse 2016;28(4):340–59.
[33] Fergusson DM, Boden JM, Horwood LJ. Exposure to childhood sexual and physical abuse and adjustment in early adulthood. Child Abuse Negl 2008;32(6):607–19.
[34] Douglas K, Chan G, Gelernter J, Arias AJ, Anton RF, Poling J, et al. 5-HTTLPR as a potential moderator of the effects of adverse childhood experiences on risk of antisocial personality disorder. Psychiatr Genet 2011;21(5):240–8.
[35] Battle CL, Shea MT, Johnson DM, Yen S, Zlotnick C, Zanarini MC, et al. Childhood maltreatment associated with adult personality disorders: findings from the Collaborative Longitudinal Personality Disorders Study. J Pers Disord 2004;18(2):193–211.
[36] Lahey BB, Loeber R, Burke JD, Applegate B. Predicting future antisocial personality disorder in males from a clinical assessment in childhood. J Consult Clin Psychol 2005;73(3):389–99.
[37] Farrington DP. Psychosocial predictors of adult antisocial personality and adult convictions. Behav Sci Law 2000;18(5):605–22.
[38] Schaeffer CM, Petras H, Ialongo N, Poduska J, Kellam S. Modeling growth in boys' aggressive behavior across elementary school: links to later criminal involvement, conduct disorder, and antisocial personality disorder. Dev Psychol 2003;39(6):1020–35.
[39] DeLisi M, Dansby T, Peters DJ, Vaughn MG, Shook JJ, Hochstetler A. Fledgling psychopathic features and pathological delinquency: new evidence. American Journal of Criminal Justice 2014;39(3):411–24.
[40] DeLisi M, Kosloski AE, Vaughn MG, Caudill JW, Trulson CR. Does childhood sexual abuse victimization translate into juvenile sexual offending? New evidence. Violence Vict 2014;29(4):620–35.
[41] DeLisi M, Vaughn M, Beaver KM, Wexler J, Barth AE, Fletcher JM. Fledgling psychopathy in the classroom: ADHD subtypes, psychopathy, and reading comprehension in a community sample of adolescents. Youth Violence Juvenile Justice 2011;9(1):43–58.
[42] Gresham FM, Lane KL, Lambros KM. Comorbidity of conduct problems and ADHD: Identification of "fledgling psychopaths". J Emot Behav Disord 2000;8(2):83–93.
[43] Johansson P, Kerr M, Andershed H. Linking adult psychopathy with childhood hyperactivity-impulsivity-attention problems and conduct problems through retrospective self-reports. J Pers Disord 2005;19(1):94–101.
[44] Lynam DR. Pursuing the psychopath: capturing the fledgling psychopath in a nomological net. J Abnorm Psychol 1997;106(3):425–38.
[45] Michonski JD, Sharp C. Revisiting Lynam's notion of the "fledgling psychopath": are HIA-CP children truly psychopathic-like? Child Adolesc Psychiatry Ment Health 2010;4(1):24–32.
[46] Raine A, Moffitt TE, Caspi A, Loeber R, Stouthamer-Loeber M, Lynam D. Neurocognitive impairments in boys on the life-course persistent antisocial path. J Abnorm Psychol 2005;114(1):38–49.
[47] Lahey BB, Loeber R, Burke J, Rathouz PJ, McBurnett K. Waxing and waning in concert: dynamic comorbidity of conduct disorder with other disruptive and emotional problems over 17 years among clinic-referred boys. J Abnorm Psychol 2002;111(4):556–67.
[48] Storebø OJ, Simonsen E. The association between ADHD and antisocial personality disorder (ASPD): a review. J Atten Disord 2016;20(10):815–24.
[49] Erskine HE, Norman RE, Ferrari AJ, Chan GC, Copeland WE, Whiteford HA, et al. Long-term outcomes of attention-deficit/hyperactivity disorder and conduct disorder: a systematic review and meta-analysis. J Am Acad Child Adolesc Psychiatry 2016;55(10):841– .
[50] Loeber R, Burke JD, Lahey BB, Winters A, Zera M. Oppositional defiant and conduct disorder: a review of the past 10 years, part I. J Am Acad Child Adolesc Psychiatry 2000;39(12):1468–84.
[51] Cohen TH, Spidell MC. How dangerous are they: an analysis of sex offenders under federal post-conviction supervision. Fed Probat 2016;80(2):21–32.
[52] Cohen TH, VanBenschoten SW. Does the risk of recidivism for supervised offenders improve over time: examining changes in the dynamic risk characteristics for offenders under federal supervision. Fed Probat 2014;78(2):41–56.
[53] DeLisi M, Caropreso DE, Drury AJ, Elbert MJ, Evans JL, Heinrichs T, et al. The dark figure of sexual offending: new evidence from federal sex offenders. J Crim Psychol 2016;6(1):3–15.
[54] Walters GD, Cohen TH. Criminal thought process as a dynamic risk factor: variable- and person-oriented approaches to recidivism prediction. Law Hum Behav 2016;40(4):411–9.
[55] Cohen J. Statistical power analysis for the behavioral sciences. . 2nd ed. New York, NY: Psychology Press; 1988.
[56] Altintas M, Bilici M. Evaluation of childhood trauma with respect to criminal behavior, dissociative experiences, adverse family experiences and psychiatric backgrounds among prison inmates. Compr Psychiatry 2018;82:100–7.
[57] Keyes KM, Eaton NR, Krueger RF, McLaughlin KA, Wall MM, Grant BF, et al. Childhood maltreatment and the structure of common psychiatric disorders. Br J Psychiatry 2012;200(2):107–15.
[58] Malvaso CG, Delfabbro PH, Day A, Nobes G. The maltreatment-violence link: exploring the role of maltreatment experiences and other individual and social risk factors among young people who offend. J Crim Just 2018;55:35–45.
[59] Reavis JA, Looman J, Franco KA, Rojas B. Adverse childhood experiences and adult criminality: how long must we live before we possess our own lives? Perm J 2013;17(2):44.
[60] Widom CS. Long-term impact of childhood abuse and neglect on crime and violence. Clin Psychol Sci Pract 2017;24(2):186–202.
[61] Mersky JP, Janczewski CE, Topitzes J. Rethinking the measurement of adversity: moving toward second-generation research on adverse childhood experiences. Child Maltreat 2017;22(1):58–68.
[62] Armstrong GS, Cain CM, Wylie LE, Muftić LR, Bouffard LA. Risk factor profile of youth incarcerated for child to parent violence: a nationally representative sample. J Crim Just 2018;58:1–9.
[63] Papalia NL, Luebbers S, Ogloff JR, Cutajar M, Mullen PE. Exploring the longitudinal offending pathways of child sexual abuse victims: a preliminary analysis using latent variable modeling. Child Abuse Negl 2017;66:84–100.
[64] Salter D, McMillan D, Richards M, Talbot T, Hodges J, Bentovim A, et al. Development of sexually abusive behaviour in sexually victimised males: a longitudinal study. Lancet 2003;361(9356):471–6.
[65] Widom CS, Ames MA. Criminal consequences of childhood sexual victimization. Child Abuse Negl 1994;18(4):303–18.
[66] Widom CS, Czaja SJ, DuMont KA. Intergenerational transmission of child abuse and neglect: real or detection bias? Science 2015;347(6229):1480–5.
[67] Braga T, Gonçalves LC, Basto-Pereira M, Maia Â. Unraveling the link between maltreatment and juvenile antisocial behavior: a meta-analysis of prospective longitudinal studies. Aggress Violent Behav 2017;33:37–50.
[68] Baglivio MT, Epps N. The interrelatedness of adverse childhood experiences among high-risk juvenile offenders. Youth Violence Juvenile Justice 2016;14(3):179–98.
[69] Loeber R, Green SM, Keenan K, Lahey BB. Which boys will fare worse? Early predictors of the onset of conduct disorder in a six-year longitudinal study. J Am Acad Child Adolesc Psychiatry 1995;34(4):499–509.
[70] Loeber R, Burke JD, Lahey BB. What are adolescent antecedents to antisocial personality disorder? Crim Behav Ment Health 2002;12(1):24–36.
[71] Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, et al. Role of genotype in the cycle of violence in maltreated children. Science 2002;297(5582):851–4.
[72] Reif A, Rösler M, Freitag CM, Schneider M, Eujen A, Kissling C, et al. Nature and nurture predispose to violent behavior: serotonergic genes and adverse childhood environment. Neuropsychopharmacology 2007;32(11):2375–83.
[73] Weder N, Yang BZ, Douglas-Palumberi H, Massey J, Krystal JH, Gelernter J, et al. MAOA genotype, maltreatment, and aggressive behavior: the changing impact of genotype at varying levels of trauma. Biol Psychiatry 2009;65(5):417–24.