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ORIGINAL PAPER

Antisocial personality and risks of cause-specific mortality: results from the Epidemiologic Catchment Area study with 27 years of follow-up

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Abstract

Purpose Little is known about the effect of antisocial personality disorder (ASPD) on the risks of cause-specific mortality in the community. This study aimed to close this gap by evaluating if ASPD increases risks of cause-specific mortality in population-based residential and institutionalized samples with 27 years of follow-up.

Methods Data were collected in four metropolitan sites as part of the Epidemiologic Catchment Area (ECA) study during 1979–1983. Records were linked to the National Death Index through the end of 2007. Cox proportional hazards models adjusted for propensity weights and sample weights were fitted to estimate the effect of ASPD on the hazard of dying.

Results 420 respondents with ASPD (median survival age 71.0 years) and 15,367 without ASPD (median survival age 84.6 years) were included in this study. Those with ASPD were more likely to die from all causes (HR = 4.46; 95% CI = 2.44–8.16), suicide (HR = 2.81; 95% CI = 1.03–7.65), malignant neoplasms (HR = 4.09; 95% CI = 2.66–6.28), chronic lower respiratory disease (HR = 5.67; 95% CI = 2.92–11.0), and human immunodeficiency virus infection (HR = 8.07; 95% CI = 2.03–32.1), but not from accidents (HR = 0.58; 95% CI = 0.17–1.93) or heart disease (HR = 1.09; 95% CI = 0.43–2.76).

Conclusions Our findings demonstrate that antisocial personality disorder is a strong predictor of all-cause mortality, and cause-specific mortality. Early identification, treatment, and prevention of ASPD are important public mental health initiatives that could reduce premature mortality among this vulnerable population.

Keywords Antisocial personality disorder · Epidemiologic catchment area study · Mortality · Suicide · HIV · Personality disorders

Introduction

As defined by DSM-V criteria, antisocial personality disorder is a maladaptive pattern of irresponsible and antisocial behavior beginning in childhood or early adolescence and continuing into adulthood [1–3]. It is characterized by the onset of conduct disorder before the age of 15 years, and pervasive disregard for and violation of the rights of others thereafter [3]. The prevalence of ASPD is estimated to be 3.6% in the United States population [4], and 18–80% in incarcerated populations [5, 6]. Individuals with antisocial personality disorder have a greater likelihood of experiencing a host of adverse outcomes, including unemployment, divorce, imprisonment, and premature death [1, 2]. ASPD also is associated with co-morbid psychiatric disorders, including major depressive disorder, bipolar disorder, alcohol and substance use disorders [2, 7], as well as an increased risk of suicidal behaviors [8, 9].

Prior studies have established that ASPD is associated with premature death [10], but such studies often have been conducted with relatively small samples, relying on the readily identifiable inmates in correctional facilities [9, 10], and comparatively little is known about antisocial personality disorder in the community. Our study extends the prior analysis of the relationship between psychiatric disorders

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and all-cause mortality in the ECA resident sample drawn from the general population in four geographic areas with a 27-year follow-up period [11].

Personality disorders are associated with increased risk of suicide, accounting for 30–40% of all completed suicides [12]. ASPD has been shown to be associated with suicide attempts [8, 13]. Our study adds to prior research by evaluating ASPD as a risk factor for completed suicides in population-weighted residential and institutionalized samples.

Those with antisocial personality disorder are often thought of as perpetrators of violent crime, but little is known about their risk of unnatural death. Death by unnatural causes has been shown to be higher among those with mental illness [14] and those with a personality disorder in particular [15]. Our study evaluates the effect ASPD in particular on the risk of unnatural death, including accidental death.

Furthermore, antisocial disorders have been linked to risky sexual practices and sexually transmitted diseases [16, 17]. Several studies have shown that engaging in antisocial behavior in adolescence is associated with poorer health in adulthood, including cardiovascular problems, cancer, and acute respiratory problems [18, 19]. Our study is unique in evaluating the risk of death from conditions that have been linked to ASPD by prior studies, such as heart disease, malignant neoplasms, chronic lower respiratory disease, and human immunodeficiency virus infection (HIV). To our knowledge, this is the first study to evaluate how ASPD affects the risk of death from these diseases in a sample weighted to represent the United States population.

Methods

Sample

Data for the current study were collected as part of the Epidemiologic Catchment Area (ECA) study, which has been described in detail previously [20–22]. The ECA used multistage sampling to select representative samples from community mental health catchment center areas in five metropolitan study sites (New Haven, Connecticut; Baltimore, Maryland; St. Louis, Missouri; Durham, North Carolina; and Los Angeles, California), including both household residents and institutionalized individuals [22]. ECA sampling methods varied depending on the location and number of institutions serving a catchment area to derive institutionalized samples demographically comparable to the residential samples [22]. Sample weights were created to adjust results by age, race, and sex to the United States population as defined in the 1980 Census [22]. For all participants, trained lay interviewers administered the Diagnostic Interview Schedule (DIS) [23] at baseline, i.e., during the first Wave of the ECA (1979–1982). The DIS collected information on sociodemographic characteristics, medical history, health services use, and psychopathology.

Household residents ($n = 15{,}440$), prison/jail inmates ($n = 478$), residents of mental hospitals ($n = 288$) and nursing homes ($n = 905$) were included in the present analyses. Residents of boarding homes ($n = 92$), chronic hospitals ($n = 66$), and residential treatment centers ($n = 27$) have been excluded from the current analysis because they were collected only as a part of the St. Louis sample. We have included the prison/jail inmates in our sample because of the high prevalence of ASPD in this setting [5]. Selection of household and institution respondents has been described in detail elsewhere [22, 24]. Individuals institutionalized for less than a year were eligible for inclusion in the residential sample based on their address prior to entering the institution [25], while those institutionalized for more than a year were assigned the address of their respective institution [25].

Data from one site (Los Angeles, California) were not included ($n = 3534$) in the current analysis because record identifiers had not been retained, and it was not possible to ascertain vital status of the participants [20]. A total of 17,111 individuals were interviewed at baseline and included in the current analytic sample. Of these, 5372 were from the New Haven, Connecticut site, 4034 were from the Baltimore, Maryland, 3282 were from the St. Louis, Missouri, and 4423 were from the Durham, North Carolina.

Information for evaluating psychopathology was missing for 1319 individuals because they were either too impaired to respond or did not complete the interview in its entirety [20]. Participants with missing information were older (mean age = 68.8 years vs. 49.9 years, $p < 0.001$), and more likely to be white (77.6% vs. 71.8%, $p < 0.001$). In addition, those with missing ASPD diagnosis were more likely to have a high school diploma or GED (88.6% vs. 70.9%, $p < 0.001$), have low socioeconomic status (SES) (79.4% vs. 60.9%, $p < 0.001$), receive disability benefits (15.1% vs. 7.1%, $p < 0.001$), be residents of nursing homes (41.1% vs. 2.3%; $p < 0.001$) or mental hospitals(11.4% vs. 0.9%; $p < 0.001$), have drug dependence (14.1% vs. 5.5%, $p = 0.002$), have alcohol dependence (37.5% vs. 11.9%; $p < 0.001$), and have had at least one hospitalization in the 12 months preceding the baseline interview (27.3% vs. 15.6%, $p < 0.001$).

Three records were excluded because they were missing either month of birth, year of birth, month of death or year of death and, therefore, it was not possible to calculate the time they contributed to the analysis. Moreover, two records were excluded because their recorded date of death preceded their date of birth.

The final sample used for the analysis includes 15,787 individuals. Of these, 6868 died during the 27 years of follow-up. Data from the 2007 National Death Index [26] were linked to the ECA data set, using last name, first

name, sex, race, date of birth, social security number, last state of residence and father's surname [11]. Month and year of death, but not day, were included in the linked dataset to ensure participant confidentiality. Therefore, the day of death was specified as '15' for all deceased individuals for the purposes of this analysis.

Cause of death was assigned based on the International Classification of Diseases [27, 28]. Unnatural death was assigned for death from suicide (ICD-9 codes E950-E959 and ICD-10 codes U03, X60–X84, Y87.0) and accidents (ICD-9 codes E800-E869, E880-E929 and ICD-10 codes V01-X59, Y85-Y86). Natural causes of death were assigned for heart disease (ICD-9 codes 390–398, 402, 404–429 and ICD-10 codes I00-I09, I11, I13, I20-I52), malignant neoplasms (ICD-9 codes 140–208 and ICD-10 codes C00-C97), chronic lower respiratory disease (ICD-9 codes 490–494, 496 and ICD-10 codes J40-J47), and human immunodeficiency virus (ICD-9 codes 042–044 and ICD-10 codes B20-B24).

Measures

Antisocial personality disorder (ASPD) was assessed according to DSM-III criteria [29]. A diagnosis of ASPD was made if an individual had conduct disorder with onset prior to age 15 years, and a pattern of antisocial behavior manifested by at least four of the following since age of 18 years: inability to sustain consistent work behavior; inability to function as a responsible parent; failure to accept social norms with respect to lawful behavior; inability to maintain enduring attachment to a sexual partner; irritability and aggressiveness; failure to plan ahead, or impulsivity; disregard for the truth; and recklessness [23, 29]. In accordance with DSM-III criteria, a diagnosis of ASPD was not given if the antisocial behavior was due to either schizophrenia or manic episodes [29].

Baseline characteristics included in the analyses were site, sex, race, age, residency, socioeconomic status (SES), education, unemployment, disability, prior major depressive disorder episodes, drug dependence, alcohol dependence, bipolar disorder, and number of hospitalizations in the preceding 12 months, as ascertained during the initial interview in 1979–1983 [22]. Socioeconomic status was calculated by taking the arithmetic mean of the occupational status score, grade percentile, and household income percentile. SES and receipt of social benefits were recorded at baseline and did not necessarily reflect the lifetime SES or use of such benefits. The Diagnostic Interview Schedule (DIS) was used by lay interviewers to evaluate the presence of psychopathology, according to DIS-DSM-III criteria [23]. All psychiatric diagnoses were evaluated as lifetime at baseline.

Statistical analysis

Sample weights were applied while comparing baseline characteristics of respondents with and without ASPD. We used Pearson Chi-square tests for categorical variables and Wilcoxon rank sum tests for continuous variables. Individuals with and without ASPD differed significantly on all baseline characteristics except education level, unemployment, race, prior major depressive disorder episodes, and number of hospitalizations in the preceding 12 months. Propensity scoring was used to adjust for these differences [30]. The propensity of being in ASPD or non-ASPD groups was calculated from a survey-weighted logistic regression model with all of the baseline covariates. Respondents were weighted by the inverse probability of being in the ASPD vs. non-ASPD group to achieve a balance between baseline characteristics [30]. Propensity weights and survey weights were multiplied to create a composite weight that accounts for both survey and propensity weighting, a method described in detail previously [31]. Post-propensity weighting balance of baseline covariates was assessed using Chi-square tests.

Cox proportional hazard models with composite weights were fit to estimate the association between ASPD and all-cause mortality, as well as deaths due to suicide, accident, heart disease, malignant neoplasms, chronic lower respiratory disease, and HIV. Individuals contributed time to the analysis until their death or day of censoring for survivors. The assumption of proportional hazards was tested by introducing an interaction term for time and ASPD status into the model.

Competing risk models were used for cause-specific mortality to account for the competing risk of death from other causes [32]. Individuals who died of causes other than the one being investigated were censored and contributed time to the study until their death from the other cause.

Study entry for the survival analysis was the date of baseline interview. Age at the time of death or censoring was treated as a continuous variable and used as the time metric for the survival analysis. Because the youngest respondent in the study was 18 years old, we used years after age 18 as time metric in our analysis. Survival analysis was selected as a method of choice because death is a rare event and this type of analysis incorporates the time component into the analysis [33].

Individuals who did not die prior to December 31, 2007, were administratively censored. In addition, individuals whose ages appeared greater than 105 years ($n=15$) at the time of administrative censoring were censored at 105 years.

All statistical tests were two sided with an alpha level of 0.05 considered statistically significant. Data analysis was conducted using SPSS, version 22 and SAS (SAS Institute, Cary, North Carolina), version 9.4. The study was approved

by the Johns Hopkins Bloomberg School of Public Health's institutional review board.

Results

Comparison of baseline characteristics

Table 1 summarizes the baseline characteristics of the survey-weighted sample according to ASPD status. The ASPD group was younger (mean age = 33 years vs. 43 years; $p < 0.001$). In addition, compared to those without ASPD, those with ASPD were more likely to be male (83.2% vs. 45.2%; $p < 0.001$), incarcerated (2.7% vs. 0.2%; $p < 0.001$), low SES (63.7% vs. 50.7%; $p < 0.001$), have ever had drug dependence (36.6% vs. 4.6%; $p < 0.001$), alcohol dependence (71.8% vs. 11.3%; $p < 0.001$), or bipolar disorder (6.8% vs. 0.8%; $p < 0.001$). There was no statistical difference in the proportion of high school graduates (72.1% vs. 70.9; $p = 0.44$), unemployed (4.2% vs. 2.2%; $p = 0.11$), African American (21.4% vs. 22.5%; $p = 0.68$), hospitalized in the preceding 12 months (17.6% vs. 11.6%; $p = 0.07$), or those with a prior major depressive disorder episodes (6.3% vs. 3.5%; $p = 0.14$).

Following complex weighting that accounted for survey and propensity weights, covariate balance between ASPD and non-ASPD groups was assessed. Groups were well balanced as illustrated in Table 1 ($p > 0.05$ for all covariates).

All-cause mortality

There were 6868 (43.5%) deaths in the sample by the time of administrative censoring on December 31, 2007. Of 420 individuals diagnosed with ASPD, 119 (28.3%) died during the follow-up period. Propensity- and survey-weighted Kaplan–Meier curves for all-cause mortality in those with and without ASPD are illustrated in Fig. 1. The median survival time for those without ASPD was 83.5 years as estimated from the Kaplan–Meier curve. In contrast, the median survival time for individuals with ASPD was significantly shorter at 71.0 years (log-rank test $p < 0.0001$).

The hypothesis of higher mortality among individuals with ASPD vs. those without ASPD was tested using Cox proportional hazards model. There was no evidence against proportional hazards, as the interaction term for time to death and ASPD status was not significant (p value = 0.15).

As shown in Table 2, the hazard ratio of dying from any cause, comparing individuals with ASPD to those with no ASPD, was 3.59 (95% CI = 2.68–4.79) in the crude survey-weighted model with no other covariates. This relationship remained significant after propensity and survey weighting to account for baseline characteristics (HR = 4.46; 95% CI = 2.44–8.16).

Mortality due to unnatural causes

There were 25 completed suicides and 210 accidental deaths in the sample by the time of administrative censoring on December 31, 2007. Of 420 individuals with the diagnosis of ASPD, 5 committed suicide and 9 died from accidents.

The proportional hazard assumption for ASPD was met in all models. In the survey-weighted models with competing risks with no other covariates, the hazard ratio of dying comparing individuals with ASPD to those with no ASPD was 15.26 (95% CI = 5.05–46.19) for death by suicide and 3.45 (95% CI = 1.26–9.44) for accidental death. After survey weighting and propensity adjusting, the relationship decreased in magnitude but remained significant for suicide (HR = 2.81; 95% CI = 1.03–7.65). We found no evidence for the association of ASPD and increased risk of accidental death (HR = 0.58; 95% CI = 0.17–1.93).

Mortality due to natural causes

There were 2925 deaths from heart disease, 1411 from malignant neoplasms, 514 from chronic lower respiratory disease, and 43 as a result of HIV infection during the 27 years of follow-up. Of 420 individuals with the diagnosis of ASPD, 27 died from heart disease, 27 died as a result of malignant neoplasms, 9 from chronic lower respiratory disease, and 6 due to HIV.

In the unadjusted survey-weighted models, ASPD increased the risk of dying from heart disease (HR = 2.81; 95% CI = 2.09–3.77), malignant neoplasms (HR = 3.78; 95% CI = 2.24–6.37), chronic lower respiratory disease (HR = 4.28; 95% CI = 1.78–10.30), and HIV (HR = 6.34; 95% CI = 2.14–18.79). After survey weighting and propensity adjustment, these relationships remained significant for malignant neoplasms (HR = 4.09; 95% CI = 2.66–6.28), chronic lower respiratory disease (HR = 5.67; 95% CI = 2.92-11.0), and HIV (HR = 8.07; 95% CI = 2.03–32.1), but not heart disease (HR = 1.09; 95% CI = 0.43–2.76).

Discussion

These analyses were undertaken to determine if ASPD increases the risks of all-cause mortality and cause-specific mortality in the community. Our findings suggest that ASPD is associated with premature death from natural and unnatural causes. The study sample consisted of both household and institutionalized residents, adjusted by age, race, and sex to the United States population as defined in the 1980 Census. Examining only household residents would be inadequate for understanding the epidemiology of ASPD since a substantial proportion of such individuals are incarcerated [5, 6]. Indeed, in our weighted sample, 2.7% of individuals

Table 1 Baseline characteristics of respondents with and without ASPD in the four Epidemiologic Catchment Area sites interviewed between 1979 and 1983, before and after propensity score and survey weighting

	Unweighted			Survey weighted			Propensity and survey weighted		
	ASPD	No ASPD		ASPD	No ASPD		ASPD	No ASPD	
	n (%)	*n* (%)	*p*	*n*(%)	*n* (%)	*p*	%	%	*p*
Site									
New Haven	67 (16.0)	4982 (32.4)	< 0.001	5891 (26.5)	287,945 (32.1)	< 0.001	45.4	33.0	0.27
Baltimore	124 (29.5)	3406 (22.2)		4028 (18.1)	158,599 (17.7)		19.8	20.5	
St. Louis	154 (36.7)	2950 (19.2)		9293 (41.8)	266,270 (29.7)		14.9	24.6	
Durham	75 (17.9)	4029 (26.2)		3034 (13.6)	183,254 (20.5)		20.0	21.9	
Sex									
Male	351 (83.6)	6169 (40.1)	< 0.001	18,501 (83.2)	404,756 (45.2)	< 0.001	54.0	44.5	0.36
Female	69 (16.4)	9198 (59.9)		3745 (16.8)	491,312 (54.8)		46.0	55.5	
Race									
Non-black	249 (59.3)	11,088 (72.2)	< 0.001	17,481 (78.6)	694,457 (77.5)	0.7	70.2	76.0	0.38
Black	171 (40.7)	4279 (27.8)		4765 (21.4)	201,611 (22.5)		29.8	24.0	
Age at first interview (years)									
Mean (SD)	32.3 (13)	50.3 (21)	< 0.001	33.3 (11)	43.4 (18)	< 0.001	41.9 (16)	49.7 (20)	0.06
Residency at baseline									
Household	272 (64.8)	14,559 (94.7)	< 0.001	21,618 (97.2)	891,725 (99.5)	< 0.001	99.4	99.4	0.90
Nursing home	3 (0.7)	360 (2.3)		6 (0.03)	2637 (0.3)		0.3	0.3	
Prison or jail	133 (31.7)	322 (2.1)		589 (2.7)	1382 (0.2)		0.4	0.3	
Mental hospital	12 (2.9)	126 (0.8)		33 (0.2)	324 (0.04)		0.02	0.05	
SES									
Non-low SES	101 (24.1)	6046 (39.5)	< 0.001	8064 (36.4)	440,775 (49.3)	< 0.001	55.4	48.1	0.42
Low SES	318 (75.9)	9272 (60.5)		14,117 (63.7)	452,565 (50.7)		44.6	51.9	
Education									
Less than high school	303 (72.1)	10,895 (70.9)	0.6	6587 (29.6)	298,631 (33.3)	0.3	22.3	32.6	0.13
High school diploma/GED	117 (27.9)	4472 (29.1)		15,660 (70.4)	597,437 (66.7)		77.7	67.4	
Unemployment									
No	401 (96.2)	14,934 (98.1)	0.04	21,169 (95.8)	869,789 (97.8)	0.1	98.4	97.8	0.55
Yes	16 (3.8)	292 (1.9)		930 (4.2)	19,651 (2.2)		1.6	2.2	
Disability									
No	378 (90.9)	14,144 (92.9)	0.1	20,040 (90.7)	847,636 (95.1)	0.03	91.4	94.6	0.26
Yes	38 (9.1)	1073 (7.1)		2053 (9.3)	43,914 (4.9)		8.6	5.4	
Major depressive disorder									
No episodes	392 (93.8)	14,835 (96.7)	0.02	20,761 (93.7)	863,858 (96.5)	0.1	97.3	96.3	0.44
One or more episodes	26 (6.2)	513 (3.3)		1403 (6.3)	31,744 (3.5)		2.7	3.7	
Drug dependence									
Never	236 (56.2)	14,663 (95.6)	< 0.001	14,106 (63.4)	853,803 (95.4)	< 0.001	88.8	94.6	0.05
Ever	184 (43.8)	681 (4.4)		8140 (36.6)	40,943 (4.6)		11.2	5.4	
Alcohol dependence									
Never	140 (33.7)	13,716 (89.6)	< 0.001	6249 (28.2)	793,705 (88.8)	< 0.001	82.7	87.5	0.14
Ever	276 (66.3)	1591 (10.4)		15,926 (71.8)	100,618 (11.3)		17.3	12.5	
Bipolar									
No	390 (93.1)	15,243 (99.3)	< 0.001	20,682 (93.2)	888,861 (99.3)	< 0.001	99.3	99.1	0.41
Yes	29 (6.9)	111 (0.7)		1499 (6.8)	6673 (0.8)		0.7	0.9	
Number of hospitalizations in the past 12 months									
None	339 (80.7)	12,984 (84.5)	0.05	18,330 (82.4)	780,513 (87.1)	0.07	88.4	86.7	0.66
One or more	81 (19.3)	2383 (15.5)		3917 (17.6)	115,555 (12.9)		11.6	13.3	

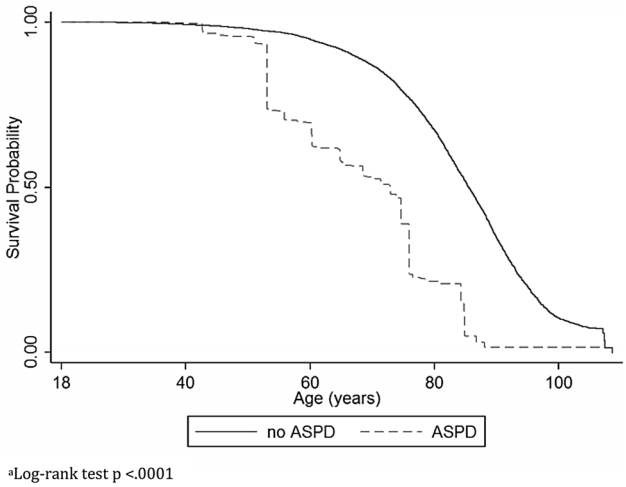

Fig. 1 Propensity- and sample weight-adjusted Kaplan–Meier all-cause survival estimates[a] for those with and without ASPD. [a]Log-rank test $p < 0.0001$

with ASPD were incarcerated at the time of the baseline interview.

The findings of the current study are in agreement with previous studies on mortality among individuals with ASPD [1]. ASPD is predictive of early death from all causes, as well as death by suicide. The hazard ratio of dying is four and a half times higher in individuals with ASPD compared to those without ASPD (HR = 4.46, $p < 0.0001$), after propensity weighting to adjust for sex, race, age, residency, site, SES, education, unemployment, disability, prior MDD episodes, drug dependence, alcohol dependence, bipolar disorder, and hospitalization in the past year. Moreover, our findings show a striking 13-year difference in the median survival time between those with and without ASPD. The hazard ratio of dying by suicide is almost three times as high comparing individuals with ASPD to those without ASPD (HR = 2.81, $p = 0.04$).

Suicide is among the top ten leading causes of death for adults in the United States [34], and the second leading cause of death for adults 25–34 years of age [34]. The rate of completed suicides is significantly greater among males, accounting for 78% of suicides in the United States [34]. Suicide prevention among young men is an important public health goal. Individuals with ASPD are predominantly male, and score high on ratings of aggression and impulsivity, traits that may contribute to high rates of attempted and completed suicide among them [8, 35]. Our findings confirm that individuals with ASPD are at significantly higher risk for completed suicide than those without this disorder. It is important to devise public health interventions that address increased suicidal risk in these vulnerable individuals.

We employed inverse probability of treatment weighting to approximate randomization on known risk factors of completed suicides such as sex [34], race [34], major depression [36], drug dependence [37], alcohol dependence [37], and bipolar disorder [13]. However, it was not possible to control for borderline personality disorder, a known risk factor for suicidal behavior [12] as well as other personality disorders because they were not evaluated during the first wave of the ECA. Borderline personality disorder in particular is known to be co-morbid with ASPD, especially among men [38]. Hence, inability to control for it is a serious limitation of our study.

To our knowledge, this is the first study to demonstrate a higher risk of death from malignant neoplasms and chronic lower respiratory disease among those with ASPD. The hazard ratio of dying from malignant neoplasms and chronic lower respiratory disease is four and six times as high in those with ASPD than those with no

Table 2 Competing risk analysis of all-cause and cause-specific mortality risk in study participants with vs. without ASPD

	Deaths	Survey weighted		Survey-weighted and propensity adjusted[a]	
	n	Hazard ratio (95% CI)	*p*	Hazard ratio (95% CI)	*p*
All-cause mortality	6868	3.59 (2.68–4.79)	< 0.0001	4.46 (2.44–8.16)	< 0.0001
Cause-specific mortality					
Heart disease	2925	2.81 (1.63–4.85)	< 0.0001	1.09 (0.43–2.76)	0.9
Malignant neoplasms	1411	3.78 (2.24–6.37)	< 0.0001	4.09 (2.66–6.28)	< 0.0001
Chronic lower respiratory disease	514	4.28 (1.78–10.30)	0.001	5.67 (2.92-11.0)	< 0.0001
Suicide	25	15.26 (5.05–46.19)	< 0.0001	2.81 (1.03–7.65)	0.04
Accidental death	210	3.45 (1.26–9.44)	< 0.0001	0.58 (0.17–1.93)	0.6
HIV	43	6.34 (2.14–18.79)	< 0.0001	8.07 (2.03–32.1)	0.003

[a]Hazard ratios adjusted for sample and propensity weights, which were calculated to balance the following baseline characteristics: site, sex, race, age, residency, socioeconomic status, education, unemployment, disability, prior major depressive disorder episodes, drug dependence, alcohol dependence, bipolar disorder, and number of hospitalizations in the 12 preceding months

ASPD, respectively. This is suggestive of lack of adequate access to treatment among this population.

Previous studies demonstrated that ASPD is associated with risky behaviors associated with acquiring HIV, such as high prevalence of needle sharing, multiple sex partners, and drug use [39]. In our sample, 36.6% of respondents with ASPD had drug dependence and a striking 71.8% had alcohol dependence at the time of the initial interview. To our knowledge, our findings are the first to demonstrate an eightfold increase in the risk of death due to HIV among those with ASPD in the community. Individuals with ASPD are at a higher risk of contracting HIV [39] and, according to our study, of dying from it. Treatment initiatives need to be devised to target this population, provide access to treat their drug and alcohol dependence and to ensure adequate management of HIV for those already infected.

Previous studies, including the Baltimore ECA study by Lee et al. [40], showed that Cluster B personality disorders are predictive of incident cardiovascular disease (OR = 2.67; 95% CI = 1.02–6.99), but also showed only a small increased in the risk of death from CVD among those with ASPD (OR = 1.09; 95% CI = 1.01–1.19). Our findings indicate no increase in the hazard of dying from heart-related problems among those with ASPD (HR = 1.09; 95% CI = 0.43–2.76). Interestingly, the models in the Baltimore ECA study were adjusted for smoking, as it was available in the subsequent waves of the ECA data. Unfortunately, smoking was not available in the wave one of multi-site ECA data collection. Hence, it was not possible to adjust for smoking in our study.

The current study has a number of strengths and limitations. Though we balanced important baseline characteristics that are associated with mortality by employing inverse probability propensity weighting, there may be other important potential confounding variables that were not evaluated. These variables that were either unmeasured or not utilized in this analysis could potentially bias the relationship between ASPD and early death. Additionally, as we utilized the 2007 National Death Registry to ascertain death causes, and did not have access to the death certificates, our death search may be incomplete.

As previously mentioned, we were unable to adjust for smoking because it was not collected at all sites during the first wave of the ECA. Smoking is a known risk factor for heart disease [41], lower respiratory disease [41], and cancer [42]. Moreover, individuals with ASPD have been shown to have higher rates of smoking [43]. By adjusting for alcohol and drug dependence in our study, conditions that have been shown to co-occur with tobacco use [44], we mitigated for some of the confounding effects of smoking. Still, unavailability of smoking is a limitation of our study and could account for some of the increased risks we have observed for death from malignant neoplasms, chronic lower respiratory disease, and cancer. Since Los Angeles participants were not included in our sample, the results of the study may not be generalizable to the Hispanic population.

We have utilized competing risk analyses, censoring individuals who died from other causes before the event of interest. This approach assumes that all causes of death are independent of each other [45]. However, an individual who died early could have developed the outcome of interest at a later date. Since the ASPD group had a lower life expectancy, the cause-specific risks are underestimated for this group. Hence, our estimates are conservative.

Despite these limitations, the current study is the first of its kind, because it includes a broad-based household and institutional sample weighted to represent the US population, with over 27 years of follow-up, and it examines the risk of cause-specific mortality. Individuals with ASPD are often thought of as untreatable [46]. Our findings highlight the importance of prevention, early identification, and treatment in reducing mortality associated with ASPD. Conduct disorder is the precursor to ASPD, and interventions that target conduct disorder traits early in the child's development have been shown to be most effective for the prevention of ASPD later in life [47]. Parenting interventions [48] and school interventions [49, 50] have shown to be effective in reducing conduct problems in children. Despite the challenges that arise, antisocial traits can be reduced, especially if intensive treatment is introduced early [47]. Timely interventions could improve long-term health trajectories and reduce early mortality and rates of death from suicide, malignant neoplasms, chronic respiratory disease, and HIV among this vulnerable population.

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Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

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