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The Mediating Effect of Depressive Symptoms on the Relationship between Traumatic Childhood Experiences and Drug Use Initiation

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Abstract

Stressful experiences such as childhood trauma and depressive symptoms have both been implicated in the initiation of drug use; however, longitudinal designs have not yet been used to elucidate their respective roles to better understand the causal sequence. In the present study, a sensitivity analysis was conducted using two mediation strategies to examine how this sequence may differ by various levels of statistical control, including (1) the standard mediational model in which the effect of lifetime traumatic stressors (Year 1) on the onset of drug use (Years 3 and 4) is mediated by levels of depressive symptoms (Year 2); and (2) a stronger test of causality such that the effect of lifetime traumatic stressors (Year 1) on the onset of drug use (Years 3 and 4) was mediated by changes in depressive symptoms (Year 1 to 2), measured by a residualized change score that controlled for levels in Year 1. Two types of trauma were studied in a community-based study of 489 Hispanic preadolescents (aged 10–12): (a) the number of lifetime traumatic stressors and (b) seven specific lifetime stressors. We also controlled for new onset traumatic stressors occurring between Years 1 and 2. Primary findings indicate that drug use initiation during early adolescence (e.g., ages 14–16) may not be tied to immediate proximal perturbations in risk factors, such as traumatic experiences and depressive symptoms. Rather, the effects of trauma on depression in this sample appear to be established earlier in childhood (ages 10–14 or younger) and persist in a relatively stable manner into middle adolescence when the risk for drug use may be heightened.

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Contributors

Drs. Fishbein and Krebs designed the study and wrote the protocol. Dr. Novak conducted the bulk of the data analyses and wrote up the results. All three contributed substantively to the writing of the manuscript. Ms. Warner processed the data, created the dataset, conducted preliminary analyses, and performed quality control checks. Dr. Hammond assembled the test battery, trained and supervised field staff, and contributed to the literature review.

Conflict of Interest

All authors declare that they have no conflicts of interest.

Keywords

drug use; youth; childhood trauma; depression; mediation; prevention

1. Introduction

Childhood adversity and trauma have been consistently related to increased risk for early use of illicit substances (Gordon et al., 2002) and other forms of psychopathology with potentially longstanding consequences (Ackerman, Newton, McPherson, Jones, & Dykman, 1998; Harrison et al., 1997; Schaaf & McCanne, 1998; Simpson & Miller, 2002). Although these relations are strong and consistent, many children who experience childhood adversity or trauma do not eventually use substances, suggesting that the presence of certain individual characteristics may be predisposing or that other factors account for the relationship. Depressive symptoms have been implicated as possible mediators of this causal chain. Although there is a very high rate of co-occurrence between depression and both trauma exposure and substance use (Crum et al., 2008; De Bellis, 2002), not all children may develop depression or engage in substance use in response to trauma. Such findings suggest that these relationships may be more complex and involve variations in nature of exposure, the timing of exposure, and the mediating role of depressive symptoms on initiation of drug use.

Although no single hypothesized pathway will likely explain the complex relationship between childhood trauma, depressive symptoms, and substance use, prospective longitudinal studies remain the gold standard by which functional relationships and causal pathways can be elucidated. However, the temporal ordering in this mediational pathway remains unclear even in panel studies due to a lack of model specifications for timing of the traumatic exposure, its corresponding effect on changes in the mediational factor of depressive symptoms, and how these changes are in turn linked to onset of substance use. The current investigation addresses these considerations by using a sensitivity analysis to estimate the mediational chain under various conditions, types of confounding related to the timing of exposure to trauma, the type of event, and degree to which depression is changed in response to traumatic exposure. Such an analysis may reveal the complexities of these relations and their sensitivity to the nature and timing of exposure to trauma and depressive symptoms.

2.1 Methods

The present study examined data from the Cicero Youth Development (CYD) Project (Fishbein et al., 2009). The primary purpose of the CYD Project was to use a prospective longitudinal panel research design to generate data on the patterns of substance use and identify characteristics that distinguish users and non-users of inhalants and other drugs. The community from which participants were sampled is largely Hispanic with over 70% of students classified as low-income, and overall test scores at or slightly above the district averages. To better understand the antecedent factors in initiation, participants were recruited prior to the expected age of drug use onset, 10–12 years old, and only drug naïve subjects at baseline were included.

2.1.1. Sample Recruitment and Survey Procedure

The Cicero School District provided full contact information for all the 4th, 5th, and 6th grade students enrolled in the target schools in the Spring of 2004. Letters were mailed and telephone calls placed to the homes of sampled dyads (parent/guardian and child). Once contact was made with a primary caregiver, the project was explained, and an appointment

in the home was scheduled to obtain consent. Of the 658 dyads who were fielded and locatable, 553 (84%) participated, 98 (15%) refused, and 7 (1%) were ineligible or incapable of participating. Excluding participants who initiated drug use prior to the study or in the first two years of data collection, the resulting eligible sample included 483 respondents. The interview instruments were administered by trained research associates in participants' homes in English and Spanish (68.3% and 31.7% of youth interviews, respectively) using laptop computers and computer-assisted personal interviewing (CAPI) technology.

2.2 Measures

2.2.1. Substance use

Substance use was measured each year. Respondents were first asked separate questions regarding any use of a variety of illicit substances, including marijuana, cocaine, crack, heroin, stimulants, sedatives, and multiple forms of inhalants. Respondents who answered affirmatively to any one of those questions (only reported after the first two years of the study given exclusions) were asked a series of substance-specific questions regarding the recency and frequency of their use of each substance (e.g., "How long has it been since you used...?"). Respondents reporting use more than 12 months ago who also reported use during the previous year were coded 0 at the current time point because they had not used substances within the prior 12 months.

2.2.2. Independent variables: Trauma and Depression

Traumatic experiences involving exposure to violence, emotional abuse, injury, and death of a close relation (e.g., family member) were assessed in each year using a 12-item scale excerpted from Early Trauma Inventory (ETI; Bremner et al. 2000)¹. Questions such as "Have you ever seen violence, like someone being beaten, in your neighborhood or at a friend's house?" were summed, with higher scores indicating higher levels of traumatic experiences. Exploratory and confirmatory factor analyses for dichotomous indicators revealed that a single solution with 7 items (shown in Table 1) fit the data best (Chi-Square=19.87, 13df, $p=.09$, CFI=.95, TLI=.94, RMSEA=.03). These seven items were summed to create a composite score representing the number of lifetime events that had occurred in the youth's lifetime up until baseline, the Year 1 measurement. We also examined the influence of each specific event in the mediational pathway linking traumatic events to drug use initiation. To control for the possibility that new events occurring between Years 1 and 2 would confound the effect of baseline traumatic events on depressive symptoms and substance use, we also included binary covariates that represented the influence of new events that occurred for each outcome in the mediational model (i.e., depressive symptoms and drug use).

Depressive symptoms were measured using the brief self-report Children's Depression Inventory (CDI; Kovacs 1992), a 27-item summed scale (Cronbach's $\alpha = 0.70$). For each item, respondents select the statement that best describes his/her feelings for the past two weeks.

2.3 Analyses

The present study sought to test the overall hypothesis that depressive symptoms mediate the relationship between childhood trauma and drug use using a structural equation modeling approach, estimated within Mplus (Release 5.1, Muthén and Muthén, 2009). Most mediational research involving panel data of at least three years typically defines the causal pathway using a single exogenous factor (X), a single mediator (M), and a single outcome (Y), each of which are lagged by measurement occasion. Applying this approach, Figure 1 illustrates that the effect of traumatic stressors during the lifetime (Year 1) on the initiation

of drug use (measured by no use at Years 1 and 2 and initiation of first use of any drug at Years 3 or 4) is mediated by the level of depressive symptoms at Year 2. This approach of using standard lagged mediation models is preferable to cross-sectional analyses because the temporal ordering of the measurements allows for a stronger test of causality. However, recent methodological work (MacKinnon, 2008) has argued that examining changes in the mediator that occur after the measurement of the exogenous factor (X) provides a much stronger test of causality than the lagged model for two reasons: (a) it better establishes temporal ordering because the magnitude of change is directly measured after the occurrence of the exogenous predictor, and (b) it uses intra-individual change to remove static differences among individuals, thus allowing each person to serve as their own case and control (i.e., within-subject change) rather than relying solely on differences between individuals (i.e., between subject change) (MacKinnon, 2008).

Given these considerations, we chose to conduct a sensitivity analysis and compare the estimates from both standard lagged mediation and change score models. We estimated the change score model (Shown in Figure 2) such that traumatic events measured at Year 1 were used to predict changes in depressive symptoms from Year 1 to 2, which in turn were used to predict the onset of drug use at Years 3 or 4. To estimate the change score, we used a residualized change score methodology, which represents the residualized difference between the observed Year 2 value of depressive symptoms and the predicted value. The predicted value was generated from a regression model in which the Year 1 value of depressive symptoms is used as the sole predictor. This strategy is favorable to a simple difference score approach or an ANCOVA model because it captures change between years using a single variable while also controlling for levels of the variable at baseline (MacKinnon, 2008).

The mediational effect was computed as the product between two paths: Path a – traumatic stress and depressive symptoms, and Path b – the effect of depressive symptoms and the onset of drug use. The direct effect of traumatic events on drug use initiation (Path c') and the mediated effect (Path ab) are used as independent variables in regression models with a dichotomous outcome (i.e., drug use initiation). The resulting path coefficients are estimated within Mplus using Weighted Least Squares and are on the probit scale. The percentage of the total effect of traumatic events on drug use initiation can be computed by dividing the estimate of the mediated effect (Path ab) by the total effect (sum of Path c' and Path ab), multiplied by 100. We used the bias-corrected bootstrap method ($n=5,000$ resamples) to compute the standard errors of the mediated effect. This approach increases power and reduces bias in the width of the confidence intervals around the mediated path (Williams and MacKinnon, 2008). In both the standard lagged mediation and change score models, we included covariates representing the effect of new traumatic events. The binary covariate (0=no new stressors from Year 1 to Year 2, 1=any new stressors from Year 1 to Year 2) was also correlated with both the mediator (depressive symptoms) and the outcome (drug use initiation). Comparing these effects to the effect of the lifetime traumatic events measured at baseline (Year 1) allows us to examine how distal (Year 1) and proximal (new onset) stressors differentially influence depressive symptoms and drug use. The regression coefficients from this model are on the probit scale, but were transformed to estimate the probability of onset.

Overall, we estimated 16 separate mediational models ($8 \times 2 = 16$) in which eight different types of traumatic events (i.e., 1 model for cumulative number of events and 7 specific event models) were examined in the context of both the standard mediational model (Figure 1) and the change score model (Figure 2). We estimated separate models for each specific traumatic stressor to avoid collinearity (see Section 2.2.2). The full mediation models were estimated within a structural equation framework within Mplus software (Release 5.1,

Muthén and Muthén, 2009). We also used the Full Information Maximum Likelihood (FIML) option within Mplus to handle the missingness at random (MAR) data pattern of the current study. As age would found to be related to attrition in preliminary analyses, controlling for age in the estimated models within the FIML approach allows the inferences to remain unbiased to the effect of attrition. An additional benefit of the bootstrap method is that it adjusts the standard errors for the variability due FIML estimation. All statistically significant results are reported at the $p < .05$ level.

2.4 Results

2.4.1. Descriptive Analyses

Table 1 presents the frequencies of the key study measures. Overall, approximately 12% of the sample had initiated any type of drug use during Years 3 or 4 of the study, corresponding roughly to the ages of 14 to 16 years old. This figure is slightly higher than estimates from the National Survey of Drug Use and Health (NSDUH) in which the prevalence of initiation for the comparable age range and types of drugs is approximately 8% (SAMHSA, 2006). In terms of traumatic stressors, the mean number of stressors was 1.7, whereby 25% reported no stressors at baseline during their lifetime, and 25% had one stressor. Approximately 50% had multiple or two or more stressors and a small percentage (5%) had five or more stressors. The most commonly reported traumatic stressor was 'being extremely scared by a family member because of screaming/yelling/anger' (37%), followed by violence in the neighborhood (34%) and serious accident or injury (25%). Between the first two years of the study, approximately 32% reported at least one new additional traumatic stressor. The range for depressive symptoms was between 0 and 31, with the average level at Year 1 and Year 2 being 7.8 and 6.9, respectively.

2.4.2. Mediational Analyses for Number of Traumatic Stressors

Table 2 displays the estimates of the direct paths linking traumatic stressors to depressive symptoms (Path a), depressive symptoms to the onset of drug use (Path b), and the introductions of new traumatic stressors (Year 1 to 2) on the endogenous outcomes of depressive symptoms and drug use. In Model 1, the number of lifetime traumatic stressors at Year 1 was positively associated with both depressive symptoms and the onset of drug use. The interpretation of these coefficients is such that for every increase in the number of traumatic stressors, there is a corresponding increase in the number of depressive symptoms by a factor of about 1.07. With respect to the relationship between trauma and drug use, recall that the when the outcome is dichotomous (e.g., drug use), the predictors are on the probit scale. Accordingly, we found that the addition of each traumatic stressor results in an increase of 0.11 on the probit scale of initiating drug use in Years 3 or 4. Translating this number to a probability scale yields a probability of onset for 0 stressors ($pr=0.06$), 1 stressors ($pr=0.07$), and 5 stressors ($pr=0.15$). Of interest is that the influence of any traumatic stressors between Years 1 and 2 was uncorrelated with the onset of drug use in Years 3 or 4.

2.4.3. Mediation of the Relationship between Stressors and Drug Use by Depression

In terms of the mediational pathways, we observed that the direct effect paths (a, b, and c') were statistically significant for Model 1. Inspection of the estimates from Model 1 in Table 3 indicates that the estimate of the pathway between lifetime traumatic stressors measured at Year 1 and onset of drug use was significantly mediated by depressive symptoms measured at Year 2. Further, the mediational pathway accounted for about 20% of the total effect ($(path\ ab)/(path\ ab + path\ c')$). In model 2, we utilized a stronger test of mediation by using a model that accounts for the temporal occurrence of primary variables. The level of depressive symptoms at Year 2 was replaced with the residualized change score,

representing the standardized change in depressive symptoms from Year 1 to Year 2 controlling for Year 1 values. Table 1 shows that the statistically significant path from traumatic symptoms to depressive symptoms in Model 1 becomes nonsignificant when changes in depressive symptoms over time are used. Changes in depressive symptoms in Model 2 (Path b) were also unrelated to onset of drug use in Years 3 or 4. Overall, the proportion mediated in Model 2 was much less (4.6%) than Model 1 and nonsignificant. Although traumatic stressors between Years 1 and 2 were unrelated to onset of drug use in Years 3 or 4, stressors were related to a rapid increase in depressive symptoms.

2.4.3. Mediation Analyses for Specific Types of Traumatic Stressors

Tables 2 and 3 show the results from the separate models (Models 2–16) in which each traumatic stressor was examined in relation to the hypothesized mediational pathway involving depressive symptoms and the onset of drug use. In general, traumatic stressors were significantly related to levels of depressive symptoms (Path a, Models 3, 5, 7, 9, 11, 13, 15) measured at Year 2. However, no significant effects were found between each traumatic stressor and changes in depressive symptoms (Path a, Models 4, 6, 8, 10, 12, 14, 16). Similarly, the static measurement of Year 2 levels of depressive symptoms were related to onset of drug use (Path b) for each of these models, but no parallel effect was found for changes in depressive symptoms and drug use. In terms of the mediated effects, only two symptoms exhibited significant mediation. About 36% of the path between the lifetime occurrence of a serious accident and drug use onset was mediated by static levels of depressive symptoms. This effect may be compared with the model showing that 11% of the effect of being extremely scared by a family member. Both stressors were non-significant using the residualized change-score approach, as there was a significant reduction in the amount of the total effect that was mediated through change in depressive symptoms compared to Year 2 levels of depressive symptoms. Similar to the findings for the number of traumatic events, experiencing traumatic stressors between Years 1 and 2 was unrelated to the onset of drug use, though highly correlated with both the levels of and change in depressive symptoms.

3. Discussion

The present study tested the hypothesis that the development of depressive symptoms would at least partially explain the variability in onset of illicit drug use relative to childhood exposure to trauma. To more closely evaluate temporal relationships, a sensitivity analysis was used, comparing standard a lagged mediational model with a change score model. Primary findings indicate that drug use initiation during early adolescence (e.g., ages 14–16) may not be tied to immediate proximal perturbations in risk factors, such as traumatic experiences and depressive symptoms. Rather, the effects of trauma on depression in this sample appear to be established earlier in childhood (ages 10–14 or younger) and persist in a relatively stable manner into middle adolescence when the risk for drug use may be heightened. These findings must be considered in the context of the sample, largely Hispanic and from a single city. However, the study location is highly demographically diverse, so it shares similarities with other large urban areas. Also worth noting is that the inclusion of alcohol use and parent reports of stressor exposure and depressive symptoms would be recommended for future studies.

The present study's focus on depressive symptoms that often arise from perturbations in neural, physiological, and psychological systems in response to stress was a logical extension for the field. Similar to other studies (e.g., Waaktaar et al., 2004), however, our mediational findings did not clearly establish a causal relationship. Hence, the next step is to identify other "intermediate phenotypes" that may develop from stress maladaptations or pre-existing individual characteristics (e.g., genetic factors, sensitivity to psychopathological

consequences of stressful triggers, etc.) to more fully explain the primary relationship. Ultimately, this program of research will advance scientific understanding of risk and resiliency regarding pathways and ultimate outcomes for youth exposed to trauma, thus providing direction for prevention programming.

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Figure 1. Hypothesized Mediation Model Linking Traumatic Stressors, Levels of Depressive Symptoms, and Initiation of Drug Use

Mediated Effect: ab

Direct Effect: c'

*All paths control for new traumatic stressors between Year 1 and 2

Lifetime traumatic stressors measured by (a) cumulative number of lifetime stressors or (b) separate models for each of the 7 specific types of stressors.

Figure 2. Hypothesized Mediation Model Linking Traumatic Stressors, Change in Depressive Symptoms, and Initiation of Drug Use

Mediated Effect: ab

Direct Effect: c'

Change in depressive symptoms measured by residualized change scores from Year 1 to Year 2, controlling for Year 1 levels of depressive symptoms

All paths control for new traumatic stressors between Year 1 and 2

Lifetime traumatic stressors measured by (a) cumulative number of lifetime stressors or (b) separate models for each of the 7 specific types of stressors.

Table 1

Descriptive Statistics for Study Measures (N=489).

Characteristic	Percent (%)	SE
Ever Had Serious Accident or Injury, Yr 1	25.3	1.9
Ever Had Serious Illness Needing Hospitalization, Yr 1	22.1	1.9
Ever Had Serious Injury/Death to Parent, Sibling, or Caregiver, Yr 1	20.1	1.8
Ever Seen Family Members Beaten by Other Family, Yr 1	15.8	1.6
Ever Seen Violence in Neighborhood or Friend's House, Yr 1	34.5	2.1
Ever Seen a Weapon Used or Threatened by Weapon, Yr 1	14.1	1.5
Ever Scared by Family Member Scream/Yelling/Anger, Yr 1	37.8	2.2
Onset of New Traumatic Stressors (TS), Yr 1 to 2 ($\Delta TS_{Yr 1 to 2}$)	31.6	2.1
Initiation of Drug Use, Yr 3 or 4	12.2	1.5
Characteristic	Mean	SE
Number (#) of Traumatic Stressors, Yr 1	1.7	0.1
Level of Depressive Symptoms, Yr 1	7.8	0.3
Level of Depressive Symptoms, Yr 2	6.9	0.3
Change in Depressive Symptoms, Yr 1 to 2	-0.01	1.28

Note: Yr=Year of Measurement; SE=Standard Error. Change in depressive symptoms measured by residualized change score, controlling for year 1 levels of depressive symptoms.

Table 2

Path Model Estimates between Traumatic Stress (TS), Depressive Symptoms (DEP), and Onset of Drug Use.

Type of Traumatic Stress (Year 1)	Model #	Mediator (Dep)	Path a Coeff SE	Path b Coeff SE	Path c' Coeff SE	$\Delta TS_{Yr 1 to 2} \rightarrow DEP$ Coeff SE	$\Delta TS_{Yr 1 to 2} \rightarrow Drug$ Coeff SE
# of Traumatic Stressors	1	Level at Yr 2	1.07 .19	0.03 .01	0.11 .05	2.71 .55	0.04 .18
	2	Δ Yr 1 to 2	0.30 .18	0.22 .01	0.13 .05	1.91 .49	0.08 .18
Serious Accident	3	Level at Yr 2	1.79 .65	0.03 .01	0.10 .17	2.27 .96	0.22 .26
	4	Δ Yr 1 to 2	0.67 .56	0.02 .01	0.14 .18	1.68 .86	0.26 .26
Serious Illness	5	Level at Yr 2	1.65 .70	0.03 .01	0.08 .18	0.95 .93	-0.52 1.2
	6	Δ Yr 1 to 2	1.06 .64	0.02 .01	0.10 .18	0.73 .79	-0.52 1.2
Injury/Death of Parent/Sibling/Caregiver	7	Level at Yr 2	1.37 .68	0.03 .01	0.22 .18	2.25 1.07	0.23 .25
	8	Δ Yr 1 to 2	1.57 .93	0.03 .01	0.27 .18	-0.18 .57	0.26 .25
Seen Family Members Beat or Attacked	9	Level at Yr 2	2.37 .65	0.03 .01	0.25 .20	3.68 1.08	.13 .31
	10	Δ Yr 1 to 2	1.14 .60	0.02 .01	0.29 .20	2.99 0.85	.18 .31
Seen Violence in Neighborhood/Friend's House	11	Level at Yr 2	1.13 .57	0.03 .01	0.15 .16	1.68 0.68	-0.08 .24
	12	Δ Yr 1 to 2	-0.29 .48	0.03 .02	0.19 .16	1.15 0.66	-0.06 .24
Seen Weapon Used/Threatened by Weapon	13	Level at Yr 2	1.63 .72	0.03 .01	0.14 .22	2.75 0.99	0.19 .36
	14	Δ Yr 1 to 2	0.47 .71	0.03 .02	0.18 .22	2.01 0.82	0.23 .36
Scared by Family Member	15	Level at Yr 2	2.30 .56	0.03 .01	0.46 .17	2.74 0.82	0.51 .23
	16	Δ Yr 1 to 2	0.62 .48	0.02 .01	0.51 .17	2.17 0.71	0.54 .23

Note: Each model adjusts for concurrent effects of new onset traumatic events. Δ Yr 1 to 2=Residualized change in depressive symptoms from year 1 to year 2, controlling for year 1 value. SE=standard error. CI=95% Confidence Interval. Path A: Direct effect from stressors to depressive symptoms. Path B: Direct effect from depressive symptoms to onset of substance use by year 3 or 4. Path C': Direct effect from traumatic stressors to onset of substance use by year 3 or 4. $\Delta TS_{Yr 1 to 2}$ =New onset traumatic events. All direct paths to onset of drug use on the Probit scale. *P-value<.05 in **BOLD**

Table 3

Estimates of Mediated Effects (Path ab).

Type of Traumatic Stress (TS)	Model #	Depressive Symptoms (Dep)	Mediation (Path ab)	SE _{ab}	Percent Mediated
Number of Traumatic Events	1	Level at Yr 2	.029	.014	20.2%
	2	Δ Yr 1 to 2	.006	.006	4.6%
Serious Accident	3	Level at Yr 2	.058	.029	36.2%
	4	Δ Yr 1 to 2	.015	.018	9.4%
Serious Illness	5	Level at Yr 2	.055	.029	25.2%
	6	Δ Yr 1 to 2	.026	.022	23.1%
Injury/Death Parent/Sibling/Caregiver	7	Level at Yr 2	.043	.028	16.3%
	8	Δ Yr 1 to 2	.005	.017	1.8%
Seen Family Members Beaten	9	Level at Yr 2	.073	.039	22.6%
	10	Δ Yr 1 to 2	.026	.024	8.2%
Seen Violence in Neighborhood/Friend's House	11	Level at Yr 2	.037	.024	20.1%
	12	Δ Yr 1 to 2	.008	.015	5.1%
Seen Weapon Used/Threatened	13	Level at Yr 2	.053	.033	26.9%
	14	Δ Yr 1 to 2	.012	.022	6.3%
Scared by Family Member	15	Level at Yr 2	.060	.030	11.5%
	16	Δ Yr 1 to 2	.012	.014	2.3%

Note: Each model adjusts for concurrent effects of new onset traumatic events, Δ Yr 1 to 2=Residualized change in depressive symptoms from year 1 to year 2, controlling for Year 1 value. SE=standard error. CI=95% Confidence Interval. Mediated path (ab) is on the Probit scale and computed by Delta Method with Bootstrap standard errors (5,000 replicates). *P-value<.05 in **BOLD**