Sensitive periods for the effect of childhood interpersonal violence on psychiatric disorder onset among adolescents

Erin C. Dunn, Yan Wang, Jenny Tse, Katie A. McLaughlin, Garrett Fitzmaurice, Stephen E. Gilman and Ezra S. Susser

Background
Although childhood adversity is a strong determinant of psychopathology, it remains unclear whether there are 'sensitive periods' when a first episode of adversity is most harmful.

Aims
To examine whether variation in the developmental timing of a first episode of interpersonal violence (up to age 18) associates with risk for psychopathology.

Method
Using cross-sectional data, we examined the association between age at first exposure to four types of interpersonal violence (physical abuse by parents, physical abuse by others, rape, and sexual assault/molestation) and onset of four classes of DSM-IV disorders (distress, fear, behaviour, substance use) ($n = 9984$). Age at exposure was defined as: early childhood (ages 0–5), middle childhood (ages 6–10) and adolescence (ages 11–18).

Results
Exposure to interpersonal violence at any age period about doubled the risk of a psychiatric disorder (odds ratios (ORs) = 1.51–2.52). However, few differences in risk were observed based on the timing of first exposure. After conducting 20 tests of association, only three significant differences in risk were observed based on the timing of exposure; these results suggested an elevated risk of behaviour disorder among youth first exposed to any type of interpersonal violence during adolescence (OR = 2.37, 95% CI 1.69–3.34), especially being beaten by another person (OR = 2.44; 95% CI 1.57–3.79), and an elevated risk of substance use disorder among youth beaten by someone during adolescence (OR = 2.77, 95% CI 1.94–3.96).

Conclusions
Children exposed to interpersonal violence had an elevated risk of psychiatric disorder. However, age at first episode of exposure was largely unassociated with psychopathology risk.

Declaration of interest
None.

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Childhood adversity is a strong determinant of psychopathology, estimated to at least double the risk for both youth- and adult-onset disorders.[1–3] However, it remains unclear whether there are developmental stages when a first episode of adversity has the most impact on risk for psychopathology. Determining whether there may be 'sensitive periods'[4,5] when experience, including exposure to adversity, imparts enduring effects could provide new insight into the mechanisms underlying risk for psychopathology. It could also help determine the optimal timing of interventions, as childhood spans multiple developmental periods when different types of interventions, including home- or school-based programmes, could be deployed to minimise the effects of adversity based on the age of the child.

Although the developmental timing of child interpersonal violence could influence subsequent psychopathology risk,[6,7] there is little agreement as to whether earlier or later exposure is more harmful. Early interpersonal violence could be more damaging than later interpersonal violence because it occurs when foundational neural circuits are developing that provide the scaffolding for more complex abilities.[8,9] Further, early violence exposure could also compromise a child's ability to master early developmental tasks, such as emotion regulation and the formation of secure attachments, creating a negative developmental cascade that influences future developmental tasks.[10,11] Alternatively, later interpersonal violence could be more harmful than earlier violence exposure because adolescents have developed the cognitive skills to conceptualise experiences of interpersonal violence and their meaning.[12–15]

The brain also undergoes substantial structural and functional change during adolescence.[16]

Among the few empirical studies examining the time-dependent effects of interpersonal violence, no consensus has emerged regarding the developmental periods associated with greatest risk for psychopathology following exposure to abuse. Both prospective[17–19] and retrospective studies[20,21] have found that earlier interpersonal violence (before age 5 or before age 12[22–24]) is more strongly associated with depression than exposure during later developmental periods. However, one retrospective study[25] and two prospective studies found interpersonal violence occurring later in childhood (age 10–12)[26] or adolescence (age 12–17)[27] was more harmful than abuse that occurred earlier. Three prospective studies[7,28,29] and two retrospective studies[30,31] found no developmental timing differences. Mixed results have also been observed for the role of developmental timing of child interpersonal violence on post-traumatic stress disorder (PTSD),[19,24,32,33] substance misuse[34–36] and other mental-health related outcomes.[17,28] These mixed results could be because of methodological differences across studies. For example, prior research includes a mix of epidemiological v. non-epidemiological samples. Prior studies have also used both administrative records and interview-based self-report to collect information about interpersonal violence exposure, as well as both symptom and diagnostic measures to determine psychopathology.

In the current study, we analysed data from the National Comorbidity Survey Replication – Adolescent Supplement

(NCS-A), the largest nationally representative study of the prevalence and correlates of DSM-IV mental disorders among English-speaking US adolescents. Our objective was to examine whether variation in the timing of a first episode of childhood interpersonal violence was a determinant of disorder-specific risk for psychopathology onset. By studying adolescents, we minimised potential bias due to retrospective recall relative to studies conducted in adults. Moreover, by conducting these analyses in an epidemiological sample, we had sufficient power to conduct within-group analyses (i.e. among those exposed to interpersonal violence) and could obtain generalisable results.

Method

Sample and procedures

The NCS-A conducted face-to-face surveys on 10 148 adolescents between ages 13 and 18 from the continental USA. Respondents were sampled through a dual-frame sample comprised of adolescents from households in the National Comorbidity Survey Replication (NCS-R)[37] ($n = 904$) and adolescents from a representative sample of schools in the adult sample areas of the NCS-R ($n = 9244$ across 320 schools). Data were collected in the adolescent's home between February 2001 and January 2004 using laptop-assisted personal interviews. The overall response rate was 75.6% (85.9% household sample; 74.7% school sample).[38] Parent written informed consent and adolescent written informed assent were obtained prior to interview completion. The Human Subjects Committees of Harvard Medical School and the University of Michigan approved NCS-A study recruitment and informed consent procedures. We analysed data from adolescents with valid sampling weights and complete data on all variables ($n = 9948$, 98.03% of the total sample). Adolescents included in our analytic sample ($n = 9948$) did not differ from those who were excluded ($n = 200$) with respect to age, ethnicity, gender and poverty level. However, the excluded sample comprised more adolescents from families living in rural areas (23.1% v. 14.8%, $P = 0.05$) and with less than a high school education (26.6% v. 15.4%, $P < 0.01$).

Measures

Psychiatric disorders

To determine whether adolescents met lifetime DSM-IV diagnostic criteria for a psychiatric disorder, trained lay interviewers administered (to adolescents) a modified version of the World Health Organization Composite International Diagnostic Interview (CIDI).[39] The CIDI is a fully structured diagnostic interview designed to generate psychiatric diagnoses from the DSM-IV and the ICD-10.[39] The NCS-A version of the CIDI was slightly modified from the version used in the NCS-R to be developmentally appropriate for youth.[40] Building from the results of a factor analysis of the NCS-A data,[41] we examined four empirically defined disorder clusters: distress disorders (major depressive disorder, dysthymia, generalised anxiety disorder, PTSD, and separation anxiety disorder), fear disorders (panic disorder with or without agoraphobia, agoraphobia without panic disorder, social phobia, and specific phobia), behaviour disorders (attention-deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), conduct disorder, and eating disorders, i.e. anorexia nervosa, bulimia nervosa, and binge-eating behaviour); and substance use disorders (alcohol misuse with or without dependence, and drug misuse with or without dependence). These four disorder clusters are a parsimonious representation of psychopathology and account for the significant comorbidity within and between disorders.

Parents ($n = 8485$, 83.61% of the total sample of adolescents) also completed a self-administered questionnaire designed to ascertain data on four child disorders where prior studies have found parent reports are important for making these diagnoses:[42] depression/dysthymia, ADHD, ODD and conduct disorder. For these disorders, an 'or' criterion was applied, whereby adolescents were classified as meeting diagnostic criteria for the disorder if it was reported by either the child or parent. Agreement between parent and child reports was excellent for depression ($\kappa = 0.80$, 95% CI 0.77–0.83) and varied for externalising disorders (ADHD $\kappa = 0.35$, 95% CI 0.29–0.40; ODD $\kappa = 0.48$, 95% CI 0.45–0.51, conduct disorder $\kappa = 0.60$, 95% CI 0.55– 0.65).

Age at onset of each disorder was assessed using question probes shown in a prior study to increase recall accuracy among adults.[43] For the four disorders where combined child and parent data were used, age at onset reported by parents was used or, when missing, the age at onset reported by the child. Overall, there was good agreement in age at onset between parent and child reports (mean difference 0.83 years (s.d.) = 0.57). Agreement between parent and child reports was excellent for depression ($r = 0.92$) and varied for externalising disorders (ADHD $r = 0.21$; ODD $r = 0.86$; conduct disorder $r = 0.49$, all $P < 0.05$).

Predictors: exposure to childhood interpersonal violence

Lifetime exposure to interpersonal violence was ascertained in the PTSD screener section of the CIDI. Adolescents reported whether they had experienced any of the four types of interpersonal violence: (a) *beaten by parents*: 'badly beaten up by your parents or the people who raised you'; (b) *beaten by other person*: 'badly beaten up by anyone else'; (c) *rape*: 'someone either having sexual intercourse with you or penetrating your body with a finger or object when you did not want them to, either by threatening you or by using force', and (d) *sexual assault/molestation*: 'other than rape, ever sexually assaulted or molested'. Interviewers were instructed to query children about these events when they were alone. Age at first exposure to each event was assessed using the same question probes (as the diagnoses) to increase recall accuracy.

Covariates

In all models, we adjusted for the following covariates: age (continuous), highest level of parent education (less than high school, high school, some college, or college graduate – referent), poverty index ratio, which was derived based on family size and the ratio of family income to the family's poverty threshold ($\leqslant 1.5$, low income; $>1.5–3$, low–middle income; $>3–\leqslant 6$, high–middle income; and >6, high income – referent), ethnicity (non-Hispanic White – referent, non-Hispanic Black, Hispanic; other), region of the country (Northeast, Midwest – referent, South, West), and urbanicity (major metropolitan area, other urbanised area, rural area – referent). We also adjusted for any psychiatric disorder occurring prior to or at the same time as the focal disorder under investigation, as described in detail below. We stratified analyses by gender, given that the prevalence of interpersonal violence[44] and psychopathology[45,46] vary between males and females.

Statistical analysis

Consistent with prior NCA-analyses,[2,47] we created time-varying outcome variables, which indicated the presence or absence of any disorder within the disorder cluster (see online supplement DS1). For each outcome, we first conducted discrete-time survival analyses using logistic regression[48] to estimate the association

between exposure to each type of interpersonal violence (coded as 0 = non-exposed and 1 = exposed) and the odds for at least one disorder within the disorder cluster (model 1). We then tested the association between first exposure to interpersonal violence categorised into three different time periods and the disorder cluster (model 2). In these models, age at first exposure was coded through a set of time-varying indicators used in previous studies[19,20,49] to denote three developmental periods: 1, early childhood, ages 0–5; 2, middle childhood, ages 6–10; and 3, adolescence, ages 11–18. For this time-varying exposure variable, person-years prior to first exposure were coded as 0. Thereafter, for adolescents reporting exposure, adversity was coded as 1, 2 or 3 in the year of first exposure and in all subsequent person-years. Thus, the reference group for each analysis was based on a different denominator, corresponding to anyone who was unexposed during the current or prior developmental period. For model 2, a test of homogeneity (d.f. = 2) was used to evaluate whether the beta coefficients (indicating the effect of age at first exposure relative to never exposed) for the three developmental periods were significantly different from each other. This homogeneity test can be thought of as a test for statistical interaction between exposure (0, unexposed; 1, exposed) and developmental period (1, early childhood; 2, middle childhood 3, adolescence). Within each exposure, these tests of homogeneity were Bonferroni corrected ($\alpha = 0.05/5 = 0.01$) to account for testing for outcomes. In cases where the null hypothesis was rejected (two-sided $P < 0.01$), we conducted *post hoc* Tukey-corrected tests to evaluate, after adjustment for multiple testing, how the effect of exposure differed by developmental period. These Tukey tests were adjusted for all pairwise comparisons. All analyses were conducted using the survey regression procedures in SAS Version 9.4 to account for the complex survey design.

Results

Exposure to interpersonal violence: prevalence and age at first exposure

Descriptive and bivariate results are reported in Table 1. The sample was diverse, although predominantly White (65.7%), was comprised of roughly equal numbers of males and females, and included a large proportion of adolescents whose families were college educated (35.6%), high income (34.5%), and living in metropolitan areas (47.5%). In the bivariate analyses, exposure to any interpersonal violence was more common among females (12.2% *v.* 8.6%; $P < 0.001$) and less common among adolescents whose parents had a college degree (8.9% among children of college graduates *v.* 10.6–12.0% among children of parents with less than a college degree) (Table 1). No differences were found in the prevalence of exposure to any interpersonal violence based on ethnicity, parental income, or place of residence.

Being beaten by other person was the most commonly reported type of interpersonal violence (4.9%, $n = 469$), followed by sexual assault/molestation (3.6%, $n = 329$), rape (2.3%, $n = 252$) and being beaten by parents (1.8%, $n = 162$) (Table 2). Most youth were first exposed to interpersonal violence during adolescence. For example, among the 10.4% of youth exposed to any interpersonal violence, 1.5% were first exposed in early childhood, 3.1% in middle childhood and 5.8% during adolescence.

Youth exposed to one type of violence were somewhat more likely to report being exposed to a second type. Specifically, the correlations for exposure ranged from $r = 0.19$ for being beaten by other person and sexual assault/molestation to $r = 0.70$ for rape and sexual assault/molestation. Correlations for the timing

of first exposure ranged from 0.15 for being beaten by other person and sexual assault/molestation to 0.68 for rape and sexual assault/molestation.

Gender differences were observed in the reporting of exposure to interpersonal violence (Table 3). Girls were more likely than boys to report exposure to all types of interpersonal violence except being beaten by other person.

Exposure to any interpersonal violence was also more commonly reported among older adolescents, defined as those aged 16–18, compared with younger adolescents, defined as those aged 13–15 (13.3% *v.* 8.1% respectively; see online Table DS1). We examined whether recency effects may have potentially introduced bias in reports of adversity by examining whether older were more likely than younger adolescents to report being first exposed during adolescence. Overall, the reported age at first exposure to interpersonal violence did not vary based on the adolescent's current age (online Table DS1). Although gender differences were observed ($P < 0.01$), these differences were small in magnitude (early childhood first exposure: 0.5% male; 1% female; middle childhood: 1.3% male; 1.8% female; and adolescence: 2.7% male and 3.1% female).

Sensitive periods for the effect of interpersonal violence on psychopathology

As reported elsewhere,[50] psychiatric disorders were common and the prevalences of these disorder classes were patterned by gender, age, ethnicity and family socioeconomic status (online Table DS2). For instance, girls reported more fear disorders (32.7% *v.* 25.7%, $P < 0.001$) and distress disorders (29.4% *v.* 17.7%, $P < 0.001$), whereas boys reported more behaviour disorders (34.3% *v.* 27.7%, $P < 0.001$) and substance use disorders (12.5% *v.* 10.2%, $P = 0.01$). Additionally, distress and substance use disorders were reported less frequently by younger adolescents ($P < 0.001$).

Exposure to interpersonal violence was associated with a higher odds for nearly all types of psychiatric disorders (Table 4; online Table DS3). For example, exposure to any interpersonal violence increased the odds for a fear disorder by a factor of 1.73, after adjusting for covariates. The largest association observed was for exposure to sexual assault/molestation and the odds for any distress disorder (OR = 2.52; 95% CI 1.83–3.46).

As indicated by the P-values for the test of homogeneity, three significant differences ($P < 0.05$) were observed indicating variation in the magnitude of association between interpersonal violence and psychiatric disorders based on age at first exposure. First, we found that the effect estimate for any exposure during adolescence *v.* unexposed on behaviour disorder (OR = 2.37) was 40% larger than the effect observed for exposure during middle childhood *v.* unexposed (OR = 1.46). Second, these results appeared driven by exposure to being beaten by other person, which was more strongly associated with the risk of a behaviour disorder during adolescence (OR = 2.44) than in early or middle childhood (OR = 0.80 and 1.07, Tukey-corrected $P < 0.05$). Third, exposure to being beaten by someone was more strongly associated with the risk for substance use disorder during adolescence than it was during early childhood (OR = 2.77 *v.* 0.75, Tukey-corrected $P < 0.05$).

Although exposure to interpersonal violence was associated with a higher odds for all types of psychiatric disorder classes in both males and females, there were no significant differences among males or among females in the developmental timing of exposure to interpersonal violence (all homogeneity $P > 0.05$; see online Table DS4).

We also conducted secondary analyses on depression, the most commonly studied outcome of interpersonal violence, to facilitate

Table 1 Distribution of covariates in the total sample and by exposure to any interpersonal violence in the National Comorbidity Survey Replication – Adolescent supplement (n = 9948)[a]

Covariates	Total sample		Exposure to any violence			Beaten by parents			Beaten by other person			Rape			Sexual assault/molestation		
	n	%	n	%	P	n	%	P	n	%	P	n	%	P	n	%	P
Gender					<0.001			0.38			<0.001			<0.001			<0.001
Male	4878	51.3	428	8.6		70	1.6		343	6.7		15	0.2		41	0.8	
Female	5070	48.7	560	12.2		92	2.0		126	3.0		237	4.6		288	6.6	
Age, years					<0.001			0.01			<0.001			<0.001			0.001
13	1633	15.1	88	4.7		21	1.6		44	2.0		13	0.6		23	1.2	
14	2176	21.0	179	9.0		24	1.0		95	5.1		36	1.6		55	3.0	
15	1862	20.7	182	9.8		24	1.1		93	5.0		44	2.2		58	3.2	
16	1969	20.9	199	10.4		39	2.2		74	3.8		57	2.4		73	4.4	
17	1721	16.9	252	16.5		40	2.9		112	8.1		73	3.9		97	5.8	
18	587	5.4	88	14.8		14	3.3		51	6.8		29	5.8		23	4.9	
Ethnicity					0.35			0.55			0.03			0.35			0.99
Hispanic	1875	14.3	243	12.7		43	2.4		130	7.2		47	1.8		80	3.8	
Black	1922	15.1	178	9.9		28	1.5		72	3.7		59	3.0		55	3.7	
Other	603	4.9	76	12.5		16	2.0		43	7.5		17	2.3		23	3.6	
White	5548	65.7	491	9.8		75	1.7		224	4.5		129	2.3		171	3.6	
Parent education					0.04			0.28			0.59			0.13			0.82
Less than high school	1637	15.4	206	12.0		41	2.0		102	5.8		48	2.4		62	4.0	
High school	3025	29.6	293	10.6		44	2.2		132	4.7		79	2.6		103	3.6	
Some college	1959	19.4	222	11.6		42	1.9		102	5.5		61	3.0		71	3.9	
College graduate	3327	35.6	267	8.9		35	1.3		133	4.5		64	1.7		93	3.4	
Parent income					0.88			0.88			0.16			0.14			0.39
Low	1687	14.6	176	11.1		36	2.1		88	5.5		44	2.4		47	3.1	
Low–middle	1985	19.1	204	10.5		35	1.9		100	6.0		57	2.7		65	3.1	
High–middle	3038	31.8	306	10.5		37	1.6		128	4.1		87	2.9		123	4.5	
High	3238	34.5	302	9.9		54	1.8		153	4.8		64	1.6		94	3.4	
Urban					0.11			0.01			0.001			0.80			0.66
Metropolitan	4432	47.5	502	11.3		94	2.3		254	5.8		117	2.2		163	3.8	
Other urban	3252	37.8	333	10.2		49	1.6		156	4.7		84	2.5		112	3.7	
Rural	2264	14.8	153	7.8		19	0.7		59	2.7		51	2.4		54	2.9	

a. Cell entries are sample sizes (n) and frequencies (column percentages for total sample and row percentage by exposure) generated from models that used sampling weights to account for the differential probability of selection of respondents within households and schools, differential non-response, and adjust for differences between the sample and the US population on selected sociodemographic characteristics. Adolescents were coded as exposed to any interpersonal violence if they reported experiencing at least one type of violence (for example, beaten by parents, beaten by other person, rape, or sexual assault/molestation). Rao-Scott chi-square P-values are shown.

Table 2 Distribution of exposure to interpersonal violence in the total person-level analytic sample and by age at first exposure[a]

Exposure to interpersonal violence	*n*	%	*P*
Any interpersonal violence	988	10.4	<0.001
Early childhood	142	1.5	
Middle childhood	280	3.1	
Adolescence	566	5.8	
Beaten by parents	162	1.8	0.78
Early childhood	58	0.7	
Middle childhood	68	0.8	
Adolescence	36	0.4	
Beaten by other person	469	4.9	<0.001
Early childhood	14	0.1	
Middle childhood	118	1.3	
Adolescence	337	3.4	
Rape	252	2.3	<0.001
Early childhood	39	0.3	
Middle childhood	57	0.5	
Adolescence	156	1.5	
Sexual assault/molestation	329	3.6	<0.001
Early childhood	51	0.6	
Middle childhood	90	1.1	
Adolescence	188	2.0	

a. Cell entries are sample sizes (*n*) and frequencies (percentages) generated from models that used sampling weights to account for the differential probability of selection of respondents within households and schools, differential non-response and adjust for differences between the same and the US population on selected sociodemographic characteristics. Adolescents were coded as exposed (*v.* unexposed) for any interpersonal violence and each type of interpersonal violence. Rao-Scott chi-square *P*-values are shown.

comparisons with prior studies. Exposure to any interpersonal violence was associated with a higher odds for depression in the total sample (see online Table DS5) and gender-stratified analyses (see online Table DS6). No significant differences emerged based on developmental timing of exposure to interpersonal violence on depression (all homogeneity $P > 0.05$).

Discussion

These data do not support the view that there are sensitive periods shaping risk for psychiatric disorder among adolescents following exposure to interpersonal violence. Although adolescents who were maltreated had, on average, about twice the risk of having an onset of a psychiatric disorder, age at first episode of exposure to interpersonal violence was generally not associated with the magnitude of increased risk. Several previous prospective[7,28,30] and retrospective studies[30,31] in much smaller samples of youth have also not found evidence for sensitive periods. The lack of identified sensitive periods here and elsewhere could be because

interpersonal violence disrupts multiple developmental processes, including attention and emotion regulation, which each could have overlapping influences on psychopathology.[10,11] Thus, there may be differentially sensitive periods for each of these developmental processes,[51] but not a single sensitive period when exposure to violence increases susceptibility to psychiatric disorders defined through symptom-level measures.

Although by and large there was little evidence to support the primary hypothesis that there were sensitive periods, we did observe three instances in which the developmental timing of exposure to adversity appeared to matter. First, youth exposed to any type of interpersonal violence during adolescence had an increased risk of experiencing a behaviour disorder relative to youth first exposed in middle childhood. Second, the effect of any interpersonal violence on behaviour disorders appeared primarily driven by being beaten by other person (i.e. who was not a parent), as youth first exposed in adolescence to this type of violence had an elevated risk of behaviour disorder relative to youth first exposed during middle childhood. Third, being beaten by another person during adolescence also elevated risk of substance use disorder relative to exposures that first occurred during the earliest stage of development. These findings are consistent with some,[17] although not all,[27,30] studies examining the effects of timing of childhood interpersonal violence on these outcomes in adolescents. To our knowledge, no studies have assessed the effect of developmental timing of exposure to adversity in relation to subsequent eating disorders.

Interpersonal violence during adolescence may exert disorder-specific effects on behaviour and substance use disorders for several reasons. In addition to more advanced cognitive abilities[14,15] and brain-related changes that occur during adolescence,[16] adolescent-onset interpersonal violence could more often result in behavioural manifestations of psychopathology relative to earlier-onset interpersonal violence exposure. Previous research indicates that the prevalence of conduct disorder increases with age, at least up to 16 years of age,[52] and that the diagnosis of eating disorders in children under 10 is rare.[53] Moreover, recent work also suggests that for some cognitive and affective capacities, the time of greatest sensitivity to the environment is during adolescence.[49,54–57]

Strengths and limitations

The current study had several strengths. We conducted these analyses in a large population-based sample of adolescents, enabling us to generate nationally representative estimates of disorder-specific effects, reduce the likelihood of retrospective recall bias relative to studies conducted in adults, and capture a larger proportion of maltreated youth relative to studies examining administrative records. Unlike prior NCS-A studies, we included data from youth whose parents did not participate.

Table 3 Distribution of exposure in the total person-level analytic sample by gender[a]

Exposure	Total sample (*n* = 9948)		Males (*n* = 4878)		Females (*n* = 5070)		
	n	%	*n*	%	*n*	%	*P*
Any interpersonal violence	988	10.4	428	4.4	560	6.0	<0.001
Beaten by parents	162	1.8	70	0.8	92	1.0	0.38
Beaten by other person	469	4.9	343	3.5	126	1.5	<0.001
Rape	252	2.3	15	0.1	237	2.2	<0.001
Sexual assault/molestation	329	3.6	41	0.4	288	3.2	<0.001

a. Cell entries are sample sizes (*n*) and frequencies (percentages) generated from models that used sampling weights to account for the differential probability of selection of respondents within households and schools, differential non-response, and adjust for differences between the same and the US population on selected sociodemographic characteristics. Adolescents were coded as exposed (*v.* unexposed) for any interpersonal violence and each type of interpersonal violence. Rao-Scott chi-square *P*-values corresponding to the association between gender and exposure to interpersonal violence are shown.

Table 4 Odds of experiencing each psychiatric disorder by the timing of exposure to interpersonal violence[a]

Exposure to interpersonal violence	Fear disorder OR (95% CI)	Homogeneity P[b]	Distress disorder OR (95% CI)	Homogeneity P[b]	Behaviour disorder OR (95% CI)	Homogeneity P[b]	Substance use disorder OR (95% CI)	Homogeneity P[b]
Any interpersonal violence	**1.73 (1.40–2.13)**	0.33	**2.47 (1.94–3.16)**	0.97	**1.82 (1.49–2.22)**	0.01	**2.39 (1.88–3.02)**	0.47
Early childhood					**1.87 (1.33–2.63)**			
Middle childhood					**1.46 (1.19–1.80)**			
Adolescence					**2.37 (1.69–3.34)**[c]			
Beaten by parents	0.83 (0.49–1.42)	0.06	**1.99 (1.35–2.95)**	0.20	**1.63 (1.15–2.30)**	0.95	**2.36 (1.37–4.04)**	0.30
Early childhood								
Middle childhood								
Adolescence								
Beaten by other person	**1.79 (1.19–2.71)**	0.80	**1.73 (1.22–2.47)**	0.13	**1.59 (1.09–2.33)**	0.003	**2.18 (1.57–3.02)**	0.01
Early childhood					0.80 (0.29–2.23)		0.75 (0.37–1.54)	
Middle childhood					1.07 (0.65–1.74)		1.22 (0.54–2.76)	
Adolescence					**2.44 (1.57–3.79)**[c]		**2.77 (1.94–3.96)**[d]	
Rape	**2.19 (1.27–3.78)**	0.99	**1.96 (1.28–3.02)**	0.23	**1.53 (1.13–2.07)**	0.19	**2.01 (1.22–3.32)**	0.86
Early childhood								
Middle childhood								
Adolescence								
Sexual assault/molestation	**1.53 (1.11–2.11)**	0.76	**2.52 (1.83–3.46)**	0.70	**1.51 (1.19–1.92)**	0.89	1.55 (0.98–2.47)	0.69
Early childhood								
Middle childhood								
Adolescence								

a. Cell entries are odds ratios and 95% confidence intervals estimating the association between exposure to interpersonal violence (exposed v. unexposed) and timing of exposure to interpersonal violence (for example, exposed during early childhood v. unexposed). Statistically significant odds ratios ($P < 0.05$) indicated in bold. Models were adjusted for gender, age, highest level of parent education, poverty index ratio, ethnicity, region of country, urbanicity and co-occurring psychiatric disorder.
b. P-values from the test of homogeneity after Bonferroni correction (0.05/4 = 0.01), which corrected for the testing of four outcomes within each exposure are shown.
c. Significant difference in the odds of the psychiatric disorder ($P < 0.05$) between exposure in middle childhood v. adolescence, based on the Tukey *post hoc* test.
d. Significant difference in the odds of the psychiatric disorder ($P < 0.05$) between exposure in early childhood v. adolescence, based on the Tukey *post hoc* test.

Some forms of violence were more common among youth without parent-reported data. We also considered exposures individually, rather than simultaneously,[58] in order to guide intervention planning and understand interpersonal violence-specific associations to risk for psychiatric disorder.

However, several limitations should be noted. First, exposure to interpersonal violence, meaning its occurrence and age at first exposure, were assessed retrospectively. Retrospective reports of interpersonal violence may be less reliable and valid than prospective reports, because of memory inaccuracies, a reluctance to disclose personal matters, or current mood states.[59] However, a major strength of our study is that the recollection period to recall experiences of interpersonal violence exposure was brief, as adolescents were recalling their exposures during earlier childhood. Further, specific question probes were used to increase recall accuracy with respect to age at first exposure to these forms of interpersonal violence. Prospective research is needed to replicate these findings. This prospective work should incorporate repeated measures of trauma exposure and mental health in order to differentiate short- *v.* long-term effects of trauma timing on psychopathology. Important to note, even if such reporting biases were present in our study, retrospective and prospective measures produce similar estimates of association with mental disorders,[60] suggesting that trauma exposure is harmful regardless of ascertainment strategy.

Second, the interpersonal violence measure did not capture information about its severity, chronicity, or duration. Thus, for the three associations where sensitive periods were observed, we are unable to discern whether these developmental timing effects persisted after accounting for these other features of the adversity, which could have possibly inflated these observed associations. These adversity characteristics should be included in future studies to disentangle their contribution to the possible effects of age at first exposure. Related to this, because of the small number of cases of adolescents with behaviour disorders, we were unable to evaluate the age at first exposure to violence on specific types of behaviour disorders. Finally, our results for adolescent first exposure may be misleading, as not everyone in the sample was old enough to have contributed data for the full exposure period. Studies in older adolescents or young adults can help illuminate the importance of both child- and adolescent-onset exposure to interpersonal violence.

Implications

In conclusion, our results suggest that, irrespective of the age at first exposure to violence and the type of interpersonal violence, exposure to violence more than doubled the odds of having a psychiatric disorder. However, age at first episode of exposure appears largely unassociated with psychopathology risk. If sensitive periods for psychopathology do exist, they may not be apparent during adolescence or easily identified through symptom-level measures. Ongoing work to investigate sensitive periods, especially in population-based samples, can help determine whether there are developmental stages when adversity has a more enduring impact. Insights from this work could inform clinical and prevention efforts by suggesting the age stages when interventions are most able to prevent the onset of mental health disorders and when public health expenditures can yield greater returns on investment.

Erin C. Dunn, ScD, MPH, Psychiatric and Neurodevelopmental Genetics Unit, Massachusetts General Hospital, Boston, Department of Psychiatry, Harvard Medical School, Boston and Stanley Center for Psychiatric Research, The Broad Institute of Harvard and MIT, Cambridge, Massachusetts; **Yan Wang**, PhD, MPH, Psychiatric and Neurodevelopmental Genetics Unit, Massachusetts General Hospital, Boston, Massachusetts; **Jenny Tse**, BA, Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, Massachusetts; **Katie A. McLaughlin**, PhD, Department of Psychology, University of Washington, Seattle, Washington; **Garrett Fitzmaurice**, PhD, Department of Psychiatry, Harvard Medical School, McLean Hospital, Laboratory for Psychiatric Biostatistics, Belmont and Department of Biostatistics, Harvard T.H. Chan School of Public Health, Boston, Massachusetts; **Stephen E. Gilman**, ScD, Department of Epidemiology and Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health and Health Behavior Branch, Division of Intramural Population Health Research, Eunice Kennedy Shriver National Institute of Child Health and Human Development, Rockville, Maryland; **Ezra S. Susser**, MD, DrPH, Department of Epidemiology, Mailman School of Public Health, Columbia University, New York and New York State Psychiatric Institute, New York, USA

Correspondence: Erin C. Dunn, ScD, MPH, Psychiatric and Neurodevelopmental Genetics Unit, Center for Genomic Medicine, Massachusetts General Hospital, 185 Cambridge Street, Simches Research Building Sixth Floor, Boston, MA 02114, USA. Email: dunnreprints@gmail.com

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