

Comorbidity Among Depression, Conduct Disorder, and Drug Use From Adolescence to Young Adulthood: Examining the Role of Violence Exposures

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We assessed relations among depression, conduct disorder, and drug use from adolescence to young adulthood, and evaluated whether exposure to violence contributed to disorder co-occurrence. We used data from the Project on Human Development in Chicago Neighborhoods. Respondents were 12–15 years old in 1995–1997 (N = 1,517), and were reinterviewed in 1997–2000 (n = 1,315), and 2000–2002 (n = 1,210). We examined exposure to violence at ages 12–15 and 14–17, and depression, conduct disorder, and drug use at ages 14–17 and 17–20. Multivariate transition models revealed an association between prior conduct disorder and drug use, as well as a relationship between prior depression and conduct disorder. Adolescent exposure to violence was associated with higher odds of conduct disorder and drug use but not depression. Comorbid relations between conduct disorder and drug use were independent of prior exposure to violence. Although preventing adolescent exposure to violence may reduce the risk of conduct disorder and drug use by young adulthood, future research needs to investigate alternative determinants of sequential comorbidity among depression, conduct disorder, and drug use in adolescence and young adulthood.

Psychiatric comorbidity is the presence, simultaneously or in sequence, of two or more disorders in an individual within a certain period (Angold, Costello, & Erkanli, 1999). It is associated with worse prognosis, more severe symptoms, and lower social competence (Schuckit, 2006). Adolescence is a particularly important time to examine comorbidity, as many forms of psychopathology and risk behaviors, including delinquency, drug use, and depression, either begin or peak at this stage.

Reciprocal relations may exist among depression, conduct disorder, and drug use. First, adolescents may self-medicate depression through drug use, and drug use may increase vulnerability to depression by exerting neurophysiological or behavioral changes on

the user: 9.0–47.9% of persons exhibit comorbid depression and drug use (O’Neil, Conner, & Kendall, 2011). Second, conduct disorder and drug use may follow a common developmental pattern of externalizing behavior: an externalizing factor accounts for 79% and 95% of conduct disorder and drug use, respectively (Measelle, Slice, & Hogansen, 2006). Third, a reciprocal association may exist between conduct disorder and depression (Johnson, Cohen, Kasen, & Brook, 2005), mediated by disrupted interpersonal functioning, school failure, and social rejection by prosocial peers: 22.7–83.3% of those with depression may also meet criteria for conduct disorder, while 8.5–45.4% of those with conduct disorder also met criteria for depression (Angold & Costello, 1993). The relatively high rates of comorbidity among depression, conduct disorder, and drug use, and the scarcity of longitudinal, population-based studies on the relations among the three symptom domains underscore the importance of research on comorbidity among depression, conduct disorder, and drug use.

Exposure to violence in adolescence may be an important influence on comorbidity among depression, conduct disorder, and drug use (Moffitt et al., 2007; Murray & Farrington, 2008). Parent–child abuse and community violence are independently associated with higher levels of depression, conduct disorder, and

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drug use (Fergusson, Boden, & Horwood, 2008; Johnson et al., 2002; Margolin, Vickerman, Oliver, & Gordis, 2010; Moffitt et al., 2007). Hence, by separately increasing the risk for depression, conduct disorder, and drug use by young adulthood, exposure to parent-child and community violence may generate a disproportionate concentration of co-occurring disorders among victims of violence.

We extend the literature on violence and comorbid psychopathology in several ways. First, we consider the reciprocal relations among depression, conduct disorder, and drug use in adolescence and young adulthood. By jointly accounting for concurrent clustering among the three symptom domains, we reduce bias in the estimation of sequential relations between pairs of disorders. In other words, we account for the fraction of the observed association between a pair of symptom domains (e.g., depression and conduct disorder) that is actually due to the association between the two domains and a third, previously unmeasured symptom domain (e.g., drug use). Second, we examine the specificity of the association between exposure to family and community violence and the three symptom domains, while accounting for symptom domain co-occurrence. Past research has usually examined the relationship between violence and single symptom domains, which may similarly inflate the strength of associations due to comorbidity. Third, we examine whether exposure to violence functions as a common determinant, and thus partly explains, the sequential associations between symptom domains. Based on prior research, we hypothesized that (a) sequential comorbid relations would exist among depression, conduct disorder, and drug use, so that a history of comorbidity would increase the risk of onset and persistence of each symptom domain; (b) exposure to violence in early and late adolescence would increase the risk of depression, conduct disorder, and drug use; and (c) these violent exposures would partly explain the relations among the three symptom domains.

METHOD

Participants and Procedures

This study used data from the Project on Human Development in Chicago Neighborhoods Longitudinal Cohort Study. Participants were selected using a three-stage stratified sampling design, where 80 neighborhood clusters were randomly selected and stratified by racial/ethnic mix and socioeconomic status, and participants were randomly and proportionately selected from households within the selected neighborhood clusters. The study sample is representative of the Chicago area with respect to racial/ethnic and socioeconomic status (SES) distribution at the neighborhood level. The participation rate was 75%. Three face-to-face in-home interviews were conducted at approximately 24-month intervals (Wave 1: 1995–1997; Wave 2: 1997–2000; Wave 3: 2000–2002).

Our study baseline sample included 1,517 adolescents (Wave 1; 75% average response rate); Wave 2 included 87% of the baseline

sample; and Wave 3 included 80%. The sample included two cohorts: respondents were aged either 12 or 15 years at Wave 1. The institutional review board of the University of Michigan School of Public Health approved all protocols.

Twenty percent of participants were lost to follow-up. Individuals who did not participate in Wave 3 were more likely than those who did to have met criteria for conduct disorder at Wave 1 (22% of those who did not participate in Wave 3 vs. 17% of those who did). There was also differential attrition by race/ethnicity and family history of mental health or drug problems: Blacks were less likely to participate in Wave 2 (32% of respondents vs. 47% of nonrespondents) whereas individuals with a family mental health or drug problem history were more likely to participate in Wave 2 (59% of respondents vs. 51% of nonrespondents).

Measures

Youth (herein referred to as “subjects”) were the respondents in all waves to the measures of depression and drug use. Primary caregivers reported on conduct disorder in Wave 1 and subjects reported on this symptom domain in Waves 2–3.

Depression was measured with the Major Depressive Disorder instrument at Waves 2 and 3. Subjects were classified as meeting criteria for major depression if they endorsed depressed mood or anhedonia for reasons other than bereavement, endorsed at least five out of nine symptoms, and expressed at least one sign of impairment. The instrument was adapted from the Depression module of the Diagnostic Interview Schedule for Children (DISC 4; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). The DISC 4 Depression module has adequate test-retest reliability ($K = .92$; Shaffer et al., 2000). Respondents were classified as meeting criteria for depression at Wave 2 if they had ever, since age 5, experienced the requisite symptoms and impairment; at Wave 3, they met criteria for depression if they had experienced the symptoms and impairment in the past year.

Primary caregivers reported on conduct disorder with the aggression (Cronbach's $\alpha = .87$) and delinquency subscales (Cronbach's $\alpha = .61$) of the Child Behavior Checklist (CBCL; Achenbach, 1991a) at Wave 1, and subjects reported on conduct disorder with the Youth Self-Report (YSR; Achenbach, 1991b) instrument at Wave 2 and the Young Adult Self-Report (YASR; Achenbach, 1997) at Wave 3. At Wave 2, subjects were considered “positive” for conduct disorder if they reported borderline aggression (score ≥ 10) or borderline delinquent behavior (score ≥ 3) in Waves 1 or 2 (Achenbach, 1991a). At Wave 3, participants met criteria for conduct disorder if they reported borderline aggression (score ≥ 10 for the 12-year-old cohort or ≥ 9 for the 15-year-old cohort) or delinquent behavior (score ≥ 6 for the 12-year-old cohort or ≥ 5 for the 15-year-old cohort) in the past 6 months. These items were rated by clinicians as reflecting an underlying construct of conduct disorder (Achenbach, Dumenci, & Rescorla, 2001, 2003).

Subjects were asked about past-year illicit drug use through the Substance Use Interview in Waves 2 and 3. The interview was adapted from the National Household Survey on Drug Abuse (National Institute of Drug Abuse, 1991) and recorded the subject's reported quantity and frequency of use of various illicit substances, including marijuana, cocaine, crack cocaine, inhalants, hallucinogens, heroin, barbiturates, tranquilizers, amphetamines, steroids, and intravenous drugs. Given the low frequency of any illicit drug use in Wave 2, it was measured as reports of any drug use up to Wave 2; in Wave 3 it was measured as any past-year use. Test-retest reliability of NSDUH past-year drug use questions is high ($k = .8-.9$; Substance Abuse and Mental Health Services Administration, 2010).

Punishment and maltreatment by their primary caregivers at ages 12–15 years was assessed at Wave 1 using a subset of items from the Conflict Tactics Scale 1985 Resurvey Physical Assault subscale (CTSR-PA; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). Primary caregivers reported the frequency of seven behaviors towards the adolescent study participant in the past year. If the primary caregiver reported throwing something at the child, pushing or grabbing the child, or slapping or spanking the child at least once in the past year, the child was considered to have experienced parent–child harsh physical punishment; this corresponds to the Minor Assault subscale of the CTSR-PA. If the primary caregiver reported kicking, biting, or hitting the child with a fist; hitting the child with something else; beating the child up; or burning or scalding the child at least once in the past year, the child was considered to have experienced parent–child maltreatment; this corresponds to the Severe Assault subscale of the CTSR-PA, with the omission of two items about threatening or using a knife or gun. These items were dropped after a pretest revealed no endorsement of these behaviors, as well as concern that their inclusion would offend participants in the Chicago study (Molnar, Buka, Brennan, Holton, & Earls, 2003). In the sample presented here, the revised CTSR-PA has adequate reliability, Cronbach's $\alpha = .65$ and construct validity (Molnar et al., 2003).

Past-year violent victimization and witnessing violence at ages 14–17 were reported at Wave 2. To assess victimization, subjects were asked whether they had been attacked with a weapon, beaten up, chased, shot at, sexually assaulted, or threatened with serious harm in the past year. To assess witnessing, subjects were asked whether they had seen someone killed, attacked with a weapon, beaten up, chased, shot, shot at, or threatened with serious harm. These measures come from My Exposure to Violence, an instrument developed by the study (Selner-O'Hagan, Kindlon, Buka, Raudenbush, & Earls, 1998). In the study sample, psychometric study of the scale of violent victimization showed good item fit, Cronbach's $\alpha = .83$, and construct validity. The witnessing scale also showed good item fit, Cronbach's $\alpha = .73$, and construct validity (Brennan, Molnar, & Earls, 2007).

Covariates considered at Wave 1 included sex, age at baseline (i.e., membership in the 12- or 15-year-old cohort), race/ethnicity,

SES, attention deficit symptoms, impulsivity, and family history of mental health and/or drug use problems. Dichotomous variables were coded as 0/1. Socioeconomic status was measured as the first principal component of parental education, parental occupation, and household income. Attention deficit symptoms were reported by the primary caregiver using the CBCL; children met criteria for attention deficit if they reached the borderline threshold for attention deficit. Impulsivity was reported by the primary caregiver with the Impulsivity subscale in the Emotionality, Activity, Sociability, and Impulsivity Temperament Survey (Earls, Brooks-Gunn, Raudenbush, & Sampson, 2005). Family history of mental health, drug, and alcohol problems was assessed at Wave 1 by asking the primary caregiver whether any family members ever had drinking problems, drug use problems, depression, problems with nerves or a nervous breakdown, talked to a doctor or counselor about—or was hospitalized for—emotional problems or problems with drugs or alcohol, attempted or committed suicide.

Data Analysis

We first assessed the relations between violence exposures at Waves 1 and 2 and the odds of each symptom domain at Wave 3 by estimating a series of multivariate generalized estimating equation (GEE) models with a logit link, including two-way interactions between specific violence exposures and symptom domains (Models 1–5). By simultaneously estimating the three symptom domains as separate outcomes, we could compare the effect of different violence exposures on depression, conduct disorder, and drug use, and establish whether violence exposures could be a plausible shared risk factor for depression, conduct disorder, and drug use. Within these models, we used pairwise odds ratios to model the cross-sectional associations between pairs of symptom domains at Wave 3. Details on model equations and sample SAS code to replicate them have been published (Cerdá, Sánchez, Galea, Tracy, & Buka, 2008). We first estimated separate models for each violence exposure separately to address multicollinearity concerns among measures of violence, and then estimated one model including all violence exposures.

Next, we assessed sequential comorbidity among the three symptom domains from Wave 2 to 3. In this multivariate GEE model, the outcome was a binary variable taking the value 1 if individual j reported the symptom domains $r_3 = 1, \dots, R_3$ at Wave 3. For each of the three symptom domains, we estimated a marginal model for the probability of reporting domain r_3 at Wave 3, given symptom domains reported at Wave 2. This model included parameters (two-way interactions between each of the symptom domains at Wave 2 and the domains at Wave 3) that represented the effect on the log odds of the probability of reporting symptom domain r_3 at Wave 3, due to reporting symptom domain r_2 at Wave 2. Details on model estimation can be found in (Cerdá et al., 2008). These models also accounted for the cross-sectional association among symptom domains at Wave 3 through

pairwise odds ratios. This type of model allows us to compare how the prevalence of, for example, conduct disorder up to Wave 2 influenced the probability of onset and persistence of depression and drug use at Wave 3, after accounting for the concurrent relations among depression, conduct disorder, and drug use at Wave 3. By using a multivariate structure where the three symptom domains are simultaneously estimated as outcomes and we include two-way interactions with the three symptom domains at Wave 2, we could also assess whether reciprocal relations existed between domains. For example, we could simultaneously assess the influence of conduct disorder by Wave 2 on drug use onset at Wave 3, and the influence of drug use by Wave 2 on conduct disorder onset at Wave 3.

We estimated a model of sequential comorbidity between depression, conduct disorder, and drug use from Waves 2–3. In Model 6, we added the confounders specified above; in Model 7, we added all measures of violent exposures at Waves 1–2 as separate predictors, to test whether the magnitude of the associations among depression, conduct disorder, and drug use detected in Model 6 decreased substantially and became nonsignificant once we accounted for prior exposure to violence. Results are presented in Figures 1–4 to illustrate those relations that are significant at the $p < .05$ level (straight lines). The numbers shown above the lines represent probabilities of onset and persistence for a particular symptom domain, conditional on the presence of the same or another symptom domain by Wave 2. Probabilities were calculated from coefficients denoting the log odds of the probability of reporting symptom domain r_3 at Wave 3, due to reporting symptom domain r_2 at Wave 2.

Missing values for any of the covariates were imputed using multiple imputation (Ragunathan, 2004). We used the PROC MIANALYZE procedure in SAS, Version 8, to combine the model estimates from five imputed datasets. Parameter estimates were averaged over the set of analyses, while standard errors were computed using the average of the squared standard errors over the set of analyses and the between-analysis parameter estimate variation (SAS Institute, 2007).

RESULTS

Table 1 presents the characteristics of the study participants across the three study waves. The 12- and 15-year-old cohorts were, respectively, an average of 12.10 and 15.20 years of age at baseline, 14.20 and 17.30 at Wave 2, and 16.80 and 19.80 years of age at Wave 3. The prevalence of violent exposures varied considerably by severity of the event: 59% had received parent–child harsh physical punishment, 33% had received parent–child maltreatment, 84% had witnessed an event, and 35% had been victimized in the past year.

Table 2 presents the conditional onset and persistence probabilities for depression, conduct disorder, and drug use at Wave 3. Depression and conduct disorder exhibited opposite developmental changes. Whereas the depression prevalence doubled and in-

Table 1. Respondent Psychiatric Symptoms, Traumatic Life Events, and Sociodemographic Characteristics, Cohorts 12 and 15, by Wave of Measurement

Variable	Wave 1	Wave 2	Wave 3
	($N = 1,517$) %	($N = 1,315$) %	($N = 1,210$) %
Conduct disorder		45.3	21.2
Substance use		30.0	30.4
Depression		16.7	29.2
Witnessed violence, past year		84.2	
Violent victimization, past year		34.8	
Parent–child harsh physical punishment, past year	59.1		
Parent–child maltreatment, past year	33.4		
Gender			
Male	49.1		
Female	50.9		
Cohort			
12	54.1		
15	45.9		
Race/ethnicity			
Black Non-Hispanic	34.0		
White Non-Hispanic	13.9		
Hispanic	40.4		
Other	11.7		
Attention deficit symptoms (% classified as borderline)	12.3		
Percent of participants reporting any family member with mental health or drug problems	57.4		

Note. Waves refer to the three times of assessment in the study: baseline and the first and second follow-up interviews.

creased from the lowest to the highest prevalent symptom domain between Waves 2 and 3, conduct disorder prevalence decreased by half and moved from the highest to the lowest symptom domain in the same period. The prevalence of drug use remained stable across the study period.

Figures 1–4 present the onset and persistence probabilities with sex, age at baseline, race/ethnicity, socioeconomic status,

Table 2. Prevalence, Onset, and Persistence Probabilities of Symptom Domains at Ages 17–20 (Wave 3), Conditional on Prevalence of Symptom Domains through Ages 14–17 (as Assessed in Waves 1 and 2)

Variable	Period			
	Through ages 14–17 Prevalence (%)	17–20 Years of age		
		Prevalence (%)	Onset ^a (%)	Persistence ^b (%)
Depression	16.7	29.2	27.0	40.3
Conduct disorder	45.3	21.2	14.1	29.8
Substance use	30.0	28.3	19.9	54.5

Note. Waves refer to the three times of assessment in the study: baseline and the first and second follow-up interviews. Sample size is 1,517 in Wave 1, 1,315 in Wave 2, and 1,210 in Wave 3.

^aPercentage of respondents who did not meet criteria for the behavior in their lifetime prior to Wave 3 and who met criteria at Wave 3. ^bPercentage of respondents who met criteria for the symptom domain in their lifetime prior to Wave 3 and who continued to meet criteria at Wave 3.

attention deficit symptoms, impulsivity, and family history of mental health and/or drug use problems in the models. (Figures 1 and 2). Conduct disorder was associated with a higher probability of drug use onset and persistence. Prior depression was associated with a higher probability of conduct disorder onset and persistence. Finally, depression, conduct disorder, and drug use had moderate to high persistence probabilities.

Table 3 presents the relationship of specific violence exposures with the odds of depression, conduct disorder, and drug use. The first set of estimates was obtained from separate models for each violence exposure (Models 1–4); the second set was obtained from one model including all violence exposures (Model 5). Parent-child harsh physical punishment, violent victimization, and witnessing were significantly associated with conduct disorder and drug use. The direction of the associations between single types of violence and conduct disorder or drug use persisted, although the magnitude decreased, once we accounted for the co-occurrence of multiple types of violence.

Figures 3 and 4 present the associations between Wave 2 and Wave 3 symptom domains, jointly adjusting for exposure to the four types of violence exposures. Although the association between prior depression and conduct disorder onset/persistence became marginally significant once we controlled for exposure to violence in early and late adolescence, the magnitude of these associations did not change.

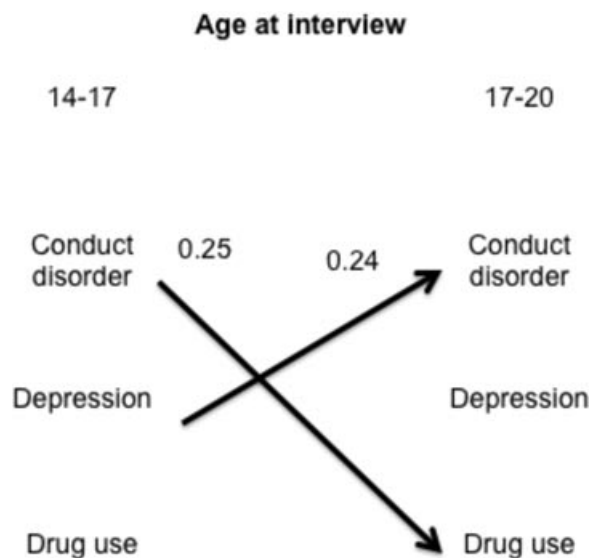


Figure 1. Probability of symptom domain onset conditional on the presence of two other domains at prior wave and controlling for sex, age at baseline, race/ethnicity, socioeconomic status, attention deficit symptoms, impulsivity, and family history of mental health and/or drug use problems. n = 1,210. The probabilities indicated above are significant at the .05 level.

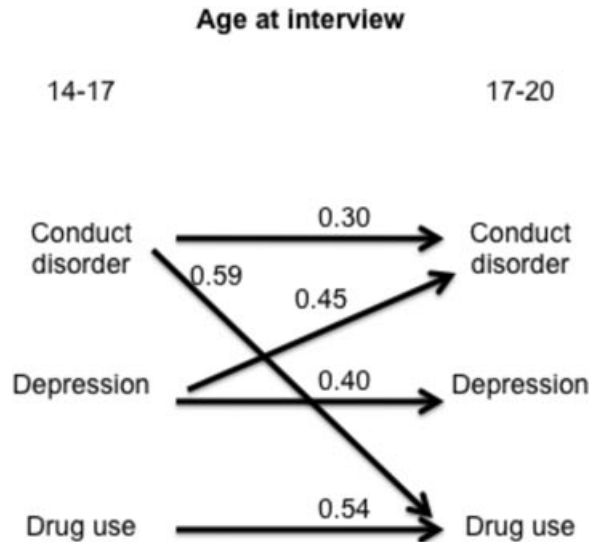


Figure 2. Probability of symptom domain persistence conditional on the presence of two other domains at prior wave and controlling for sex, age at baseline, race/ethnicity, socioeconomic status, attention deficit symptoms, impulsivity, and family history of mental health and/or drug use problems. n = 1,210. The probabilities indicated above are significant at the .05 level.

Table 3. Associations Between Past-Year Violence Exposures by Ages 12–15 and 14–17, and Depression, Conduct Disorder, and Drug Use at Ages 17–20^a

Variable	Separate model for each violence exposure				Model including all violence exposures							
	Depression		Conduct disorder		Drug use		Depression		Conduct disorder		Drug use	
	OR	95%CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Violence by ages 12–15												
Harsh physical parental punishment, past year	0.85	[0.63, 1.14]	1.55	[0.99, 2.43]	1.39	[1.03, 1.86]	0.86	[0.55, 1.35]	1.38	[0.83, 2.27]	1.23	[0.91, 1.66]
Maltreatment, past year	0.85	[0.57, 1.28]	1.38	[0.93, 2.05]	1.24	[0.92, 1.67]	0.91	[0.53, 1.57]	1.21	[0.77, 1.89]	1.13	[0.84, 1.54]
Violence by ages 14–17												
Violent victimization, past year	1.18	[0.89, 1.57]	1.74	[1.00, 3.07]	2.23	[1.57, 3.17]	1.17	[0.85, 1.62]	1.47	[0.94, 2.31]	1.96	[1.41, 2.72]
Witnessed violence, past year	1.27	[0.81, 2.00]	3.14	[1.12, 8.85]	2.76	[1.51, 5.04]	1.26	[0.77, 2.06]	2.72	[1.06, 6.97]	2.18	[1.25, 3.80]

Note. n = 1,210. OR = Odds ratio.

^aAll models included measures of for socioeconomic status, race/ethnicity, gender, family mental history of mental health/ drug use problems, impulsivity, and attention-deficit hyperactivity disorder.



Figure 3. Probability of symptom domain onset conditional on the presence of two other domains at prior wave and controlling for exposure to violence, sex, age at baseline, race/ethnicity, socioeconomic status, attention deficit symptoms, impulsivity, and family history of mental health and/or drug use problems. $n = 1,210$. The probabilities indicated above are significant at the .05 level.

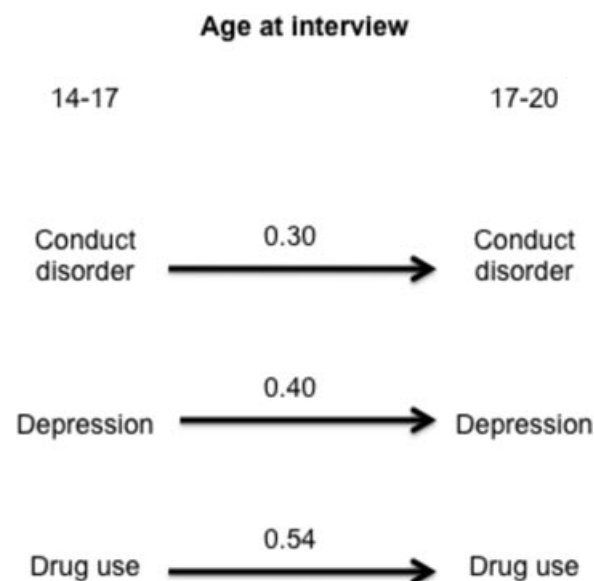


Figure 4. Probability of symptom domain persistence conditional on the presence of two other domains at prior wave and controlling for exposure to violence, sex, age at baseline, race/ethnicity, socioeconomic status, attention deficit symptoms, impulsivity, and family history of mental health and/or drug use problems. $n = 1,210$. The probabilities indicated above are significant at the .05 level.

DISCUSSION

Using data from a population-based cohort study, we (a) simultaneously examined sequential relations among depression, conduct disorder, and drug use from adolescence to young adulthood; (b) compared the influence of four types of violence on depression, conduct disorder, and drug use; and (c) examined the influence of violence exposures on the sequential relations among depression, conduct disorder, and drug use. Symptom domains were related in the following manner: (a) prior depression predicted conduct disorder onset and persistence, and (b) conduct disorder predicted drug use onset and persistence. Relations among symptom domains were not reciprocal. Exposure to parent-child and community violence was associated with conduct disorder and drug use, even after accounting for concurrent comorbidity among depression, conduct disorder, and drug use. The relationship between multiple violent exposures and each symptom domain did not explain the magnitude of the sequential relations among depression, conduct disorder, and drug use.

A sequential relation existed between prior depression and conduct disorder transitions, independent of drug use (Measelle et al., 2006). The link between depression and conduct disorder may be explained by a failure model, whereby depression in adolescence impedes relations with prosocial peers and contributes to inter-

personal conflict, reinforcing the persistence of conduct disorder into young adulthood. (Ingoldsby, Kohl, McMahon, & Lengua, 2006). Although the absence of an association between prior conduct disorder and depression transitions contradicts several prior studies (Johnson et al., 2005; Kim, Capaldi, & Stoolmiller, 2003; Silberg, Rutter, D'Onofrio, & Eaves, 2003), there may be several reasons for this. First, we may be measuring conduct disorder too late, and childhood conduct disorder may contribute to the onset of depression in adolescence. Second, prior findings of a sequential relation between conduct disorder and depression may actually reflect concurrent occurrence of conduct disorder and depression, which we were able to account for in our analyses.

We also established a sequential relation between conduct disorder and drug use, independent of depression. The extreme risky behaviors epitomized by these two symptom domains might partly result from aberrant neural processing of behavior-motivating rewards, behavior-inhibiting punishments, and impaired integration of reward-punishment information in brain regions that determine future behaviors (Crowley et al., 2010), as well as common genetic roots and an adverse social environment (Button et al., 2007; Miles, van den Bree, & Pickens, 2002).

This is one of the first studies to compare the relative influence of different forms of violence on depression, conduct disorder, and drug use. Parent-child harsh physical punishment and

maltreatment and community violence were associated with increased odds of conduct disorder and drug use (Molnar, Browne, Cerdá, & Buka, 2005; Perepletchikova & Kaufman, 2010). Harsh physical punishment and maltreatment ceased to predict conduct disorder and drug use once we included witnessing and victimization, indicating that community violence may partly mediate the relationship between harsh physical punishment and maltreatment and conduct disorder and drug use (Breslau, Davis, & Andreski, 1995). Although violence was not significantly associated with depression, the relationship between community violence and depression was in the expected direction (Johnson et al., 2002; Kennedy, Bybee, Sullivan, & Greeson, 2009, 2010).

Despite the association between exposure to violence and conduct disorder and drug use, the relations among depression, conduct disorder, and drug use were not partly explained by exposure to violence. Hence, we cannot conclude that sequential comorbidity among these symptom domains is due to concurrent, yet independent, increases in conduct disorder and drug use following exposure to violence. Although several studies have found that individuals with comorbid disorders such as depression, conduct disorder, and drug use were more likely to have experienced violence (Ethier, Lemelin, & Lacharite, 2004; Widom, DuMont, & Czaja, 2007), we are not aware of any studies that examined whether adolescent exposure to violence explained the relations between conduct disorder and either depression or drug use. Alternative shared risk factors may explain these relations, including a common genetic risk for depression, conduct disorder, and drug use, low parental warmth and parental monitoring, and association with deviant peers (Cerdá, Sagdeo, Johnson, & Galea, 2010).

This study has several limitations. Lack of access to diagnostic measures of conduct disorder led us to draw qualitative cutoffs for the presence of high conduct disorder symptoms. We did draw, however, on prior literature to identify cases of borderline symptoms from the Achenbach scales. Second, lack of measures of conduct disorder prior to Wave 1 impeded us from assessing true conduct disorder onset; instead, this study examines a shift to borderline conduct disorder within the study period. Third, onset of depression, conduct disorder, or drug use at Wave 3 may reflect onset between Waves 2 and 3 because assessments occurred every 2 years, and the measures asked about occurrence of the outcome in the past year. Fourth, this study does not account for early childhood victimization, which could have contributed to adolescent victimization and depression, conduct disorder, and drug use. Finally, the measurement of early-adolescent parent-child harsh physical punishment and maltreatment solely at Wave 1, and witnessing/victimization at Wave 2, impeded us from examining the role of time-varying violence exposures on depression, conduct disorder, and drug use.

Multiple problems such as depression, conduct disorder, and drug use are often initiated in adolescence. This peak in comorbidity makes studies of the determinants of intersections between multiple forms of psychopathology in adolescence and young adult-

hood particularly important. This is one of the first studies to examine simultaneously the relations between depression, conduct disorder, and drug use from adolescence to young adulthood and to examine the influence of exposure to violence in adolescence on psychopathology. We found that, though a history of violence did increase the risk for conduct disorder and drug use, sequential comorbidity between the three symptom domains was independent of prior exposure to violence. Future research needs to investigate factors that place individuals at risk for developing sequential relations between depression and conduct disorder, and between conduct disorder and drug use.

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