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The Impact of Exposure to Interpersonal Violence on Gender Differences in Adolescent-Onset Major Depression: Results from the National Comorbidity Survey Replication (NCS-R)

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Abstract

Background—Beginning in adolescence, females are at significantly higher risk for depression than males. Despite substantial efforts, gaps remain in our understanding of this disparity. This study tested whether gender differences in adolescent-onset depression arise because of female's greater exposure or sensitivity to violence.

Methods—Data came from 5692 participants in the National Comorbidity Survey Replication. Trained interviewers collected data about major depression and participants' exposure to four types of interpersonal violence (physical abuse, sexual assault, rape, and witnessing violence) using a modified version of the Composite International Diagnostic Interview. We used discrete time survival analysis to investigate gender differences in the risk of adolescent onset depression.

Results—5.7% of the sample met DSM-IV criteria for depression by age 18; 5.8% of the sample reported being physically abused, 11.7% sexually assaulted, 8.5% raped, and 13.2% witnessed violence by age 18. Females had 1.51 times higher odds of depression by age 18 than males. Exposure to all types of violence was associated with an increased odds of depression in both the past year and the years following exposure. Adjusting for exposure to violence partially attenuated the association between gender and depression, especially for sexual assault (OR attenuated=1.28;

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15.23%) and rape (OR attenuated=1.32; 12.59%). There was no evidence that females were more vulnerable to the effects of violence than males.

Discussion—Gender differences in depression are partly explained by females' higher likelihood of experiencing interpersonal violence. Reducing exposure to sexual assault and rape could therefore mitigate gender differences in depression.

Keywords

depression; violence; mediation; stress exposure; stress reactivity

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Gender differences in depression are one of the most consistent findings in psychiatric epidemiology.[1–3] Although boys and girls experience similar levels of depression during childhood,[4] with some studies even finding that boys experience more depression than girls, [5] females begin to outnumber their male counterparts by a ratio of two-to-one starting somewhere between ages 13 and 15.[6; 7] This disparity persists into adulthood,[8; 9] and across various measures of depression (e.g. depressive symptoms; diagnosis of major depression),[7; 10] and racial/ethnic groups.[11; 12] Despite research on the ways biological (e.g. hormones, pubertal timing, maturation), psychological (e.g. gender-role development; coping style), and social factors (e.g. exposure to stress) shape the onset and persistence of gender differences in depression, gaps remain in our understanding of the causes of this disparity.[13–16]

One likely explanation involves exposure to interpersonal violence.[3; 17] Exposure to violence is associated with an increased risk of depression in both adolescence and adulthood;[18–20] in addition, females are more likely than males to experience violence, especially sexual abuse, rape or sexual assault, and witness family violence. [21–25] Since the risk of exposure to some types of violence, including being sexually victimized, sharply increases for females around early adolescence[26] – the time when gender differences in depression emerge – exposure to interpersonal violence could explain excess lifetime risks of depression among females and therefore have implications for preventing gender-based disparities.

There are two mechanisms by which exposure to interpersonal violence could contribute to gender differences in depression: 1) through females' *differential exposure* to violence; and 2) through females' *differential sensitivity* to the effects of exposure to violence.[6; 15] The *differential stress exposure hypothesis* argues that females are more likely to be exposed to stressors, which increases their risk of becoming depressed. This is a hypothesis of "mediation" and is empirically tested in four steps that determine whether: (1) there are gender differences in depression; (2) there are gender differences in exposure to violence, with females presumably being more likely to be exposed; (3) exposure to violence is associated with depression; and (4) the association between gender and depression is reduced after accounting for exposure to violence (i.e. the "mediator").[27; 28] The *differential stress sensitivity hypothesis* states that males and females experience the effects of interpersonal violence differently. This hypothesis is one of "effect modification," and support for it is found when females are shown to be more likely to experience depression in response to stressors than males, explaining why they are more depressed. Girls may be

more likely to respond to violence in the form of depression than boys for several reasons. For instance, girls are more likely than boys to ruminate.[29] This cognitive style may sensitize girls to stress in ways that lead them to experience heightened stress reactivity. Girls are also more likely than boys to experience internalizing symptoms whereas externalizing symptoms are more common in boys.[30; 31]

Studies using both adolescent and adult samples have investigated the differential stress exposure[32–34] and differential stress reactivity hypotheses.[34–36] Fergusson and colleagues [33] used longitudinal data to examine whether exposure to sexual abuse by age 16 and sexual assault between ages 16–28 accounted for the association between gender and both depression and anxiety. They found the odds of experiencing depression and anxiety declined by 24% (from OR=2.5 to OR=1.9) after controlling for exposure to sexual violence, suggesting that these exposures partially accounted for gender differences in depression. Kessler [32] also estimated that the odds of a first-onset of depression were reduced by half after accounting for exposure to rape and sexual trauma.

Research on the differential stress sensitivity hypothesis is inconsistent. A recent review [35] of thirty studies showed that while some studies did not observe gender differences in the effects of abuse on risk for depression,[34; 37; 38] others did find that females were more vulnerable to the effects of child sexual abuse or physical abuse[36; 39] and exposure to violence.[40] For instance, Molnar and colleagues[19] found that exposure to childhood sexual abuse was associated with a higher risk of dysthymia in females (OR=1.9) compared to males (OR=1.5), but not depression, where the odds ratio was 1.8 for both males and females. However, these results were obtained from a stratified analysis where no statistical test for interaction was conducted. Thus, while there is more evidence to support the differential stress exposure hypothesis, both models are plausible explanations for gender differences in depression. In fact, these models are not mutually exclusive, but may work simultaneously.

The literature on both hypotheses is limited in several ways. First, few studies conduct the statistical analyses required to test either hypothesis, instead providing only descriptive support that may be suggestive of mediation or moderation. Second, even when necessary statistical tests are employed, the focus of prior research has been on how exposure to violence during childhood might increase risk for adult-onset depression. Instead, our goal was a better understanding of how exposure to violence during childhood precipitates the first onset of childhood and adolescent-onset depression. By focusing specifically on the effects of violence during childhood and adolescence, new knowledge can be gained about the origins of gender differences in depression and the factors that lead to the start of an excess burden of depression among females compared to males. Third, prior studies have tended to focus on how exposure to violence increases risk for depression over many years, rather than testing for whether it can also increase risk immediately following the first exposure. Fourth, few studies test both hypotheses simultaneously. This is a shortcoming as both hypotheses are plausible; evidence in support of one hypothesis does not rule out the possibility of the other. The implications for intervention are different for each one. Finally, many studies have tested these hypotheses by focusing exclusively on exposure to sexual

violence. However, other types of victimization may lead to gender differences in depression.

In this study, we overcome these limitations by testing the differential stress exposure (mediation) and differential stress sensitivity (effect modification) hypotheses using data from a large, nationally-representative survey of adults. We focus on four types of violence: physical abuse, rape, sexual assault and witnessing violence. These four types are highly prevalent, especially among youth, and confer a moderate to large general risk for many psychiatric disorders, including depression.[25; 41; 42] These exposures were also more common in the current sample when compared to other types of interpersonal violence exposures, such as being threatened with a weapon, stalked, and kidnapped or held captive.[25] We also formally test whether the risk for child or adolescent-onset depression is limited to the same year in which the exposure occurred or whether the effects of violence exposure persist for subsequent years. This approach is informative given that exposure to adversity can exert an immediate or long-term effect on disease risk, as argued by life course approaches.[43–46] Given past evidence of substantially higher rates of interpersonal violence among females, we hypothesized that gender differences in adolescent-onset depression would be due to females' differential stress exposure, rather than females' differential sensitivity to violence.

Methods

Sample

Participants came from the *National Comorbidity Survey Replication* (NCS-R), a nationally-representative, cross-sectional, population-based survey of 9282 English-speaking people between ages 18 and 99 living in the 48 contiguous United States.[9; 47–49]

Procedures

The NCS-R survey was administered in person between February 2001 and April 2003, in two parts. Part One focused on diagnostic assessments of primary psychiatric disorders and was completed by 9282 participants. Part Two, which was administered to a probability sample of 5692 participants drawn from Part One, focused on a set of secondary psychiatric disorders, risk factors, consequences, and other background characteristics. The current research study focuses on the 5692 participants who completed both Part 1 and Part 2 because questions from Part 2 were required for the present analysis. Verbal consent was obtained prior to beginning each interview. Recruitment and consent procedures were approved by the Human Subjects Committees at both *Harvard Medical School* and the *University of Michigan*.

Instruments and Variables

To determine whether participants met DSM-IV diagnostic criteria for major depression (Part One), trained, non-clinician interviewers administered a modified version of the *World Health Organization's Composite International Diagnostic Interview* (WMH-CIDI), a widely-used, valid, reliable, and structured tool that generates psychiatric diagnoses from the DSM-IV and the International Classification of Disease, 10th Revision.[50] Detailed data were

collected on the course of each psychiatric disorder, including age at first onset. We classified participants as having experienced an adolescent-onset depression or an exposure to violence only when their first event onset occurred by age 18, an age commonly used to mark the end of adolescence.[51]

In the post-traumatic stress disorder screening section of the interview (Part Two), interviewers asked participants to report whether they had ever experienced a list of potentially traumatic events, including the four exposures examined here. Physical abuse was assessed by the question "As a child were you ever badly beaten up by your parents or the people who raised you?" Sexual assault was assessed by the question "Other than rape, were you ever sexually assaulted where someone touched you in appropriately, or when you did not want them to?" Rape was assessed based on the following criteria: "having 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 using force, or when you were so young that you didn't know what was happening." Witnessing violence was assessed by the question "When you were a child, did you ever witness serious physical fights at home, like when your father beat up your mother?" If a participant endorsed an event, the interviewer then asked the participant to report how old he/she was the first time the event occurred.

Statistical Analysis

First, we examined the distribution of adolescent-onset depression in the total sample and by gender and exposure to interpersonal violence. We then constructed a "person-year" dataset in which we conducted discrete-time survival analyses of the onset of major depression by age 18.[52] In this dataset, each participant contributed multiple rows of data, with each row representing a year they were at risk of depression onset between ages 4 (the earliest onset age in the sample) through 18. The discrete-time survival model is then estimated using logistic regression in the "person-year" dataset, in which the dependent variable is the first onset of depression in each person-year (0=no depression; 1=depression). Odds ratios from this analysis indicate the risk of depression associated with both time-invariant (gender) and time-varying (exposure to violence) predictors.[53] Participants contributed data (i.e., had a row) for each year at which they were at risk for experiencing the outcome. Once participants experienced the outcome or reached age 18, they were censored, meaning that they could not longer contribute a row of data to the analysis. The baseline hazard of depression was modeled by the linear and squared terms of age.

We treated the four types of violence exposures as time-varying covariates in the survival analyses in order to estimate the risk of depression associated with each type of violence in the following time periods: exposure to violence in the past year (past year), exposure to violence in the years following exposure (years after exposure), and no exposure to violence reported during any time period (no exposure). This enabled us to consider how the association between violence exposure and depression may differ between the year of first exposure and the years following first exposure, through age 18. We also created a time-varying binary variable depicting whether or not the participant had been exposed to violence. In this specification, participants were coded "0" in the years they were unexposed

and “1” in the year they were exposed as well as any subsequent years. This variable was necessary to test our moderation hypothesis, given that sample size constraints prevented us from using the three-category variable to test this hypothesis.

We fitted five logistic regression models in which the dependent variable was the onset of depression in each person-year. Model 1 contained the main effect of gender on depression; Models 2 and 3 contained the main effect of violence, for each type and by different time period, on depression; Model 4 contained the main effects of gender and exposure to violence on depression; and Model 5 added an interaction term into Model 4, which represented the cross-product of predictors gender and exposure to violence. We evaluated mediation by comparing the coefficient relating the effect of gender on depression in Model 1 to the coefficient in Model 4 and estimated the proportion of attenuation (i.e. estimated a percent change in the gender difference in depression).[27; 28] We tested for moderation by testing whether the cross-product term in Model 5 was equal to zero.

All analyses were conducted using the survey regression procedures available in SAS Version 9.2 to account for the complex survey design. We used sampling weights to account for differential probability of selection of respondents within households, differential non-response, and adjust for differences between the sample and the US population on selected socio-demographic characteristics.[48] All models controlled for age at interview, participants’ parental education as a measure of socioeconomic status in childhood (highest level attained by either mother or father; 1=less than high school; 2=high school; 3=college; 4=missing response) and self-reported race/ethnicity (1=hispanic; 2=black; 3=other; 4=white).

Results

We excluded 3.4% (n=194) of participants who were missing data on variables that described their exposure to violence. This provided an analytic sample of 5498 participants who contributed 80,142 person-years of data. Participants who were excluded from the analysis were no different from participants that were included with respect to their gender (χ^2=0.31, p=0.58). However, compared to participants who were included, a higher proportion of participants who were excluded experienced an adolescent-onset depression (12.80% vs. 5.66%; χ^2=17.0, p<0.0001).

In the total sample, 5.7% (6.5% of females and 4.7% of males) met DSM-IV criteria for a major depressive episode by age 18. As shown in Table 1, 5.8% of the sample reported having been badly beaten up by their parents or caregivers, 11.7% had been sexually assaulted, 8.5% raped, and 13.2% witnessed serious physical fights at home. A higher proportion of females were exposed to all types of violence except physical abuse; this finding is consistent with the differential stress exposure hypothesis.

Table 2 reports the results of Models 1–4. As shown in Model 1, females had 1.51 times the odds of depression by age 18 compared to males (95% CI, 1.24–1.83). As shown in Model 2, exposure to all 4 types of violence was associated with an increased odds of depression in both the past year and the years following exposure compared to no exposure. Past year

exposure to violence conferred the largest increase in the odds of depression, with the association most pronounced for physical abuse (OR, 7.81; 95% CI, 3.21–19.01) and rape (OR, 6.97; 95% CI, 4.04–11.86). Although the magnitude of the association was smaller, there remained an increased odds of depression, relative to the reference group (no exposure), for each type of violence exposure in the years following first exposure to violence (years after exposure), with the increased odds being most pronounced for sexual assault (OR, 3.37; 95% CI, 2.54–4.46) and rape (OR, 3.05; 95% CI, 2.21–4.20). In comparing the effects of past year exposure to an alternative reference group, specifically years after exposure (Model 3), we found that past year exposure was associated with an increased odds of depression for physical abuse (OR, 3.55; 95% CI, 1.46–8.64), rape (OR, 2.29; 95% CI, 1.25–4.19), and witnessing violence (OR, 2.75; 95% CI 1.31–5.80), but not sexual assault (OR, 1.30; 95% CI, 0.74–2.28).

Adjusting for exposure to violence in Model 4 partially attenuated the association between gender and depression. The percent change in coefficients for female from Model 1 (OR=1.48) to Model 4 was greatest for sexual assault (OR attenuated to 1.28; 15.23%) and rape (OR attenuated to 1.32; 12.59%), suggesting that exposure to these forms of violence partially mediated the association between gender and depression. Only modest changes in coefficients were observed for physical abuse (OR attenuated to 1.48; 1.98%) and witnessing violence (OR attenuated to 1.46; 3.31%), suggesting that these variables likely do not explain the association between gender and depression.

We also examined whether exposure to any type of violence explained gender differences in depression (results not shown). In a model that included all four types of violence, females continued to have an increased risk of depression relative to males, though the odds ratio declined by 17.22% to 1.25 (95% CI, 1.01–1.56).

Model 5 (not shown) tested the stress reactivity hypothesis (effect modification). The tests of interaction between gender and each type of violence were all statistically non-significant: physical abuse (β=0.10, p=0.66), sexual assault (β=0.01, p=0.98), rape (β=0.28, p=0.44), and witnessing violence (β=0.09, p=0.52). Thus, we did not find evidence to support the differential stress reactivity hypothesis.

Comment

This study used data from a nationally representative survey to test the differential stress exposure and stress sensitivity hypotheses as explanations for gender differences in adolescent-onset depression. Consistent with prior literature,[18–20; 25] we found that exposure to violence was strongly associated with heightened risk for depression. We extend existing research by demonstrating that the risk for depression is not limited to the same year in which the exposure occurred, but persists for subsequent years. This finding underscores the need for both early identification and ongoing prevention and intervention programs to identify youth exposed to violence.

Exposure to rape and sexual assault also partially explained gender differences in depression, as suggested by the differential stress exposure hypothesis. Specifically, the

gender difference in the odds of adolescent-onset depression was reduced by 12.59% and 15.23%, respectively, after accounting for exposure to these types of violence. It was also reduced by 17.22% after accounting for exposure to any type of violence and became close to statistically non-significant. Although the magnitude of these mediated effects may seem small, they are potentially meaningful when considered within a public health approach to prevention, which seeks to shift the underlying distribution of risk for depression within the entire population.[54] Thus, this finding suggests that if the associations reported are causal, reducing girls' exposure to violence, particularly rape and sexual assault, would decrease overall gender disparities in depression. However, even after accounting for exposure to any type of violence, the finding that gender differences in depression persist indicates that gender differences are not fully explained. This finding underscores the need for future research to examine exposure to violence along with other potentially relevant determinants for explaining gender differences in depression. These should span biological to social factors.

There was no support for the differential stress sensitivity hypothesis. The lack of support for this hypothesis suggests that these types of adversities are equally damaging for all youth, regardless of their gender. We also found that witnessing violence was neither a mediator nor moderator of the association between gender and depression, suggesting that witnessing violence is unlikely to play a role in explaining gender differences in depression.

Strengths of this study include the use of a population-based sample, DSM-IV criteria to define depression, and focus on the developmental period of adolescence, the time when gender differences in depression emerge. The cross-sectional design of the study, where exposure and outcome data were collected retrospectively, is a weakness. Although we took account of timing in the exposure-depression association in our analytic approach, it is possible our estimates are inaccurate. Prospective research is needed to provide a stronger test of these hypotheses. Recall bias could have also occurred if participants who were exposed to violence or experienced depression reported their experiences differently from those without these experiences. If differences in reporting were patterned by gender, study results could be biased. Future research should examine whether systematic differences exist in reporting these experiences by gender and psychiatric status and use externally validated measures of exposure to violence along with self-reported measures. We also did not have detailed data on the duration of exposure to violence, making it impossible to determine whether effects in the years after exposure were truly long-term or the cumulative effect of continuing or ongoing exposure. The exclusion of participants with missing data on the violence exposure could have created a bias, as a greater percentage of these participants had experienced depression relative to the participants who were included. Finally, we focused on one set of stressors as an explanation for gender difference in depression. Many factors are likely to play a role in explanting gender differences in adolescent-onset depression. Future research should include a more comprehensive set of potential determinants, as others have done,[55] so that comparisons can be made across risk factors and conclusions can be drawn about the relative importance of one factor over another.

This study contributes to the literature by showing how gender-based disparities in depression could be reduced by lowering girls' exposure to interpersonal violence,

especially rape and sexual assault, the two types of violence that had much higher exposures for females than males. Early exposure to sexual traumas have been shown in longitudinal studies to be associated with myriad negative sequelae across the life course, including physical and social outcomes in addition to psychiatric disorders.[56] Prevention efforts aimed at reducing girls' exposure to these traumas, particularly during childhood and adolescence, could help to significantly reduce current gender-based disparities in depression that emerge during childhood and adolescence and that carry forward over the life course.

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References

1. Kuehner C. Gender differences in unipolar depression: An update of epidemiological findings and possible explanations. Acta Psychiatric Scandinavia. 2003; 108(3):163–174.
2. Piccinelli M, Wilkinson G. Gender differences in depression: Critical review. British Journal of Psychiatry. 2000; 177:486–492. [PubMed: 11102321]
3. Nolen-Hoeksema, S.; Hilt, LM. Gender differences in depression. In: Gotlib, IH.; Hammen, CL., editors. Handbook of depression. Second ed. New York, NY: The Guilford Press; 2009. p. 386-404.
4. Rutter M. Child psychiatry: the interface between clinical and developmental research. Psychological Medicine. 1986; 16:151–169. [PubMed: 3961041]
5. Twenge JM, Nolen-Hoeksema S. Age, gender, race, SES, and birth cohort differences on the Children's Depression Inventory: a meta analysis. Journal of Abnormal Psychology. 2002; 111:578–588. [PubMed: 12428771]
6. Nolen-Hoeksema S, Girgus JS. The emergence of gender differences in depression during adolescence. Psychological Bulletin. 1994; 115(3):424–443. [PubMed: 8016286]
7. Hankin BL, Abramson LY, Moffitt TE, Silva PA, McGee R, Angell KA. Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10 year longitudinal study. Journal of Abnormal Psychology. 1998; 107:128–141. [PubMed: 9505045]
8. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributinos of DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry. 2005; 62:593–602. [PubMed: 15939837]
9. Kessler RC, Berglund P, Demler O, Jin R, Koretz D, Merikangas KR, et al. The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). Journal of the American Medical Association. 2003; 289:3095–4105. [PubMed: 12813115]
10. Galambos NL, Leadbeater BJ, Barker ET. Gender differences in and risk factors for depression in adolescents: A 4-year longitudinal study. International Journal of Behavior Development. 2004; 28:16–25.
11. Blazer DG, Kessler RC, McGonagle KA, Swartz MS. The prevalence and distribution of major depression in a national community sample: The National Comorbidity Survey. American Journal of Psychiatry. 1994; 151:979–986. [PubMed: 8010383]
12. Williams DR, Gonzalez HM, Neighbors HNR, Abelson JM, Sweetman J, et al. Prevalence and distribution of major depressive disorder in african americans, caribbean blacks, and non-hispanic whites. Archives of General Psychiatry. 2007; 64:305–315. [PubMed: 17339519]
13. Zahn-Waxler C, Shirtcliff EA, Marceau K. Disorders of childhood and adolescence: Gender and psychopathology. Annual Review of Clinical Psychology. 2008; 4:275–303.
14. Crick NR, Zahn-Waxler C. The development of psychopathology in females and males: Current progress and future challenges. Development and Psychopathology. 2003; 15:719–742. [PubMed: 14582938]

15. Hankin, BL.; Wetter, E.; Cheely, C. Sex differences in child and adolescent depression: A developmental psychopathological approach. In: Abela, JRZ.; Hankin, BL., editors. Handbook of depression in children and adolescents. New York, NY: The Guilford Pres; 2008. p. 377-414.
16. Hyde JS, Mezulis AH, Abramson LY. The ABCs of depression: integrating affective, biological, and cognitive models to explain the emergence of gender differences in depression. Psychological Review. 2008; 115(2):291–313. [PubMed: 18426291]
17. Kessler RC. The effects of stressful life events on depression. Annual Review of Psychology. 1997; 48:191–214.
18. Chapman DP, Whitfield CL, Felitti VJ, Dube SR, Edwards VJ, Anda RF. Adverse childhood experiences and the risk of depressive disorders in adulthood. Journal of Affective Disorders. 2004; 82:217–225. [PubMed: 15488250]
19. Molnar BE, Buka SL, Kessler RC. Child sexual abuse and subsequent psychopathology: Results from the national comorbidity survey. American Journal of Public Health. 2001; 91:753–760. [PubMed: 11344883]
20. Widom CS, DuMont K, Czaja SJ. A prospective investigation of major depressive disorder and comorbidity in abused and neglected children grown up. Archives of General Psychiatry. 2007; 64:49–56. [PubMed: 17199054]
21. Cutler SE, Nolen-Hoeksema S. Accounting for sex differences in depression through female victimization: Childhood sexual abuse. Sex Roles. 1991; 24(7/8):425–438.
22. Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, Koss MP, Marks JS. Relationship of chilhood abuse and household dysfunction in many of the leading causes of death in adults: the Adverse Childhood Experiences (ACE) study. American Journal of Preventive Medicine. 1998; 14:245–258. [PubMed: 9635069]
23. Putnam FW. Ten-year research update review: Child sexual abuse. Journal of the American Academy of Child and Adolescent Psychiatry. 2003; 42(3):269–278. [PubMed: 12595779]
24. Turner HA, Finkelhor D, Ormrod R. The effect of lifetime victimization on the mental health of children and adolescents. Social Science and Medicine. 2006; 62:13–27. [PubMed: 16002198]
25. Koenen, KC.; Roberts, AL.; Stone, DM.; Dunn, EC. The epidemiology of early childhood trauma. In: Lanius, RA.; Vermetten, E.; Pain, C., editors. The hidden epidemic: The impact of early life trauma on health and disease. New York, NY: Cambridge University; 2010. p. 13-24.
26. U.S. Department of Health and Human Services, Administration on Children YaF. Child Maltreatment (2006). Washington, DC: U.S. Government Printing Office; 2008.
27. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: Conceptuali, strategi, and statistical considerations. Journal of Personality and Social Psychology. 1986; 51(6):1173–1182. [PubMed: 3806354]
28. Kraemer HC, Stice E, Kazdin A, Offord D, Kupfer D. How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. American Journal of Psychiatry. 2001; 158(6):848–856. [PubMed: 11384888]
29. Nolen-Hoeksema S. Responses to depression and their effects on the duration of depressive episodes. Journal of Abnormal Psychology. 1991; 100:569–582. [PubMed: 1757671]
30. Leadbeater BJ, Kuperminc GP, Blatt SJ, Hertzog C. A multivariate model of gender differences in adolescents' internalizing and externalizing problems. Developmental Psychology. 1999; 35(5): 1268–1282. [PubMed: 10493653]
31. Slopen N, Williams DR, Fitzmaurice GM, Gilman SE. Sex, stressful life events, and adult onset depression and alcohol dependence: Are men and women equally vulnerable? Social Science and Medicine. 2011; 73(4):615–622. [PubMed: 21782304]
32. Kessler, RC. Gender differences in major depression: Epidemiological findings. In: Frank, E., editor. Gender and its effects on psychopathology. Washington, DC: American Psychiatric Press; 2000. p. 61-84.
33. Fergusson DM, Swain-Campbell NR, Horwood LJ. Does sexual violence contribute to elevated rates of anxiety and depression in females? Psychological Medicine. 2002; 32:991–996. [PubMed: 12214797]

34. Kendler KS, Thornton LM, Prescott CA. Gender differences in the rates of exposure to stressful life events and sensitivity to their depressogenic effects. American Journal of Psychiatry. 2001; 158:587–593. [PubMed: 11282693]
35. Gershon A, Minor K, Hayward C. Gender, victimization, and psychiatric outcomes. Psychological Medicine. 2008; 28:1377–1391. [PubMed: 18387212]
36. Schilling EA, Aseltine RH, Gore S. Adverse childhood experiences and mental health in young adults: A longitudinal study. BMC Public Health. 2007; 7(30)
37. Nelson EC, Heath AC, Madden PA, Cooper ML, Dinwiddie SH, Bucholz KK, Glowinski A, McLaughlin T, Dunne MP, Statham DJ. and others. Association between self-reported childhod sexual abuse and adverse psychosocial outcomes: Results from a twin study. Archives of General Psychiatry. 2002; 59:139–145. [PubMed: 11825135]
38. Pimlott-Kubiak S, Corina KM. Gender, victimization, and outcomes: Reconcepualizing reisk. Journal of Consulting and Clinical Psychology. 2003; 71:528–539. [PubMed: 12795576]
39. MacMillan HL, Fleming JE, Streiner DL, Lin E, Boyle MH, Jamieson E, Duku EK, Walsh CA, Wong MY, Beardslee WR. Childhood abuse and lifetime psychopathology in a community sample. American Journal of Psychiatry. 2001; 158:1878–1883. [PubMed: 11691695]
40. Buckner JC, Beardslee WR, Bassuk EL. Exposure to violence and low-income children's mental health: Direct, moderated, and mediated relations. American Journal of Orthopsychiatry. 2004; 74:413–423. [PubMed: 15554803]
41. Buka SL, Stichick TF, Birdthistle I, Earls FJ. Youth exposure to violence: prevalence, risks, and consequences. American Journal of Orthopsychiatry. 2001; 71(3):298–310. [PubMed: 11495332]
42. Maniglio R. Child sexual abuse in the etiology of depression: A systematic review of reviews. Depression and Anxiety. 2010; 27:631–642. [PubMed: 20336807]
43. Costello EJ, Foley DL, Angold A. 10-year research update review: The epidemiology of child and adolescent psychiatric disorders: II. developmental epidemiology. Journal of the American Academy of Child and Adolescent Psychiatry. 2006; 45(1):8–25. [PubMed: 16327577]
44. Ben-Shlomo Y, Kuh D. A life course approach to chronic disease epidemiology: conceptual models, empirical challenges and interdisciplinary perspectives. Int J Epidemiol. 2002; 31(2):285–293. [PubMed: 11980781]
45. Halfon N, Hochstein M. Life course health development: an integrated framework for developing health, policy, and research. Milbank Q. 2002; 80(3):433–479. iii. [PubMed: 12233246]
46. Hertzman C, Power C. Health and human development: understandings from life-course research. Dev Neuropsychol. 2003; 24(2–3):719–44. [PubMed: 14561568]
47. Kessler RC, Chiu WT, Demler O, Walters EE. Prevalence, severeity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry. 2005; 62:617–627. [PubMed: 15939839]
48. Kessler RC, Berglund P, Chiu WT, Demler O, Heeringa S, Hiripi E, Jin R, Pennell B, Walters EE, Zaslavsky A. and others. The US national comorbidity survey replication (NCS-R): Design and field procedures. International Journal of Methods in Psychiatric Research. 2004; 13(2):69–92. [PubMed: 15297905]
49. Kessler RC, Merikangas KR. The national comorbidity survey replication (NCS-R): background and aims. International Journal of Methods in Psychiatric Research. 2004; 13(2):60–68. [PubMed: 15297904]
50. Wittchen HU. Reliability and validity studies of the WHO--Composite International Diagnostic Interview (CIDI): a critical review. Journal of Psychiatric Research. 1994; 28(1):57–84. [PubMed: 8064641]
51. Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Division of Adolescent and School Health;, Health Resources and Services Administration, Maternal and Child Health Bureau, Office of Adolescent Health, National Adolescent Health Information Center, University of California SF. Executive Summary—Improving the Health of Adolescents & Young Adults: A Guide for States and Communities. Atlanta, GA: 2004.
52. Cox DR. Regression models and life-tables. Journal of the Royal Statistical Society, Series B (Methodological). 1972; 34(2):187–220.

53. Singer, JD.; Willett, JB. Applied longitudinal data analysis. New York, NY: Oxford Press; 2003.

54. Rose, G. The strategy of preventive medicine. New York: Oxford University Press; 1992.

55. Leach LS, Christensen H, Mackinnon AJ, Windsor TD, Butterworth P. Gender differences in depression and anxiety across the adult lifespan: The role of psychosocial mediators. Social Psychiatry and Psychitric Epidemiology. 2008; 43:983–998.

56. Trickett PK, Noll JG, Putnam FW. The impact of sexual abuse on female development: Lessons from a multigenerational, longitudinal research study. Development and Psychopathology. 2011; 23(2):453–476. [PubMed: 23786689]

Table 1

Prevalence of interpersonal violence exposure among females and males (N=5498)

	Total			Gender						
				Females (n=3191)			Males (n=2307)			
Exposure by Age 18	**Number**	**%**	**(s.e.)**	**Number**	**%**	**(s.e.)**	**Number**	**%**	**(s.e.)**	**χ^2**
Physical Abuse	322	5.81	0.31	182	6.61	0.47	140	5.37	0.47	1.42
Sexual Abuse	649	11.71	0.56	533	18.14	0.81	116	4.46	0.47	251.46***
Rape	472	8.53	0.60	417	14.19	1.11	56	2.15	0.33	142.71***
Witnessed Violence	731	13.20	0.68	429	14.61	0.84	302	11.61	0.91	7.24**
Exposure to Any Interpersonal Violence	1468	26.48	1.23	1008	34.33	1.80	460	17.64	1.12	88.43***

Cell entries, which came from the person-level dataset, are weighted number of respondents, weighted percent of exposure (column percent), standard errors, and Rao-Scott χ^2 statistics.

* p<0.05 level

** p<0.01 level

*** p<0.001 level

Table 2

Results of discrete-time survival models testing the immediate and long-term risk associated with exposure to violence on gender differences in adolescent-onset depression

	Model 1	Model 2	Model 3	Model 4
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Physical Abuse				
Female	1.51 (1.24, 1.83)			1.48 (1.21, 1.80)
Past Year		7.81 (3.21, 19.01)	3.55 (1.46, 8.64)	7.44 (3.04, 18.26)
Years After Exposure		2.20 (1.46, 3.33)	1.00	2.18 (1.45, 3.27)
No exposure		1.00		1.00
Sexual Assault				
Female	1.51 (1.24, 1.83)			1.28 (1.03, 1.58)
Past Year		4.37 (2.65, 7.19)	1.30 (0.74, 2.28)	4.03 (2.44, 6.67)
Years After Exposure		3.37 (2.54, 4.46)	1.00	3.12 (2.33, 4.18)
No exposure		1.00		1.00
Rape				
Female	1.51 (1.24, 1.83)			1.32 (1.07, 1.64)
Past Year		6.97 (4.04, 11.86)	2.29 (1.25, 4.19)	6.24 (3.63, 10.72)
Years After Exposure		3.05 (2.21, 4.20)	1.00	2.78 (1.97, 3.90)
No exposure		1.00		1.00
Witnessing Violence				
Female	1.51 (1.24, 1.83)			1.46 (1.19, 1.78)
Past Year		4.97 (2.55, 9.71)	2.75 (1.31, 5.80)	4.80 (2.46, 9.37)
Years After Exposure		1.81 (1.42, 2.30)	1.00	1.75 (1.37, 2.34)
No exposure		1.00		1.00

Cell entries are adjusted exponentiated beta coefficients (OR) and 95% confidence intervals. All models contained controls for age, parental education (highest level attained by either mother or father; 1=less than high school; 2=high school; 3=college; 4=missing response), as a measure of socioeconomic status in childhood, and self-reported race/ethnicity (1=hispanic; 2=black; 3=other; 4=white). Female coded 0=male, 1=female. Past year refers to exposure to violence in the past year. Years after exposure refers to exposure to violence any time beyond the past year. No exposure refers to no exposure to violence reported during any time period.