Adult trauma and adult symptoms: Does childhood trauma drive the relationship?

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INTRODUCTION
The current paper attempts to use structural modeling to explain observed relationships between childhood trauma, adult trauma, and adult dissociation and mental health. Many researchers have noted these relationships without attempting to piece apart the nature of the relationships, only assuming that all variables might have causal effects on all others (with the exception of adult variables causing childhood variables). Theoretically, if childhood trauma causes revictimization (adult interpersonal trauma) as well as dissociation and mental health problems, a model specifying paths to revictimization, dissociation, and mental health beginning at childhood trauma should best fit observed data. Conversely, if childhood trauma does not cause these outcomes, and underlying characteristics of the individual cause both traumatization and mental health problems, a model that leaves out paths from adult trauma to outcome variables should not fit the data well. It is hypothesized that a model specifying paths from childhood trauma to adult trauma, dissociation, and mental health, and setting paths originating at adult trauma to zero will most parsimoniously predict observed patterns of association between these variables.

METHODS
The current study surveyed 307 (198 women, 97 men, 2 declined to report gender) undergraduates recruited from the University of Oregon psychology Human Subjects Pool. Participants were compensated with partial course credit. Demographics in this sample were representative of the UO undergraduate population with a mean age of 20.96 years (SD = 4.89), mostly (94%) single, and ethnicity represented as follows: 85.1% Caucasian, 9.3% Asian American, 2.0% Hispanic/Latino/a, 11% other groups.

RESULTS
Upon completing a thorough check of the data’s suitability, the hypothesized model was run in Mplus. The model was specified as follows. Scores from the DES and the TSC dissociation subscale were included as indicators of the latent construct dissociation. Scores from the TSC depression subscale and TSC anxiety subscale were included as indicators of the latent variable mental health. Childhood trauma was included as a predictor of adult trauma and dissociation, and dissociation was included as a predictor of mental health. Mental health was in turn included as a predictor of adult trauma experiences. Paths between adult betrayal trauma and dissociation, and dissociation and mental health trauma were set at zero. This hypothesized model fit the data very well. The comparative fit index (CFI) was .98, and the chi-square test of model fit was 23.31 (df = 7, N = 307, p = .001). Standardized parameter estimates indicated significant relationships for all estimated paths. See the figure below for a visual representation of the model with standardized path estimates. A more complex model in which the hypothesized model is nested includes direct paths between adult trauma and dissociation, and child trauma and mental health. This model would also fit the data very well, with a CFI of .98. Although these findings do not provide a measure of the degree to which the hypothesized model fits the data better than this more complex model, the chi-square test statistic for the difference between the two models was no longer significant. It is quite possible that the relationship between childhood trauma and mental health could be represented by a single direct path coefficient. However, this model is consistent with a body of research supporting the idea that childhood trauma creates a diathesis, possibly through dissociation, and is itself a stress. It is entirely possible that many diathesis-stress models could be reframed as early stress-later stress models.

CONCLUSIONS
The analyses conducted provide some evidence to support the notion that childhood trauma is responsible for the bulk of the variance in dissociation and mental health that can be attributed to trauma. Adult trauma experiences are significantly related to dissociation when controlling for the effects of childhood trauma, and may have a relationship with mental health such that adult mental health symptoms make adult trauma more likely. Overall, the hypothesized model is preferable over the full model because it provides a more parsimonious explanation of the data without significantly deteriorating. It is somewhat unclear, however, whether equivalent models that specify different relationships between trauma and the latent constructs might be just as likely. There is not strong enough theoretical grounding one way or another to make this determination.

These data do clearly suggest that childhood trauma is the driving force behind the relationship between trauma and dissociation. Given that most reports are retrospective, and experiencing childhood trauma is correlated with experiencing adult trauma, it is often difficult to determine the nature of observed relationships. However, the model supported by the current analyses has important implications for diathesis-stress models of psychopathology. Frequently, it is assumed by diathesis-stress researchers that underlying vulnerabilities toward developing psychopathology are biological or genetic in nature. Many researchers talk about a cognitive vulnerability to depression or anxiety, but do not discuss how the vulnerability is acquired, leaving the reader to assume that it is somehow inherent. However, this paper is consistent with a body of research supporting the idea that childhood trauma creates within a person a diathesis, possibly through dissociation, and is itself a stress. It is entirely possible that many diathesis-stress models could be reframed as early stress-later stress models.

REFERENCES