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Comparison of Three Models of Adverse Childhood Experiences: Associations With Child and Adolescent Internalizing and Externalizing Symptoms

Lauren M. Henry[1], Kathy Gracey[2], April Shaffer[2], Jon Ebert[2], Tarah Kuhn[2], Kelly H. Watson[1], Meredith Gruhn[1], Allison Vreeland[1], Rachel Siciliano[1], Lindsay Dickey[1], Victoria Lawson[1], Cassandra Broll[1], David A. Cole[1], and Bruce E. Compas[1]

[1] Department of Psychology and Human Development, Vanderbilt University
[2] Department of Psychiatry, Vanderbilt University Medical Center

Exposure to adverse childhood experiences (ACEs) is prevalent and confers risk for psychopathology later in life. Approaches to understanding the impact of ACEs on development include the independent risk approach, the Dimensional Model of Adversity and Psychopathology (DMAP) distinguishing between threat and deprivation events, and the cumulative risk approach. The present research provides an empirical confirmation of DMAP and a comparison of these three approaches in predicting internalizing and externalizing symptoms in youth. In Study 1, mental health professionals ($N = 57$) rated ACEs as threat or deprivation events. These ratings were used to create composites to represent the DMAP approach in Study 2. With cross-sectional and longitudinal data from children and adolescents in state custody ($N = 23{,}850$), hierarchical linear regression analyses examined independent risk, DMAP, and cumulative risk models in predicting internalizing symptoms, disinhibited externalizing symptoms, and antagonistic externalizing symptoms. All three approaches produced significant models and revealed associations between exposure to ACEs and symptoms. Individual risk accounted for significantly more variance in symptoms than cumulative risk and DMAP. Cumulative risk masked differential associations between ACEs and psychological symptoms found in the individual risk and DMAP approaches.

General Scientific Summary

This study suggests that ACEs are not homogeneous in the risk that they confer for psychopathology. However, the preferential approach for operationalizing ACEs may be goal dependent. Entering ACEs simultaneously into a model using an individual risk approach may identify youth at greatest risk for developing psychopathology, whereas grouping ACEs theoretically (e.g., DMAP) can advance research on mechanisms of risk.

Keywords: adverse childhood experiences, childhood and adolescence, psychopathology

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Adverse childhood experiences (ACEs) may include physical, sexual, and emotional abuse; physical and emotional neglect; exposure to natural disaster; medical trauma; parental death; and caregiver impairment attributable to psychopathology, substance abuse, and criminal behavior (Felitti et al., 1998). ACEs are highly prevalent in the lives of children and adolescents. For example, in 2014, an estimated 702,000 children in the United States were victims of abuse or neglect (U.S. Department of Health & Human Services, 2016). Further, ACEs are powerful predictors of psychopathology across the life span (Gilbert et al., 2009; McGrath et al., 2017). Given the prevalence and profound impact of ACEs, identifying youth at greatest risk for developing internalizing and externalizing symptoms is important, and uncovering mechanisms by which maladaptive outcomes occur can provide targets for

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Lauren M. Henry https://orcid.org/0000-0003-0286-9319
April Shaffer https://orcid.org/0000-0003-1855-1954
Tarah Kuhn https://orcid.org/0000-0003-0606-8877
Meredith Gruhn https://orcid.org/0000-0002-5523-8871
Bruce E. Compas https://orcid.org/0000-0002-5884-181X
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Correspondence concerning this article should be addressed to Lauren M. Henry, Department of Psychology and Human Development, Vanderbilt University, 230 Appleton Place, Nashville, TN 37203, United States. Email: Lauren.M.Henry@vanderbilt.edu

intervention. Parsimonious, theory-driven frameworks that organize ACEs by underlying dimensions may contribute to the advancement of these goals. The current study examined three approaches to understanding associations between ACEs and internalizing and externalizing symptoms: individual risk, the Dimensional Model of Adversity and Psychopathology (DMAP; McLaughlin et al., 2014; Sheridan & McLaughlin, 2014), and cumulative risk (Evans et al., 2013; Felitti et al., 1998).

The *individual risk approach* examines ACEs separately as unique indicators of risk for psychopathology. In a large body of research, individual ACEs have been associated with an array of psychological problems and disorders (e.g., Cutajar et al., 2010; Taylor et al., 2018). For example, Fergusson et al. (1996) found that compared with adults reporting no experience of sexual abuse in childhood, individuals with a sexual abuse history were more likely to develop major depression, anxiety disorders, conduct disorder, substance abuse/dependence, and suicidal behavior in adulthood. However, associations between childhood sexual abuse and psychopathology were either reduced or fully accounted for after adjusting for exposure to other ACEs (i.e., parent criminal behavior, parent substance abuse) as well as demographic factors (e.g., gender, ethnicity) and other risk factors.

ACEs tend to co-occur, such that children who have been exposed to one ACE have likely experienced others (Finkelhor et al., 2007; McLaughlin et al., 2012). Examining ACEs separately may underestimate the cumulative potential for ACEs to interfere with normative development and overestimate the associations between particular ACEs and psychological disorders (Mullen et al., 1996; Rutter, 1979, 1981). Therefore, the importance of examining the effects of ACEs simultaneously has been underscored in research (Cecil et al., 2017). Epidemiologic studies examining multiple ACEs as predictors of psychopathology in single models have demonstrated little specificity between individual ACEs and particular disorders (e.g., Green et al., 2010; Kessler et al., 2010).

Grouping ACEs by their underlying characteristics could reveal patterns in the ways that ACEs influence psychopathology. The *DMAP approach* (McLaughlin et al., 2014; Sheridan & McLaughlin, 2014) plots ACEs onto dimensions of *threat* and *deprivation*. McLaughlin et al. (2014) define threat as experiences involving the potential of harm or the experience of actual harm and deprivation as the absence of expected and typical environmental inputs and supports. They posit that exposure to threatening experiences and environmental deprivation influence development in ways that are at least partially distinct, with threat affecting emotion reactivity and regulation and deprivation affecting cognition (McLaughlin et al., 2014). Neurodevelopmental research provides initial support for DMAP, showing that threat and deprivation experiences uniquely impact brain structure and function. Specifically, youth exposed to threat events show reduced volume in the amygdala, medial prefrontal cortex, and hippocampus and heightened activation in the amygdala, whereas youth exposed to deprivation events exhibit reduced volume and altered function in frontoparietal regions (for a review, see McLaughlin et al., 2019). Data-driven approaches have provided early support for the DMAP model, as well. Using network analysis, Sheridan et al. (2020) identified a two-cluster solution consistent with the DMAP theoretical model, such that threat variables (i.e., physical abuse, sexual abuse, exposure to community violence, and exposure to family violence) clustered with performance on an automatic emotion regulation task and deprivation variables (i.e., parent education, access to cognitively stimulating materials and experiences) clustered with cognitive performance tasks in a community sample of children and adolescents. Contrary to expectations, physical neglect clustered with the threat variables.

The DMAP approach does not explicitly hypothesize that threat and deprivation are associated with distinct forms of psychopathology. Rather, DMAP suggests different pathways to psychopathology. However, several studies have shown differences in the associations among threat and deprivation and internalizing and externalizing symptoms. For example, Busso et al. (2017) found threat (i.e., exposure to interpersonal violence) to be associated with greater levels of internalizing symptoms. Threat was indirectly related to more externalizing symptoms through blunted physiological reactivity. Deprivation (i.e., poverty) was associated with more externalizing, but not internalizing, symptoms. In addition, Miller et al. (2018) found threat (i.e., physical abuse and harsh discipline) to be related to increased levels of both internalizing and externalizing symptoms and deprivation (i.e., lack of environmental enrichment) to be related to increased externalizing symptoms through deficits in verbal abilities. Deprivation was not a significant predictor of internalizing symptoms.

Finally, the *cumulative risk approach* sums exposure to individual risk factors to generate a total risk score (Evans et al., 2013; Felitti et al., 1998). Rutter (1979, 1981) was one of the first to observe a relation between cumulative ACEs and development. Subsequently, the seminal articles from the original ACEs study documented a dose-response association between a cumulative index of ACEs and increased risk for psychological and physical illness (Anda et al., 2006; Felitti et al., 1998). Based on these and other studies, the cumulative risk approach has become the most common model for determining risk for psychopathology from exposure to ACEs. Studies applying this approach have directed much needed attention and resources to ACEs research and intervention, and in practice, cumulative risk scores help identify children who are at the greatest risk for poor outcomes for intervention (Shonkoff, 2016). Yet, without considering the type of ACEs included in total risk scores, the cumulative risk approach implicitly assumes that all ACEs are equal and additive in conferring risk for internalizing and externalizing psychopathology and different experiences influence development through the same underlying mechanisms (Evans et al., 2013; Schilling et al., 2008).

In one of the few studies to examine all three approaches, Lambert et al. (2017) compared findings from the individual risk, DMAP, and cumulative risk approaches to understand the relation between ACEs and emotion regulation and cognitive control. Using the individual risk approach, Lambert et al. found exposure to sexual abuse and community violence to be associated with deficits in emotion regulation. Using the DMAP approach, the authors found threat (sum of physical, emotional, and sexual abuse and exposure to community violence) to be associated with deficits in emotion regulation and deprivation (poverty) to be related to deficits in cognitive control. Using the cumulative risk approach, these specific associations were concealed, such that exposure to more ACEs was only associated with reduced cognitive control. Although it was not the study focus, zero-order correlations showed small-to-medium associations between threat variables and anxiety and depression, and no associations between the deprivation variable (poverty) and anxiety and depression.

In sum, researchers have examined ACEs as individual risk factors, classified as threat and deprivation events, and as part of a cumulative index of total number of ACEs. This body of work has begun to document how ACEs affect development, and in doing so, facilitated the identification of youth at risk for developing psychopathology. Although DMAP is a promising framework for organizing the unique effects of ACEs on development, empirical support for this approach is in its early stages. No research has compared the three approaches in predicting internalizing and externalizing symptoms, which might guide research and clinical applications. Through two studies, we provide an empirical classification of a relatively large and diverse group of ACEs as threat and deprivation events, and we compare the individual risk, DMAP, and cumulative risk approaches in predicting internalizing and externalizing symptoms in a large sample of children and adolescents with a range of exposure to ACEs.

In Study 1, we conducted a subject matter expert review of DMAP, such that mental health professionals and researchers rated 10 ACEs categorically as threat or deprivation events. First, we hypothesized that physical abuse, sexual abuse, emotional abuse, medical trauma, natural disaster, witness to family violence, and witness to community violence would be categorized as threat. Second, we hypothesized that neglect, caregiver substance abuse, and caregiver mental illness would be categorized as deprivation. In Study 2, we used the Study 1 classifications to create threat and deprivation composites, and individual risk, DMAP, and cumulative risk approaches were compared as predictors of internalizing and externalizing symptoms in youth. First, using the individual risk approach, we hypothesized that all ACEs would be positively related to both internalizing and externalizing symptoms. Second, using the DMAP approach, we hypothesized that (a) higher threat scores would account for unique variance in internalizing and externalizing symptoms after controlling for levels of deprivation and (b) higher deprivation scores would account for unique variance in externalizing, but not internalizing, symptoms after controlling for levels of threat. Third, we hypothesized that higher cumulative risk scores would predict higher levels of internalizing and externalizing symptoms. Finally, we examined incremental utility of the three approaches in predicting internalizing and externalizing symptoms.

Study 1

Method

Participants

We recruited mental health professionals and researchers in psychology via personal email and postings on psychology and ACEs-related listservs. Included in this sample were faculty at university-based medical centers specializing in the study of complex developmental trauma. Faculty members distributed our survey to their associates, including colleagues within their departments and part of the National Child Traumatic Stress Network. Further, our survey was distributed to Centers of Excellence for Children in State Custody, including psychologists in administrative roles and a group of approximately 30 master's-level clinicians who supervise Child and Adolescent Needs and Strengths (CANS) assessments in their respective regions. Finally, our survey was distributed to more than 90 masters-level clinicians who were part of a yearlong Attachment, Regulation, and Competency Learning Collaborative, most of whom were employed in community outpatient clinics in Middle Tennessee.

Sixty-six individuals completed our survey, which was distributed as a hyperlink to Qualtrics online survey software. The majority of participants (68%) were exclusively in clinical practice or were child welfare professionals. The remaining participants served dual roles as clinical practitioners or child welfare professionals and ACEs researchers (23%), had little or no involvement with youth who had experienced ACEs (6%), or were exclusively involved in ACEs research (3%). Eighty-six percent of participants reported having a high level of knowledge about ACEs, as compared with a little (12%) or none (2%). Considering our goal to recruit experts in ACEs research and practice, we excluded participants with little or no involvement with or knowledge of youth with ACEs (i.e., nonexperts, $n = 9$). There were significant differences between ratings of experts and nonexperts, with nonexperts more likely to rate medical trauma, $t(56) = 3.02$, $p = .004$, natural disaster, $t(56) = 2.57$, $p = .01$, witness to family violence, $t(56) = 3.45$, $p = .001$, and caregiver substance abuse, $t(56) = 3.04$, $p = .01$, as deprivation. In the final sample ($N = 57$), 60% of participants had a master's degree, 39% had a doctoral degree, and 1% had an alternative degree (i.e., Ed.S.).

Procedure

We developed an online survey for the purposes of Study 1 using Qualtrics online survey software. First, participants read Sheridan and McLaughlin's (2014) definitions for threat and deprivation. Threat is "the presence of an atypical (i.e., unexpected) experience characterized by actual or threatened death, injury, sexual violation, or other harm to one's physical integrity," and deprivation is "the absence of expected environmental inputs in cognitive (e.g., language) and social domains as well as the absence of species- and age-typical complexity in environmental stimulation" (Sheridan & McLaughlin, 2014, p. 580). Next, participants classified 10 ACEs (physical abuse, sexual abuse, medical trauma, natural disaster, family violence, community violence, neglect, emotional abuse, caregiver substance abuse, and caregiver mental illness) as threat or deprivation in a forced-choice task. Instructions stated, "If you believe a traumatic experience contains elements of both 'threat' and 'deprivation,' do your best to select a single term that best captures the experience." All procedures were approved by the Vanderbilt University Institutional Review Board.

Results

The majority of participants categorized sexual abuse (100%), physical abuse (98%), natural disaster (90%), medical trauma (86%), witness to family violence (83%), and witness to community violence (83%) as threat. The majority of participants categorized neglect (97%), caregiver substance abuse (86%), caregiver mental illness (79%), and emotional abuse (68%) as deprivation. Thus, all 10 adversities were classified as either threat or deprivation for use in analyses in Study 2. Rates of agreement were somewhat higher for ACEs that were classified as threat (M percent agreement = 91%) than for those classified as deprivation (M percent agreement = 81%), with ratings being the most variable for emotional abuse.

Discussion

Study 1 provided an empirical confirmation of threat and deprivation categories of ACEs for Study 2. Specifically, our subject matter expert review supports the face validity of the DMAP model. Participants' classifications of ACEs as threat and deprivation largely supported hypotheses 1 and 2. Contrary to our hypotheses, participants classified emotional abuse as a deprivation event. In practice, emotional abuse and emotional neglect can be combined into the construct of psychological maltreatment. By definition, psychological maltreatment involves acts of commission (e.g., verbal attacks) and omission (e.g., emotional withdrawal and unresponsiveness; APSAC, 2019; Glaser, 2002). Theoretically, acts of commission would be classified as threat and acts of omission would be classified as deprivation in DMAP (Sheridan & McLaughlin, 2014). Emotional neglect was not included in our forced-choice task, and so participants may have conceptualized the emotional abuse item more generally as psychological maltreatment. To understand the nature of these effects more completely, future empirical confirmations of DMAP should include both emotional abuse and emotional neglect items and measure the degree to which individual ACEs represent aspects of both threat and deprivation.

Participants' classifications of caregiver mental illness and caregiver substance abuse as deprivation were consistent with hypothesis 2. Research suggests that up to half of all individuals meet criteria for a mental disorder in their lifetime (Kessler et al., 2007; Moffitt et al., 2010), and the number of children who will have experienced an ACE by this standard is striking. As discussed by McLaughlin (2016), caregiver substance abuse and mental illness can be conceptualized as *contexts that put children at risk* for the occurrence of ACEs, rather than ACEs themselves. In some situations, a caregiver who is substance abusing or mentally ill may reflect an environment devoid of cognitive inputs and sensory, motor, linguistic, and social experiences for their children (McLaughlin et al., 2017). In other situations, children of substance abusing or mentally ill caregivers may still have access to rich, stimulating environments. It could be useful to characterize these childhood experiences as *adverse* only when substance abuse and mental illness interfere with the capacity to parent. In Study 2, we take this approach. In the scientific community, consensus has not yet been achieved in operationalizing ACEs (McLaughlin, 2016). Agreement on a definition and consistency in its use will further enhance our ability to effectively study childhood adversity.

Study 2

Method

Participants

The sample for Study 2 included 23,850 children and adolescents in state custody in a southeastern state between 2012 and 2017. Youth ranged in age from 5 to 18 years old (M = 12.99, SD = 4.08). They were primarily male (58%) and White (69% White; 23% Black, 6% Multiracial, <1% American Indian/Alaska Native).

Measures

Child and Adolescent Needs and Strengths (CANS; Lyons, 2009) is an assessment tool designed to describe youth and family characteristics comprehensively and concisely, supporting clinical decision-making without labor-intensive scoring procedures (Lyons, 2009). A youth's entrance into state custody triggers the initial administration of the CANS. Typically, youths enter state custody as a consequence of substantiated ACEs or a court order for delinquent and disruptive behaviors. Caseworkers complete the CANS about a youth using their aggregate knowledge from various sources (e.g., the youth, biological parents, foster parents, teachers). To use the tool, an individual must complete training and be certified based on reliability of .70 or greater on a case vignette (Praed Foundation, 2015). For the items used in the current study, interrater reliabilities range from .55 to .98 (Anderson et al., 2003). Research supports predictive validity for clinical decision-making, as well as concurrent and discriminant validity with another commonly used measure in the children's mental health services system (Child and Adolescent Functional Assessment Scale; Dilley et al., 2003; Lyons et al., 2004).

All key variables used in the current study are items, or calculated composites of items, from CANS data. Demographic data (i.e., age, gender, race) were integrated into the de-identified CANS dataset provided by the Department of Children's Services (DCS). CANS items are scored on 4-point scales, ranging from 0 to 3. Although anchors differ slightly by item, generally, 0 indicates no evidence of an adversity or symptom, and 3 indicates frequent and severe exposure to an adversity or severe experience of a symptom. For example, with regard to physical abuse, 0 = *no evidence of physical abuse*; 1 = *one episode of physical abuse or suspicion of physical abuse*; 2 = *repeated physical abuse;* and 3 = *severe and repeated physical abuse to necessitate hospital treatment*. The majority of ACEs items represent lifetime exposure. However, the neglect item represents exposure within the 30 days prior to the CANS assessment. The ACEs items related to caregiver functioning (i.e., caregiver substance abuse, caregiver mental illness) represent the extent to which the caregiver's current problems impede their capacity to parent. For example, 0 = *caregiver has no substance use (mental health) needs*; 1 = *caregiver is in recovery from substance use (mental health) difficulties*; 2 = *Caregiver has some substance use (mental health) difficulties that interfere with his or her capacity to parent;* and 3 = *caregiver has substance use (mental health) difficulties that make it impossible for him/her to parent at this time*.

Factor Analysis and Creation of Composite Variables

The dependent variables were derived using principal axis factor analysis with oblique (Oblimin) rotation. We selected items from the CANS that reflected internalizing and externalizing symptoms, using the Child Behavior Checklist as a guide (Achenbach & Rescorla, 2001; see Table 1 in the online supplemental materials).[1] Items were required to have loadings greater than .40 on one factor to be retained (Stevens, 2012). With loadings less than .40 on all factors, sleep, fire setting, and runaway items were excluded. We settled on a three-factor solution by examining the scree plot

[1] Attention deficit–hyperactivity disorder (ADHD) is grouped into the Mixed Syndromes section of the Child Behavior Checklist (Achenbach & Rescorla, 2001) rather than the Externalizing Problems Scale. However, considering other research suggesting that ADHD is part of a child externalizing spectrum (e.g., Burt et al., 2005; Kotov et al., 2017; Tackett, 2010), we decided to subject the CANS item "inattention/hyperactivity" to factor analysis, as an item potentially reflecting externalizing symptoms.

(Cattell, 1966) and selecting Eigenvalues greater than 1 (Kaiser, 1960; see Table 2 in the online supplemental materials). Depression, anxiety, and suicide risk clustered together as expected, and so we labeled Factor 1 as Internalizing symptoms (Cronbach's alpha = .73). Externalizing symptoms were best represented as two separate factors. Since Achenbach's original factor analysis of children's psychological symptoms into internalizing and externalizing domains (Achenbach, 1966), creators of the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017) have proposed to further separate externalizing symptoms into disinhibited and antagonistic spectra (Krueger et al., 2002, 2007). Considering similarities between the items in our factors and the disorders and syndromes subsumed in these spectra, we labeled Factor 2 as Disinhibited Externalizing (delinquency, legal, and substance use; Cronbach's alpha = .85) and Factor 3 as Antagonistic Externalizing (anger control, danger to others, oppositional, conduct, sanction seeking behavior, and impulsivity/hyperactivity; Cronbach's alpha = .87).

We created composites for the independent (i.e., threat, deprivation, and cumulative risk) and dependent variables (i.e., internalizing symptoms, disinhibited externalizing symptoms, and antagonistic externalizing symptoms). The 4-point scale was preserved for each item to retain information about ACE and symptom severity. Composites for threat and deprivation were derived empirically using Study 1 ratings. The threat composite was created by averaging scores for six ACEs: physical abuse, sexual abuse, medical trauma, natural disaster, family violence, and community violence. The deprivation composite was created by averaging scores for four ACEs: neglect, emotional abuse, caregiver substance abuse, and caregiver mental illness. Although cumulative risk is typically represented as a sum score in the literature, we created the cumulative risk composite by averaging scores for all 10 ACEs to facilitate comparison with DMAP in analyses.

Missing Data

CANS data were collected on 23,850 children and adolescents upon their entrance into state custody (T1). At T1, data on the race variable were missing from 584 participants. We conducted *t* tests to understand trends in the missingness of these data (all $p < .05$). Youth with missing data on race were significantly older, and they had lower scores on witness to family violence, witness to community violence, neglect, caregiver substance use, caregiver mental health, threat, deprivation, and cumulative risk variables. They were not significantly different from individuals without missing race data with regard to internalizing or externalizing symptoms.

A second wave of CANS data (T2) was available for 16,503 of the original 23,850 children and adolescents. T2 data were collected within six months of T1—either upon leaving state custody or six months postintake. In 2017, DCS enacted protocol changes to support and promote caseworkers' ability to extend assessments to key points throughout the duration of a custodial event, which facilitated more consistent collection of assessments, including T2 data used in the present study. Youth without T2 data were significantly different from youth with data on the majority of key variables. However, these differences were small in magnitude ($.11 \leq d \leq .25$). Youth with missing data at T2 were older in age ($M = 13.56$, $SD = 4.06$) than youth with data [$M = 12.73$, $SD = 4.07$, $t(14{,}112.46) = 14.55$, $p < .001$], more likely to be male than youth with data, $\chi^2(1) = 13.91$, $p < .001$, had fewer internalizing symptoms at T1 ($M = 1.16$, $SD = 1.53$) than youth with data [$M = 1.41$, $SD = 1.64$, $t(14{,}972.14) = -11.08$, $p < .001$], had more disinhibited externalizing symptoms at T1 ($M = 2.32$, $SD = 2.52$) than youth with data [$M = 2.09$, $SD = 2.61$, $t(14{,}569.95) = 6.57$, $p < .001$], and had fewer antagonistic externalizing symptoms at T1 ($M = 3.52$, $SD = 3.64$) than youth with data [$M = 4.32$, $SD = 4.08$, $t(15{,}704.16) = -15.03$, $p < .001$]. In addition, they had significantly less exposure to nine ACEs (*t* test for exposure to natural disaster was nonsignificant), corresponding to lower cumulative risk scores ($M = .31$, $SD = .33$) than youth with data [$M = .38$, $SD = .34$, $t(14{,}655.75) = -16.92$, $p < .001$], lower threat scores ($M = .16$, $SD = .26$) than youth with data [$M = .20$, $SD = .29$, $t(15{,}480.85) = -10.77$, $p < .001$], and lower deprivation scores ($M = .52$, $SD = .56$) than youth with data [$M = .66$, $SD = .57$, $t(14{,}339.67) = -16.98$, $p < .001$]. We generated two hypotheses for the observed missingness at T2: these youth aged out of DCS services, and/or they were exposed to less severe ACEs or symptoms and left state custody soon after they entered. To allow inclusion of youth with partial data at T1 and T2 and to reduce the bias often associated with listwise deletion, we used full information maximum likelihood (FIML) estimation using Amos (Version 7.0).[2] We sought to improve the performance of FIML estimation by accounting for potential nonrandom missingness through the inclusion of auxiliary variables (Collins et al., 2001): Age and gender were included as covariates in all regression equations, ACEs were predictors in all regression equations, and T1 symptoms were controlled for when predicting symptoms at T2.

Effect Size

Given the large sample size, we had .80 power to detect correlations of $r = .02$ with $\alpha = .001$. Consequently, we elected to place greater emphasis on effect size than statistical significance. In Tables 2 through 7, we denote statistically significant effects at the family-wise alpha levels in bold type and use superscripts to indicate effect size based on Cohen's (1988) criteria.

Results

Descriptive Statistics

Table 1 provides the means, standard deviations, and possible and actual ranges for ACEs variables, ACEs composites, and psychopathology at T1 and T2. In addition, Table 1 reports the prevalence of key study variables in the sample. Seventy-seven percent of participants reported at least one ACE, with caregiver mental illness (45%) and caregiver substance abuse (45%) being the most common.

Bivariate Correlations

We computed bivariate correlations to understand the simple associations among our variables. First, we examined relations between demographics and key study variables (Tables 2 and 3).

[2] Regression analyses were also computed for individuals with full data only at T1 and T2 (i.e., listwise deletion) and findings did not differ from FIML, likely because of our large sample size.

Table 1
Descriptive Statistics of Key Study Variables

Measure	% > 0	*M*	*SD*	Range	
				Potential	Actual
Cumulative risk	77	0.36	0.34	0–3	0–2.5
Threat	43	0.19	0.28	0–3	0–2.7
Physical abuse	22	0.29	0.60	0–3	0–3
Sexual abuse	18	0.27	0.62	0–3	0–3
Medical trauma	5	0.07	0.34	0–3	0–3
Natural disaster	1	0.02	0.17	0–3	0–3
Witness family violence	26	0.38	0.71	0–3	0–3
Witness community violence	10	0.12	0.40	0–3	0–3
Deprivation	72	0.62	0.57	0–3	0–3
Neglect	36	0.53	0.79	0–3	0–3
Emotional abuse	27	0.36	0.67	0–3	0–3
Caregiver substance abuse	45	0.87	1.10	0–3	0–3
Caregiver mental illness	45	0.70	0.88	0–3	0–3
Internalizing	54	1.33	1.61	0–9	0–9
Disinhibited externalizing	51	2.16	2.58	0–9	0–9
Antagonistic externalizing	71	4.07	3.97	0–18	0–18
Internalizing T2	63	1.47	1.53	0–9	0–9
Disinhibited externalizing T2	48	1.55	2.03	0–9	0–9
Antagonistic externalizing T2	77	4.00	3.62	0–18	0–18

Note. % > 0 represents the percent of participants with a score greater than 0. Values for all variables presented at T1 unless otherwise noted.

Younger age and female gender were associated with exposure to several ACEs variables and composites. Non-White race was associated with witnessing community violence, and White race was associated with exposure to caregiver substance abuse and mental illness. Medium effects emerged between older age and internalizing and antagonistic externalizing symptoms, and large effects emerged between older age and disinhibited externalizing symptoms. Small effects emerged between male gender and non White race and externalizing symptoms.

Second, we examined correlations between individual ACEs and internalizing and externalizing symptoms (see Table 2). The majority of ACEs were related to internalizing symptoms with small positive effects, and several ACEs were related to externalizing symptoms with small positive effects. Unexpectedly, care-

Table 2
Bivariate Correlations With Individual ACEs

Measure	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1. Age	—																	
2. Gender	**.12**[a]	—																
3. Race	**.09**	**.05**	—															
4. PA	.01	**−.04**	−.01	—														
5. SA	**.05**	**−.23**[a]	**−.06**	**.30**[b]	—													
6. MT	.02	−.01	−.01	**.16**[a]	**.09**	—												
7. ND	.01	.001	.001	**.05**	**.03**	**.04**	—											
8. WFV	**−.09**	**−.05**	**−.03**	**.46**[b]	**.20**[a]	**.12**[a]	**.05**	—										
9. WCV	**.08**	**.03**	**.11**[a]	**.17**[a]	**.09**	**.12**[a]	**.08**	**.32**[b]	—									
10. Neglect	**−.12**[a]	**−.09**	**−.03**	**.37**[b]	**.25**[a]	**.17**[a]	**.04**	**.42**[b]	**.20**[a]	—								
11. EA	**.03**	**−.09**	**−.07**	**.58**[c]	**.36**[b]	**.18**[a]	**.05**	**.49**[b]	**.21**[a]	**.49**[b]	—							
12. CSA	**−.39**[b]	**−.11**[a]	**−.16**[a]	**−.03**	**−.06**	−.003	.02	**.10**[a]	.01	**.16**[a]	.02	—						
13. CMI	**−.30**[b]	**−.10**[a]	**−.12**[a]	**.11**[a]	**.07**	**.04**	**.03**	**.19**[a]	**.03**	**.20**[a]	**.18**[a]	**.41**[b]	—					
14. Intern	**.34**[b]	**−.08**	**−.09**	**.23**[a]	**.27**[a]	**.11**[a]	**.03**	**.16**[a]	**.14**[a]	**.15**[a]	**.29**[a]	**−.14**[a]	.01	—				
15. D extern	**.62**[c]	**.25**[a]	**.11**[a]	**−.05**	**−.04**	.02	.01	**−.09**	**.13**[a]	**−.16**[a]	**−.06**	**−.35**[b]	**−.31**[b]	**.26**[a]	—			
16. A extern	**.44**[b]	**.23**[a]	**.16**[a]	**.15**[a]	**.11**[a]	**.07**	**.02**	**.07**	**.17**[a]	**.03**	**.13**[a]	**−.32**[b]	**−.17**[a]	**.43**[b]	**.59**[c]	—		
17. Intern T2	**.34**[b]	**−.09**	**−.08**	**.21**[a]	**.25**[a]	**.09**	.01	**.15**[a]	**.11**[a]	**.14**[a]	**.26**[a]	**−.14**[a]	−.002	**.71**[c]	**.22**[a]	**.37**[b]	—	
18. D extern T2	**.58**[c]	**.23**[a]	**.12**[a]	**−.03**	−.03	.02	.01	**−.07**	**.12**[a]	**−.12**[a]	**−.04**	**−.33**[b]	**−.29**[a]	**.25**[a]	**.79**[c]	**.51**[c]	**.28**[a]	—
19. A extern T2	**.34**[b]	**.21**[a]	**.16**[a]	**.16**[a]	**.11**[a]	**.06**	.02	**.08**	**.14**[a]	**.06**	**.13**[a]	**−.28**[a]	**−.13**[a]	**.34**[b]	**.41**[b]	**.72**[c]	**.46**[b]	**.54**[c]

Note. PA = physical abuse; SA = sexual abuse; MT = medical trauma; ND = natural disaster; WFV = witness to family violence; WCV = witness to community violence; EA = emotional abuse; CSA = caregiver substance abuse; CMI = caregiver mental illness; intern = internalizing; D extern = disinhibited externalizing; A extern = antagonistic externalizing. Female = 0, male = 1; White = 0, non-White = 1. Values for all variables presented at T1 unless otherwise noted. Significant effects based on family-wise error rate $p < .0004$ are bold.
[a] Small effect $.10 \leq r \leq .29$. [b] Medium effect $.30 \leq r < .49$. [c] Large effect $r \geq .50$.

Table 3
Bivariate Correlations With Composite Variables

Measure	1	2	3	4	5	6	7	8	9	10	11
1. Age	—										
2. Gender	**.12**[a]	—									
3. Race	**.09**	**.05**	—								
4. CR	**−.22**[a]	**−.16**[a]	**−.11**[a]	—							
5. Threat	.01	**−.11**[a]	−.01	**.79**[c]	—						
6. Deprivation	**−.34**[b]	**−.15**[a]	**−.16**[a]	**.89**[c]	**.42**[b]	—					
7. Intern	**.34**[b]	**−.08**	**−.09**	**.21**[a]	**.31**[b]	**.08**	—				
8. D extern	**.62**[c]	**.25**[a]	**.11**[a]	**−.26**[a]	**−.03**	**−.36**[b]	**.26**[a]	—			
9. A extern	**.44**[b]	**.23**[a]	**.16**[a]	**−.03**	**.18**[a]	**−.17**[a]	**.43**[b]	**.59**[c]	—		
10. Intern T2	**.33**[b]	**−.09**	**−.08**	**.18**[a]	**.28**[a]	**.06**	**.71**[c]	**.22**[a]	**.37**[b]	—	
11. D extern T2	**.58**[c]	**.23**[a]	**.12**[a]	**−.23**[a]	−.02	**−.33**[b]	**.25**[a]	**.79**[c]	**.51**[c]	**.28**[a]	—
12. A extern T2	**.34**[b]	**.21**[a]	**.16**[a]	.01	**.18**[a]	**−.13**[a]	**.34**[b]	**.41**[b]	**.72**[c]	**.46**[b]	**.54**[c]

Note. CR = cumulative risk; intern = internalizing; D extern = disinhibited externalizing; A extern = antagonistic externalizing. Female = 0, male = 1; White = 0, non-White = 1. Values for all variables presented at T1 unless otherwise noted. Significant effects based on family-wise error rate $p < .001$ are bold.
[a] Small effect $.10 \leq r \leq .29$. [b] Medium effect $.30 \leq r < .49$. [c] Large effect $r \geq .50$.

giver substance abuse was negatively related to internalizing symptoms. Neglect, caregiver substance abuse, and caregiver mental illness were negatively related to disinhibited externalizing symptoms and caregiver substance abuse and caregiver mental illness were negatively related to antagonistic externalizing symptoms.

Third, we computed correlations between the ACEs composites and internalizing and externalizing symptoms (see Table 3). Cumulative risk showed small positive associations with internalizing symptoms and small negative associations with disinhibited externalizing symptoms. Threat showed small-to-medium positive associations with internalizing symptoms and small positive associations with antagonistic externalizing symptoms. Deprivation showed medium negative associations with disinhibited externalizing symptoms and small negative associations with antagonistic externalizing symptoms.

Linear Regression Analyses

We conducted hierarchical linear regression analyses to test ACEs in the individual risk, DMAP, and cumulative risk approaches as predictors of internalizing symptoms at T1 (see Table 4) and T2 (see Table 5), disinhibited externalizing symptoms at T1 (see Table 6) and T2 (see Table 7), and antagonistic externalizing symptoms at T1 (see Table 8) and T2 (see Table 9). In each regression equation, we controlled for age, gender, and race. When predicting T2 symptoms, we also controlled for symptoms at T1. To best test of variance explained by each model, we randomly split the dataset into a test sample (n = 11,890, denoted as *test*, below) and a validation sample (n = 11,960, denoted as *validation*, below) and calculated R^2 values in each sample. To take full advantage of the large sample size, however, coefficients for predictors were calculated using the full sample (N = 23,850).

Individual Risk Model. The addition of ACEs variables contributed significantly to the variance in internalizing symptoms at T1 ($\Delta R^2_{\text{Test}} = .110$, $\Delta R^2_{\text{Validation}} = .102$) and T2 ($\Delta R^2_{\text{Test}} = .008$, $\Delta R^2_{\text{Validation}} = .007$). At T1, eight of 10 ACEs were associated with more internalizing symptoms. Caregiver substance abuse was negatively related to internalizing symptoms ($\beta = -.06$), and the relation between natural disaster and internalizing symptoms was nonsignificant. At T2, only exposure to sexual abuse and emotional abuse predicted more internalizing symptoms. ACEs variables contributed significantly to the variance in disinhibited externalizing symptoms at T1 ($\Delta R^2_{\text{Test}} = .030$, $\Delta R^2_{\text{Validation}} = .030$) and T2 ($\Delta R^2_{\text{Test}} = .001$, $\Delta R^2_{\text{Validation}} = .002$). At T1, medical trauma and witnessing community violence were associated with more externalizing symptoms, and physical abuse ($\beta = -.02$), neglect ($\beta = -.05$), emotional abuse ($\beta = -.02$), caregiver substance abuse ($\beta = -.08$), and caregiver mental illness ($\beta = -.09$) were associated with fewer disinhibited externalizing symptoms. At T2, witnessing community violence predicted more disinhibited externalizing symptoms, and caregiver substance abuse ($\beta = -.02$) and caregiver mental illness ($\beta = -.02$) predicted fewer disinhibited externalizing symptoms. ACEs variables contributed significantly to the variance in antagonistic externalizing symptoms at T1 ($\Delta R^2_{\text{Test}} = .067$, $\Delta R^2_{\text{Validation}} = .061$) and T2 ($\Delta R^2_{\text{Test}} = .006$, $\Delta R^2_{\text{Validation}} = .004$). At T1, physical abuse, sexual abuse, medical trauma, witnessing community violence, and emotional abuse were associated with more antagonistic externalizing symptoms, and caregiver substance abuse was associated with fewer antagonistic externalizing symptoms ($\beta = -.15$). At T2, physical abuse, sexual abuse, and neglect predicted more antagonistic externalizing symptoms, and caregiver substance abuse predicted fewer antagonistic externalizing symptoms ($\beta = -.02$).

DMAP Model. Threat and deprivation contributed significantly to the variance in internalizing symptoms at T1 ($\Delta R^2_{\text{Test}} = .089$, $\Delta R^2_{\text{Validation}} = .083$) and T2 ($\Delta R^2_{\text{Test}} = .006$, $\Delta R^2_{\text{Validation}} = .006$). Threat predicted more internalizing symptoms at T1 ($\beta = .26$) and T2 ($\beta = .07$). Deprivation predicted more internalizing symptoms at T1 ($\beta = .06$) and T2 ($\beta = .02$). Threat and deprivation contributed significantly to the variance in disinhibited externalizing symptoms at T1 ($\Delta R^2_{\text{Test}} = .021$, $\Delta R^2_{\text{Validation}} = .020$) and T2 ($\Delta R^2_{\text{Test}} = .001$, $\Delta R^2_{\text{Validation}} = .002$). Threat predicted more disinhibited externalizing symptoms at T1 only ($\beta = .06$). Deprivation predicted fewer externalizing symptoms at T1 ($\beta = -.17$) and T2 ($\beta = -.04$). Threat and deprivation contributed significantly to the

Table 4
Individual Risk, Cumulative Risk, and DMAP Predicting Internalizing Symptoms at T1

		Individual risk				DMAP				Cumulative risk			
Block	Predictor	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*
1	Age	.14 (.002)	[.14, .14]	.37[b]	**60.52**	.14 (.002)	[.14, .14]	.37[b]	**60.52**	.14 (.002)	[.14, .14]	.37[b]	**60.52**
	Gender	−.39 (.02)	[−.43, −.35]	−.12[a]	**−19.65**	−.39 (.02)	[−.43, −.35]	−.12[a]	**−19.65**	−.39 (.02)	[−.43, −.35]	−.12[a]	**−19.65**
	Race	−.40 (.02)	[−.44, −.36]	−.11[a]	**−18.76**	−.40 (.02)	[−.44, −.36]	−.11[a]	**−18.76**	−.40 (.02)	[−.44, −.36]	−.11[a]	**−18.76**
2	Age	.14 (.002)	[.14, .14]	.35[b]	**55.32**	.15 (.002)	[.15, .15]	.38[b]	**61.68**	.17 (.002)	[.17, .17]	.42[c]	**71.27**
	Gender	−.22 (.02)	[−.26, −.18]	−.07[a]	**−11.79**	−.26 (.02)	[−.30, −.22]	−.08[a]	**−14.05**	−.27 (.02)	[−.31, −.23]	−.08[a]	**−14.19**
	Race	−.36 (.02)	[−.40, −.32]	−.10[a]	**−17.63**	−.37 (.02)	[−.41, −.33]	−.10[a]	**−17.83**	−.32 (.02)	[−.36, −.28]	−.09[a]	**−15.36**
	PA	.16 (.02)	[.12, .20]	.06[a]	**8.47**								
	SA	.35 (.02)	[.31, .39]	.13[a]	**21.33**								
	MT	.19 (.03)	[.13, .25]	.04[a]	**7.02**								
	ND	.03 (.05)	[−.07, .13]	.003	.61								
	WFV	.06 (.02)	[.02, .10]	.03[a]	**3.63**								
	WCV	.24 (.02)	[.20, .28]	.06[a]	**9.80**								
	Neglect	.05 (.01)	[.03, .07]	.03[a]	**3.90**								
	EA	.30 (.02)	[.26, .34]	.13[a]	**16.09**								
	CSA	−.09 (.01)	[−.11, −.07]	−.06[a]	**−8.90**								
	CMI	.13 (.01)	[.11, .15]	.07[a]	**11.55**								
	Threat					1.50 (.04)	[1.42, 1.58]	.26[b]	**41.25**				
	Dep					.18 (.02)	[.14, .22]	.06[a]	**9.38**				
	CR									1.32 (.03)	[1.26, 1.38]	.28[b]	**46.36**
1		R^2_T = .1386, F(3, 23,846) = 1,279.28				R^2_T = .1386, F(3, 23,846) = 1,279.28				R^2_T = .1386, F(3, 23,846) = 1,279.28			
		R^2_V = .1512, F(3, 23,846) = 1,416.45				R^2_V = .1512, F(3, 23,846) = 1,416.45				R^2_V = .1512, F(3, 23,846) = 1,416.45			
2		R^2_T = .2490, F(13, 23,836) = 607.95				R^2_T = .2281, F(5, 23,844) = 1,409.03				R^2_T = .2120, F(4, 23,845) = 1,604.01			
		R^2_V = .2531, F(13, 23,836) = 621.57				R^2_V = .2347, F(5, 23,844) = 1,462.33				R^2_V = .2195, F(4, 23,845) = 1,676.67			
Δ 1 → 2		ΔR^2_T = .1104, ΔF(10, 23,836) = 350.33				ΔR^2_T = .0894, ΔF(2, 23,844) = 1,381.47				ΔR^2_T = .0734, ΔF(1, 23,845) = 2,220.92			
		ΔR^2_V = .1019, ΔF(10, 23,836) = 325.31				ΔR^2_V = .0834, ΔF(2, 23,844) = 1,299.70				ΔR^2_V = .0683, ΔF(1, 23,845) = 2,085.79			

Note. PA = physical abuse; SA = sexual abuse; MT = medical trauma; ND = natural disaster; WFV = witness to family violence; WCV = witness to community violence; EA = emotional abuse; CSA = caregiver substance abuse; CMI = caregiver mental illness; Dep = deprivation; CR = cumulative risk. Significant effects based on the relevant family-wise error rate (FWER) are bold. Individual risk model FWER $p < .004$; cumulative risk model FWER $p < .013$; DMAP model FWER $p < .010$. R^2_T represents value derived in the test sample (n = 11,890). R^2_V represents value derived in the validation sample (n = 11,960).
[a] Small effect $.02 \leq f^2 \leq .24$. [b] Medium effect $.25 \leq f^2 < .39$. [c] Large effect $f^2 \geq .40$.

variance in antagonistic externalizing symptoms at T1 (ΔR^2_{Test} = .049, $\Delta R^2_{\text{Validation}}$ = .044) and T2 (ΔR^2_{Test} = .005, $\Delta R^2_{\text{Validation}}$ = .003). Threat predicted more antagonistic externalizing symptoms at T1 (β = .24) and T2 (β = .06). Deprivation predicted fewer antagonistic externalizing symptoms at T1 only (β = −.11).

Cumulative Risk Model. Cumulative risk contributed significantly to the variance in internalizing symptoms at T1 (ΔR^2_{Test} = .073, $\Delta R^2_{\text{Validation}}$ = .068) and T2 (ΔR^2_{Test} = .005, $\Delta R^2_{\text{Validation}}$ = .005). Higher cumulative risk scores were associated with more internalizing symptoms at T1 (β = .28) and T2 (β = .07). Cumulative risk contributed significantly to the variance in disinhibited externalizing symptoms at T1 (ΔR^2_{Test} = .010, $\Delta R^2_{\text{Validation}}$ = .009) and T2 (ΔR^2_{Test} = .0003, $\Delta R^2_{\text{Validation}}$ = .001). Higher cumulative risk scores predicted fewer disinhibited externalizing symptoms at T1 (β = −.10) and T2 (β = −.02). Cumulative risk contributed significantly to the variance in antagonistic externalizing symptoms at T1 (ΔR^2_{Test} = .011, $\Delta R^2_{\text{Validation}}$ = .013) and T2 (ΔR^2_{Test} = .004, $\Delta R^2_{\text{Validation}}$ = .002). Higher cumulative risk scores predicted more antagonistic externalizing symptoms at T1 (β = .11) and T2 (β = .05).

Model Comparison

We conducted partial F tests to compare the incremental predictive utility of the individual risk, DMAP, and cumulative risk models in predicting internalizing and externalizing symptoms. These analyses were possible given that the equations for the three models are hierarchically nested[3]:

Individual risk:

$$Y = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3 + \beta_4 x_4 + \beta_5 x_5 + \beta_6 x_6 + \beta_7 x_7 + \beta_8 x_8 + \beta_9 x_9 + \beta_{10} x_{10} + \varepsilon;$$

DMAP:

$$Y = \beta_0 + \beta_1 (x_1 + x_2 + x_3 + x_4 + x_5 + x_6)/6 + \beta_2 (x_7 + x_8 + x_9 + x_{10})/4 + \varepsilon;$$

Cumulative risk:

$$Y = \beta_0 + \beta_1 (x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8 + x_9 + x_{10})/10 + \varepsilon.$$

The individual risk model is the fullest model, with every ACE variable (x_i) receiving its own unconstrained regression weight. The DMAP model is a more restricted version of the individual risk model, in which the regression weights for all of the threat variables are constrained to be equal (β_1), as are the weights for all of the deprivation variables (β_2). The cumulative risk model is the

[3] Control variables are omitted from these expressions (but not from the actual analyses) for clarity.

Table 5
Individual Risk, Cumulative Risk, and DMAP Predicting Internalizing Symptoms at T2

		Individual risk				DMAP				Cumulative risk			
Block	Predictor	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*
1	Age	.04 (.002)	[.04, .04]	.10[a]	**16.23**	.04 (.002)	[.04, .04]	.10[a]	**16.23**	.04 (.002)	[.04, .04]	.10[a]	**16.23**
	Gender	−.16 (.02)	[−.20, −.12]	−.05[a]	**−9.26**	−.16 (.02)	[−.20, −.12]	−.05[a]	**−9.26**	−.16 (.02)	[−.20, −.12]	−.05[a]	**−9.26**
	Race	−.12 (.02)	[−.16, −.08]	−.04[a]	**−6.26**	−.12 (.02)	[−.16, −.08]	−.04[a]	**−6.26**	−.12 (.02)	[−.16, −.08]	−.04[a]	**−6.26**
	Int	.62 (.006)	[.61, .63]	.66[c]	**111.14**	.62 (.006)	[.61, .63]	.66[c]	**111.14**	.62 (.006)	[.61, .63]	.66[c]	**111.14**
2	Age	.04 (.002)	[.04, .04]	.11[a]	**16.35**	.04 (.002)	[.04, .04]	.11[a]	**17.23**	.04 (.002)	[.04, .04]	.12[a]	**18.98**
	Gender	−.12 (.02)	[−.16, −.08]	−.04[a]	**−6.80**	−.14 (.02)	[−.18, −.10]	−.04[a]	**−8.02**	−.14 (.02)	[−.18, −.10]	−.04[a]	**−8.05**
	Race	−.11(.02)	[−.15, −.07]	−.03[a]	**−5.77**	−.12 (.02)	[−.16, −.08]	−.03[a]	**−6.15**	−.10 (.02)	[−.14, −.06]	−.03[a]	**−5.43**
	Int	.59 (.006)	[.58, .60]	.63[c]	**99.81**	.60 (.006)	[.59, .61]	.64[c]	**101.91**	.60 (.006)	[.59, .61]	.64[c]	**103.62**
	PA	.04 (.02)	[.36, .44]	.02[a]	2.49								
	SA	.11 (.02)	[.07, .15]	.05[a]	**7.55**								
	MT	.003 (.02)	[−.04, .04]	<−.001	.13								
	ND	−.08 (.05)	[−.18, .02]	<−.009	−1.60								
	WFV	.03 (.01)	[.01, .05]	.01	2.15								
	WCV	.01 (.02)	[−.03, .05]	.003	.53								
	Neglect	.02 (.01)	[.00, .04]	.01	1.62								
	EA	.06 (.02)	[.02, .10]	.03[a]	**3.57**								
	CSA	−.008 (.009)	[−.03, .01]	−.006	−.88								
	CMI	.03 (.01)	[.01, .05]	.01	2.39								
	Threat					.36 (.03)	[.30, .42]	.07[a]	**10.40**				
	Dep					.06 (.02)	[.02, .10]	.02[a]	**3.20**				
	CR									.33 (.03)	[.27, .39]	.07[a]	**12.33**
1		R^2_T = .4891, F(4, 23,845) = 5,707.74				R^2_T = .4891, F(4, 23,845) = 5,707.74				R^2_T = .4891, F(4, 23,845) = 5,707.74			
		R^2_V = .5140, F(4, 23,845) = 6,304.70				R^2_V = .5140, F(4, 23,845) = 6,304.70				R^2_V = .5140, F(4, 23,845) = 6,304.70			
2		R^2_T = .4975, F(14, 23,835) = 1,685.87				R^2_T = .4949, F(6, 23,843) = 3,893.10				R^2_T = .4943, F(5, 23,844) = 4,660.86			
		R^2_V = .5207, F(14, 23,835) = 1,849.70				R^2_V = .5195, F(6, 23,843) = 4,296.57				R^2_V = .5189, F(5, 23,844) = 5,142.45			
Δ 1 → 2		ΔR^2_T = .0084, ΔF(10, 23,835) = 39.89				ΔR^2_T = .0057, ΔF(2, 23,843) = 135.27				ΔR^2_T = .0051, ΔF(1, 23,844) = 242.30			
		ΔR^2_V = .0067, ΔF(10, 23,835) = 33.42				ΔR^2_V = .0055, ΔF(2, 23,843) = 136.74				ΔR^2_V = .0048, ΔF(1, 23,844) = 240.34			

Note. Int = internalizing symptoms at T1; PA = physical abuse; SA = sexual abuse; MT = medical trauma; ND = natural disaster; WFV = witness to family violence; WCV = witness to community violence; EA = emotional abuse; CSA = caregiver substance abuse; CMI = caregiver mental illness; Dep = deprivation; CR = cumulative risk. Significant effects based on the relevant family-wise error rate (FWER) are bold. Individual risk model FWER $p < .004$; cumulative risk model FWER $p < .013$; DMAP model FWER $p < .010$. R^2_T represents value derived in the test sample (n = 11,890). R^2_V represents value derived in the validation sample (n = 11,960).
[a] Small effect $.02 \leq f^2 \leq .24$. [c] Large effect $f^2 \geq .40$.

most restricted model in which the weights for all predictors are constrained to be equal. The constraints included in the DMAP and cumulative risk models are implicit in the computation of the threat, deprivation, and cumulative risk composites, within which all ACEs are given equal weights. A priori, the individual risk model should account for more variance than the DMAP and cumulative risk models, and DMAP should account for more variance than the cumulative risk model; the fuller models have more item-level information, and so more predictive power, than the reduced models. However, it is not predetermined that the difference in variance across models will be significant. Partial F tests were conducted to determine whether the three models are *significantly* different from one another.

First, we compared individual risk (full model) with DMAP (reduced model). The individual risk model predicted significantly more of the variance in internalizing symptoms at T1 [test: ΔR^2 = .021, F(8, 23,836) = 83.04, $p < .001$; validation: ΔR^2 = .018, F(8, 23,836) = 73.78, $p < .001$] and T2 [test: ΔR^2 = .003, F(8, 23,835) = 15.88, $p < .001$; validation: ΔR^2 = .001, F(8, 23,835) = 7.51, $p < .001$], disinhibited externalizing symptoms at T1 [test: ΔR^2 = .009, F(8, 23,836) = 50.35, $p < .001$; validation: ΔR^2 = .010, F(8, 23,836) = 54.43, $p < .001$] and T2 [test: ΔR^2 = .0004, F(8, 23,835) = 3.07, p = .002; validation: ΔR^2 = .0004, F(8, 23,835) = 3.84, $p < .001$], and antagonistic externalizing symptoms at TI [test: ΔR^2 = .018, F(8, 23,836) = 77.01, $p < .001$; validation: ΔR^2 = .017, F(8, 23,836) = 73.82, $p < .001$] and T2 [test: ΔR^2 = .001, F(8, 23,835) = 8.84, $p < .001$; validation: ΔR^2 = .002, F(8, 23,835) = 9.73, $p < .001$] than the DMAP model.

Second, we compared individual risk (full model) with cumulative risk (reduced model). The individual risk model predicted significantly more of the variance in internalizing symptoms at T1 [test: ΔR^2 = .037, F(9, 23,836) = 130.43, $p < .001$; validation: ΔR^2 = .034, F(9, 23,836) = 119.35, $p < .001$] and T2 [test: ΔR^2 = .003, F(9, 23,835) = 17.23, $p < .001$; validation: ΔR^2 = .002, F(9, 23.835) = 10.33, $p < .001$], disinhibited externalizing symptoms at T1 [test: ΔR^2 = .020, F(9, 23,836) = 100.81, $p < .001$; validation: ΔR^2 = .021, F(9, 23,836) = 100.93, $p < .001$] and T2 [test: ΔR^2 = .001, F(9, 23,835) = 6.18, $p < .001$; validation: ΔR^2 = .001, F(9, 23,835) = 7.23, $p < .001$], and antagonistic externalizing symptoms at T1 [test: ΔR^2 = .056, F(9, 23,836) = 211.46, $p < .001$; validation: ΔR^2 = .049, F(9, 23,836) = 182.40, $p < .001$] and T2 [test: ΔR^2 = .002, F(9, 23,835) = 13.64, $p < .001$; validation: ΔR^2 = .002, F(9, 23,835) = 10.18, $p < .001$] than the cumulative risk model.

Third, we compared DMAP (full model) with cumulative risk (reduced model). DMAP predicted significantly more of the variance in internalizing symptoms at T1 [test: ΔR^2 = .016, F(1,

Table 6
Individual Risk, Cumulative Risk, and DMAP Predicting Disinhibited Externalizing Symptoms at T1

		Individual risk				DMAP				Cumulative risk			
Block	Predictor	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*
1	Age	.38 (.003)	[.37, .39]	.60[c]	**120.34**	.38 (.003)	[.37, .39]	.60[c]	**120.34**	.38 (.003)	[.37, .39]	.60[c]	**120.34**
	Gender	.93 (.03)	[.87, .99]	.18[a]	**35.61**	.93 (.03)	[.87, .99]	.18[a]	**35.61**	.93 (.03)	[.87, .99]	.18[a]	**35.61**
	Race	.27 (.03)	[.21, .33]	.05[a]	**9.49**	.27 (.03)	[.21, .33]	.05[a]	**9.49**	.27 (.03)	[.21, .33]	.05[a]	**9.49**
2	Age	.34 (.003)	[.33, .35]	.53[c]	**99.34**	.34 (.003)	[.33, .35]	.54[c]	**103.55**	.37 (.003)	[.36, .38]	.58[c]	**114.83**
	Gender	.81 (.03)	[.75, .87]	.16[a]	**31.16**	.87 (.03)	[.81, .93]	.17[a]	**33.59**	.86 (.03)	[.80, .92]	.16[a]	**32.98**
	Race	.08 (.03)	[.02, .14]	.01	**2.98**	.15 (.03)	[.09, .21]	.03[a]	**5.36**	.22 (.03)	[.16, .28]	.04[a]	**7.74**
	PA	−.10 (.03)	[−.16, −.04]	−.02[a]	**−3.91**								
	SA	−.05 (.02)	[−.09, −.01]	−.01	−2.27								
	MT	.15 (.04)	[.07, .23]	.02[a]	**4.03**								
	ND	.09 (.07)	[−.05, .23]	.006	1.17								
	WFV	.002 (.02)	[−.04, .04]	<.001	.10								
	WCV	.65 (.03)	[.59, .71]	.10[a]	**19.55**								
	Neglect	−.15 (.02)	[−.19, −.11]	−.05[a]	**−7.98**								
	EA	−.09 (.03)	[−.15, −.03]	−.02[a]	**−3.62**								
	CSA	−.19 (.01)	[−.21, −.17]	−.08[a]	**−14.63**								
	CMI	−.25 (.02)	[−.29, −.21]	−.09[a]	**−15.60**								
	Threat					.50 (.05)	[.40, .60]	.06[a]	**10.12**				
	Dep					−.78 (.03)	[−.84, −.72]	−.17[a]	**−29.37**				
	CR									−.77 (.04)	[−.85, −.69]	−.10[a]	**−19.68**
1		R^2_T = .4344, F(3, 23,846) = 6,104.65				R^2_T = .4344, F(3, 23,846) = 6,104.65				R^2_T = .4344, F(3, 23,846) = 6,104.65			
		R^2_V = .4116, F(3, 23,846) = 5,559.14				R^2_V = .4116, F(3, 23,846) = 5,559.14				R^2_V = .4116, F(3, 23,846) = 5,559.14			
2		R^2_T = .4644, F(13, 23,836) = 1,589.50				R^2_T = .4553, F(5, 23,844) = 3,986.14				R^2_T = .4440, F(4, 23,845) = 4,759.77			
		R^2_V = .4417, F(13, 23,836) = 1,450.33				R^2_V = .4315, F(5, 23,844) = 3,618.91				R^2_V = .4204, F(4, 23,845) = 4,323.43			
Δ 1 → 2		ΔR^2_T = .0300, ΔF(10, 23,836) = 133.33				ΔR^2_T = .0209, ΔF(2, 23,844) = 457.65				ΔR^2_T = .0096, ΔF(1, 23,845) = 410.55			
		ΔR^2_V = .0301, ΔF(10, 23,836) = 128.51				ΔR^2_V = .0199, ΔF(2, 23,844) = 417.36				ΔR^2_V = .0088, ΔF(1, 23,845) = 363.09			

Note. PA = physical abuse; SA = sexual abuse; MT = medical trauma; ND = natural disaster; WFV = witness to family violence; WCV = witness to community violence; EA = emotional abuse; CSA = caregiver substance abuse; CMI = caregiver mental illness; Dep = deprivation; CR = cumulative risk. Significant effects based on the relevant family-wise error rate (FWER) are bold. Individual risk model FWER $p < .004$; cumulative risk model FWER $p < .013$; DMAP model FWER $p < .010$. R^2_T represents value derived in the test sample (n = 11,890). R^2_V represents value derived in the validation sample (n = 11,960).
[a] Small effect .02 ≤ f^2 ≤ .24. [c] Large effect f^2 ≥ .40.

23,844) = 495.92, $p < .001$; validation: $\Delta R^2 = .015$, F(1, 23,844) = 472.38, $p < .001$] and T2 [test: $\Delta R^2 = .001$, F(1, 23,843) = 27.97, $p < .001$; validation: $\Delta R^2 = .001$, F(1, 23,843) = 32.83, $p < .001$], disinhibited externalizing symptoms at T1 [test: $\Delta R^2 = .011$, F(1, 23,844) = 496.22, $p < .001$; validation: $\Delta R^2 = .011$, F(1, 23,844) = 464.58, $p < .001$] and T2 [test: $\Delta R^2 = .0004$, F(1, 23,843) = 31.06, $p < .001$; validation: $\Delta R^2 = .001$, F(1, 23,843) = 34.32, $p < .001$], and antagonistic externalizing symptoms at T1 [test: $\Delta R^2 = .038$, F(1, 23,844) = 1,255.06, $p < .001$; validation: $\Delta R^2 = .031$, F(1, 23,844) = 1,026.04, $p < .001$] and T2 [test: $\Delta R^2 = .001$, F(1, 23,843) = 51.91, $p < .001$; validation: $\Delta R^2 = .0003$, F(1, 23,843) = 13.78, $p < .001$] than the cumulative risk model.

Discussion

In Study 2, we first hypothesized that all individual ACEs would be associated with more internalizing and disinhibited and antagonistic externalizing symptoms. This hypothesis was partially supported at T1, with eight of 10 ACEs associated with internalizing symptoms, two of 10 ACEs associated with disinhibited externalizing symptoms, and five of 10 ACEs associated with antagonistic externalizing symptoms, in the expected direction. Effect sizes for individual ACEs are small in magnitude, which is commensurate with results from studies of children in state custody (e.g., Tarren-Sweeney, 2008) as well as large epidemiological studies (e.g., Green et al., 2010; Kessler et al., 2010).

Some findings emerged as contrary to the first hypothesis. Most notable was the direction of a subset of the associations. Contrary to a robust literature (e.g., Loukas et al., 2003), exposure to caregiver substance abuse was related to *fewer* internalizing and externalizing symptoms. A similar but weaker pattern emerged for caregiver mental illness and to an even lesser extent for neglect. Of note, this pattern has been observed in other research involving children in state custody. For example, in a study designed to examine cumulative risk as a predictor of mental health symptoms in youth who were court ordered to out-of-home care due to substantiated maltreatment, Raviv et al. (2010) found small negative bivariate associations between caregiver alcohol abuse and youth depression symptoms, caregiver substance abuse and anxiety and youth externalizing symptoms, and neglect (lack of supervision and educational neglect) and youth externalizing symptoms. Drawing on work by Pears et al. (2008), Raviv et al. (2010) suggested that while exposure to experiences such as caregiver substance abuse and neglect does not promote positive functioning, certain profiles or patterns of maltreatment may be more strongly associated with symptoms than others. Children and adolescents in state custody are less commonly recruited into research studies than community and clinical

Table 7
Individual Risk, Cumulative Risk, and DMAP Predicting Disinhibited Externalizing Symptoms at T2

		Individual risk				DMAP				Cumulative risk			
Block	Predictor	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*
1	Age	.07 (.003)	[.06, .08]	.14[a]	**23.43**	.07 (.003)	[.06, .08]	.14[a]	**23.43**	.07 (.003)	[.06, .08]	.14[a]	**23.43**
	Gender	.18 (.02)	[.14, .22]	.04[a]	**8.98**	.18 (.02)	[.14, .22]	.04[a]	**8.98**	.18 (.02)	[.14, .22]	.04[a]	**8.98**
	Race	.05 (.02)	[.01, .09]	.01	2.19	.05 (.02)	[.01, .09]	.01	2.19	.05 (.02)	[.01, .09]	.01	2.19
	Ext	.54 (.005)	[.53, .55]	.69[c]	**112.75**	.54 (.005)	[.53, .55]	.69[c]	**112.75**	.54 (.005)	[.53, .55]	.69[c]	**112.75**
2	Age	.07 (.003)	[.06, .08]	.13[a]	**21.42**	.07 (.003)	[.06, .08]	.13[a]	**21.86**	.07 (.003)	[.06, .08]	.14[a]	**23.12**
	Gender	.17 (.02)	[.13, .21]	.04[a]	**8.41**	.17 (.02)	[.13, .21]	.04[a]	**8.78**	.17 (.02)	[.13, .21]	.04[a]	**8.54**
	Race	.02 (.02)	[−.02, .06]	.004	.73	.02 (.02)	[−.02, .06]	.006	1.18	.04 (.02)	[.00, .08]	.008	1.78
	Ext	.53 (.005)	[.52, .54]	.68[c]	**108.31**	.53 (.005)	[.52, .54]	.68[c]	**109.56**	.54 (.005)	[.53, .55]	.69[c]	**111.32**
	PA	.007 (.02)	[−.03, .05]	.002	.35								
	SA	.002 (.02)	[−.04, .04]	<.001	.14								
	MT	.008 (.03)	[−.05, .07]	.001	.28								
	ND	−.13 (.06)	[−.25, −.01]	−.01	−2.38								
	WFV	.007 (.02)	[−.03, .05]	.002	.42								
	WCV	.08 (.03)	[.02, .14]	.02[a]	**3.10**								
	Neglect	−.02 (.01)	[−.04, .00]	−.009	−1.53								
	EA	−.03 (.02)	[−.07, .01]	−.01	−1.64								
	CSA	−.04 (.01)	[−.06, −.02]	−.02[a]	**−3.60**								
	CMI	−.04 (.01)	[−.06, −.02]	−.02[a]	**−3.38**								
	Threat					.09 (.04)	[.01, .17]	.01	2.37				
	Dep					−.14 (.02)	[−.18, −.10]	−.04[a]	**−6.72**				
	CR									−.13 (.03)	[−.19, −.07]	−.02[a]	**−4.35**
1		R^2_T = .6361, F(4, 23,845) = 10,421.23				R^2_T = .6361, F(4, 23,845) = 10,421.23				R^2_T = .6361, F(4, 23,845) = 10,421.23			
		R^2_V = .6458, F(4, 23,845) = 10,868.71				R^2_V = .6458, F(4, 23,845) = 10,868.71				R^2_V = .6458, F(4, 23,845) = 10,868.71			
2		R^2_T = .6372, F(14, 23,835) = 2,990.58				R^2_T = .6369, F(6, 23,843) = 6,969.09				R^2_T = .6364, F(5, 23,844) = 8,346.18			
		R^2_V = .6478, F(14, 23,835) = 3,130.90				R^2_V = .6473, F(6, 23,843) = 7,293.35				R^2_V = .6468, F(5, 23,844) = 8,732.96			
Δ 1 → 2		ΔR^2_T = .0011, ΔF(10, 23,835) = 7.30				ΔR^2_T = .0007, ΔF(2, 23,843) = 24.22				ΔR^2_T = .0003, ΔF(1, 23,844) = 17.36			
		ΔR^2_V = .0020, ΔF(10, 23,835) = 13.32				ΔR^2_V = .0015, ΔF(2, 23,843) = 51.17				ΔR^2_V = .0010, ΔF(1, 23,844) = 67.93			

Note. Ext = externalizing symptoms at T1; PA = physical abuse; SA = sexual abuse; MT = medical trauma; ND = natural disaster; WFV = witness to family violence; WCV = witness to community violence; EA = emotional abuse; CSA = caregiver substance abuse; CMI = caregiver mental illness; Dep = deprivation; CR = cumulative risk. Significant effects based on the relevant family-wise error rate (FWER) are bold. Individual risk model FWER $p < .004$; cumulative risk model FWER $p < .013$; DMAP model FWER $p < .010$. R^2_T represents value derived in the test sample (n = 11,890). R^2_V represents value derived in the validation sample (n = 11,960).
[a] Small effect $.02 \leq f^2 \leq .24$. [c] Large effect $f^2 \geq .40$.

youth, and associations between ACEs and symptoms may differ in this unique sample. More research involving children in state custody is warranted to better understand the impact of ACEs on mental health and help stakeholders ensure that youth needs are met.

We conducted exploratory analyses to shed light on the ways that children and adolescents who have been exposed to caregiver substance abuse and mental illness may differ from the rest of our sample (see Table 3 in the online supplemental materials). Youth with exposure to caregiver substance abuse and mental illness were more likely to be female and White, and consistent with other research on children in state custody, youth were younger in age with a medium-to-large effect (Besinger et al., 1999). Given their younger age, these youth may have been removed from their homes by DCS before psychopathology emerged in their developmental course (Kessler et al., 2007). In fact, some research suggests that psychopathology associated with exposure to parental substance abuse is more likely to onset in adulthood (Benjet et al., 2010). This subset of youth may also assume emotional and instrumental caregiving behaviors for their substance abusing and/or mentally ill caregivers, which could temporarily suppress symptom expression (Burnett et al., 2006). In addition, early intervention by DCS may be a protective factor, preventing prolonged exposure to these ACEs, as well as other ACEs. Also consistent with research on children in state custody, youth with exposure to caregiver substance abuse and mental illness were more exposed to several other ACEs, including neglect and witnessing family violence (Besinger et al., 1999; Raviv et al., 2010; Seay & Kohl, 2013; see Table 3 in the online supplemental materials). The experience of children in state custody is likely characterized by a complex temporal sequence of increased exposure to, and subsequent protection from, ACEs. Small negative associations between physical abuse and emotional abuse and disinhibited externalizing symptoms were also found at T1. Given the various factors that could have contributed to these unexpected effects, these findings should be viewed with caution and require replication with this population and other populations of children exposed to ACEs in longitudinal designs.

Also contrary to hypothesis one, specificity between ACEs and symptoms increased over time. For example, despite numerous significant effects for internalizing symptoms at T1, only sexual abuse and emotional abuse remained significant predictors of internalizing symptoms at T2. Several factors likely contributed to our inability to observe meaningful growth in psychological symptoms due to ACEs over time. At the T1 assessment, youth with greater ACEs exposure may have approached a symptom ceiling, an effect which was likely exacerbated by our short follow-up period (six months or less between T1 and T2). Although small in magnitude, there was a mean reduction in externalizing symptoms from T1 to T2; it is possible that DCS

Table 8
Individual Risk, Cumulative Risk, and DMAP Predicting Antagonistic Externalizing Symptoms at T1

		Individual risk				DMAP				Cumulative risk			
Block	Predictor	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*
1	Age	.40 (.006)	[.39, .41]	.41[c]	**71.25**	.40 (.006)	[.39, .41]	.41[c]	**71.25**	.40 (.006)	[.39, .41]	.41[c]	**71.25**
	Gender	1.37 (.05)	[1.27, 1.47]	.17[a]	**29.86**	1.37 (.05)	[1.27, 1.47]	.17[a]	**29.86**	1.37 (.05)	[1.27, 1.47]	.17[a]	**29.86**
	Race	.96 (.05)	[.86, 1.06]	.11[a]	**19.13**	.96 (.05)	[.86, 1.06]	.11[a]	**19.13**	.96 (.05)	[.86, 1.06]	.11[a]	**19.13**
2	Age	.33 (.006)	[.32, .34]	.34[b]	**55.27**	.36 (.006)	[.35, .37]	.37[b]	**61.87**	.42 (.006)	[.41, .43]	.43[c]	**74.27**
	Gender	1.50 (.05)	[1.40, 1.60]	.19[a]	**33.10**	1.51 (.04)	[1.43, 1.59]	.19[a]	**33.60**	1.49 (.05)	[1.39, 1.59]	.19[a]	**32.41**
	Race	.77 (.05)	[.67, .87]	.09[a]	**15.68**	.86 (.05)	[.76, .96]	.10[a]	**17.54**	1.04 (.05)	[.94, 1.14]	.12[a]	**20.92**
	PA	.47 (.05)	[.37, .57]	.07[a]	**10.15**								
	SA	.49 (.04)	[.41, .57]	.08[a]	**12.55**								
	MT	.25 (.06)	[.13, .37]	.02[a]	**3.84**								
	ND	.12 (.13)	[−.13, .37]	.005	.97								
	WFV	.08 (.04)	[.00, .16]	.01	2.10								
	WCV	.93 (.06)	[.81, 1.05]	.09[a]	**16.01**								
	Neglect	.08 (.03)	[.02, .14]	.02[a]	2.29								
	EA	.27 (.04)	[.19, .35]	.05[a]	**6.07**								
	CSA	−.56 (.02)	[−.60, −.52]	−.15[a]	**−24.11**								
	CMI	−.02 (.03)	[−.08, .04]	−.004	−.57								
	Threat					3.39 (.09)	[3.21, 3.57]	.24[a]	**39.12**				
	Dep					−.74 (.05)	[−.84, −.64]	−.11[a]	**−15.90**				
	CR									1.34 (.07)	[1.20, 1.48]	.11[a]	**19.44**
1		R^2_T = .2353, F(3, 23,846) = 2,445.47				R^2_T = .2353, F(3, 23,846) = 2,445.47				R^2_T = .2353, F(3, 23,846) = 2,445.47			
		R^2_V = .2337, F(3, 23,846) = 2,424.16				R^2_V = .2337, F(3, 23,846) = 2,424.16				R^2_V = .2337, F(3, 23,846) = 2,424.16			
2		R^2_T = .3020, F(13, 23,836) = 793.37				R^2_T = .2840, F(5, 23,844) = 1,891.31				R^2_T = .2463, F(4, 23,845) = 1,947.93			
		R^2_V = .2952, F(13, 23,836) = 767.82				R^2_V = .2777, F(5, 23,844) = 1,833.43				R^2_V = .2466, F(4, 23,845) = 1,951.39			
Δ 1 → 2		ΔR^2_T = .0667, ΔF(10, 23,836) = 227.93				ΔR^2_T = .0487, ΔF(2, 23,844) = 810.90				ΔR^2_T = .0110, ΔF(1, 23,845) = 348.42			
		ΔR^2_V = .0615, ΔF(10, 23,836) = 207.84				ΔR^2_V = .0440, ΔF(2, 23,844) = 726.17				ΔR^2_V = .0129, ΔF(1, 23,845) = 408.73			

Note. PA = physical abuse; SA = sexual abuse; MT = medical trauma; ND = natural disaster; WFV = witness to family violence; WCV = witness to community violence; EA = emotional abuse; CSA = caregiver substance abuse; CMI = caregiver mental illness; Dep = deprivation; CR = cumulative risk. Significant effects based on the relevant family-wise error rate (FWER) are bold. Individual risk model FWER $p < .004$; cumulative risk model FWER $p < .013$; DMAP model FWER $p < .010$. R^2_T represents value derived in the test sample (n = 11,890). R^2_V represents value derived in the validation sample (n = 11,960).
[a] Small effect .02 ≤ f^2 ≤ .24. [b] Medium effect .25 ≤ f^2 < .39. [c] Large effect f^2 ≥ .40.

intervention at T1 contributed to the early amelioration of symptoms in some participants.

Consistent with hypothesis 2 and findings from Miller et al. (2018), exposure to threat predicted higher levels of internalizing and externalizing symptoms. The findings for deprivation were more complex; we found small positive associations with internalizing symptoms (previous research found no direct association) and a small *negative* association with externalizing symptoms (previous research found a positive association). Our threat composite incorporated ACEs used in previous work (e.g., physical, emotional, and sexual abuse and exposure to violence). We used a combination of ACEs (neglect, emotional abuse, caregiver substance abuse, and caregiver mental illness) to represent deprivation, but poverty has been most commonly used to represent deprivation in the literature (e.g., Busso et al., 2017; Lambert et al., 2017). Poverty is an experience that confers risk for exposure to a variety of ACEs, including threat and deprivation events (Sheridan & McLaughlin, 2014; McLaughlin, 2016). Differences in the way we operationalized deprivation likely contributed to inconsistencies between our findings and previous research. As described in the Discussion of Study 1, caregiver substance abuse and mental illness may also be better characterized as contexts that put youth at risk for the occurrence of ACEs, rather than ACEs themselves (see McLaughlin, 2016). Results of the LONGSCAN study showed that individuals experiencing chronic ACEs were more likely to have parents who were substance abusing or had depressive symptoms (Thompson et al., 2015). In the current study, youth exposed to caregiver substance abuse and mental illness were also more likely to have been exposed to several ACEs (e.g., neglect, witnessing family violence).

The DMAP and cumulative risk equations are nested versions of the individual risk equation, and accordingly, results from hypothesis 1 provide important contextual information to aid in interpreting results from hypotheses 2 and 3. Negative associations between caregiver substance abuse and mental illness and externalizing symptoms emerged in bivariate analyses and were sustained in regression analyses. When included in the deprivation composite, caregiver substance abuse reduced the magnitude of the positive association between deprivation and internalizing symptoms at T1. Partially consistent with hypotheses 3 and the body of literature on ACEs (Anda et al., 2006), higher cumulative risk scores were associated with more internalizing symptoms and antagonistic externalizing symptoms. As with the deprivation findings, the direction and magnitude of the cumulative risk scores were affected by caregiver substance abuse and mental illness variables.

General Discussion

The strong and pervasive association of ACEs with psychopathology is a major public health concern (Shonkoff et al., 2012). To advance research in this field, we compared three approaches

Table 9
Individual Risk, Cumulative Risk, and DMAP Predicting Antagonistic Externalizing Symptoms at T2

		Individual risk				DMAP				Cumulative risk			
Block	Predictor	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*	*b* (*SE*)	95% CI	β	*t*
1	Age	−.003 (.005)	[−.01, .01]	−.003	−.47	−.003 (.005)	[−.01, .01]	−.003	−.47	−.003 (.005)	[−.01, .01]	−.003	−.47
	Gender	.31 (.04)	[.23, .39]	.04[a]	**7.65**	.31 (.04)	[.23, .39]	.04[a]	**7.65**	.31 (.04)	[.23, .39]	.04[a]	**7.65**
	Race	.34 (.04)	[.26, .42]	.04[a]	**7.84**	.34 (.04)	[.26, .42]	.04[a]	**7.84**	.34 (.04)	[.26, .42]	.04[a]	**7.84**
	Ext	.63 (.006)	[.62, .64]	.70[c]	**111.50**	.63 (.006)	[.62, .64]	.70[c]	**111.50**	.63 (.006)	[.62, .64]	.70[c]	**111.50**
2	Age	<−.001 (.006)	[−.01, .01]	<−.001	−.07	.001 (.006)	[−.01, .01]	.001	.18	.008 (.005)	[.00, .02]	.009	1.42
	Gender	.40 (.04)	[.32, .48]	.06[a]	**9.60**	.38 (.04)	[.30, .46]	.05[a]	**9.32**	.36 (.04)	[.28, .44]	.05[a]	**8.89**
	Race	.36 (.04)	[.28, .44]	.05[a]	**8.04**	.36 (.04)	[.28, .44]	.05[a]	**8.23**	.39 (.04)	[.31, .47]	.05[a]	**8.85**
	Ext	.61 (.006)	[.60, .62]	.68[c]	**104.24**	.61 (.006)	[.60, .62]	.68[c]	**106.14**	.62 (.006)	[.61, .63]	.69[c]	**110.01**
	PA	.23 (.04)	[.15, .31]	.04[a]	**5.64**								
	SA	.16 (.03)	[.10, .22]	.03[a]	**4.59**								
	MT	.006 (.06)	[−.11, .12]	<.001	.10								
	ND	−.16 (.12)	[−.40, .08]	−.008	−1.40								
	WFV	.05 (.03)	[−.01, .11]	.01	1.45								
	WCV	.02 (.05)	[−.08, .12]	.002	.41								
	Neglect	.11 (.03)	[.05, .17]	.03[a]	**3.77**								
	EA	−.04 (.04)	[−.12, .04]	−.008	−.99								
	CSA	−.07 (.02)	[−.11, −.03]	−.02[a]	**−3.49**								
	CMI	.04 (.02)	[.00, .08]	.01	1.60								
	Threat					.77 (.08)	[.61, .93]	.06[a]	**9.64**				
	Dep					.008 (.04)	[−.07, .09]	.001	.19				
	CR									.55 (.06)	[.43, .67]	.05[a]	**9.16**
1		R^2_T = .5091, F(4, 23,845) = 6,182.33				R^2_T = .5091, F(4, 23,845) = 6,182.33				R^2_T = .5091, F(4, 23,845) = 6,182.33			
		R^2_V = .5084, F(4, 23,845) = 6,164.42				R^2_V = .5084, F(4, 23,845) = 6,164.42				R^2_V = .5084, F(4, 23,845) = 6,164.42			
2		R^2_T = .5153, F(14, 23,835) = 1,810.27				R^2_T = .5139, F(6, 23,843) = 4,201.12				R^2_T = .5128, F(5, 23,844) = 5,020.24			
		R^2_V = .5126, F(14, 23,835) = 1,790.21				R^2_V = .5110, F(6, 23,843) = 4,152.03				R^2_V = .5107, F(5, 23,844) = 4,977.01			
Δ 1 → 2		ΔR^2_T = .0062, ΔF(10, 23,835) = 30.67				ΔR^2_T = .0048, ΔF(2, 23,843) = 117.69				ΔR^2_T = .0037, ΔF(1, 23,844) = 183.07			
		ΔR^2_V = .0042, ΔF(10, 23,835) = 20.43				ΔR^2_V = .0026, ΔF(2, 23,843) = 63.06				ΔR^2_V = .0023, ΔF(1, 23,844) = 112.28			

Note. Ext = externalizing symptoms at T1; PA = physical abuse; SA = sexual abuse; MT = medical trauma; ND = natural disaster; WFV = witness to family violence; WCV = witness to community violence; EA = emotional abuse; CSA = caregiver substance abuse; CMI = caregiver mental illness; Dep = deprivation; CR = cumulative risk. Significant effects based on the relevant family-wise error rate (FWER) are bold. Individual risk model FWER $p < .004$; cumulative risk model FWER $p < .013$; DMAP model FWER $p < .010$. R^2_T represents value derived in the test sample (n = 11,890). R^2_V represents value derived in the validation sample (n = 11,960).
[a] Small effect $.02 \leq f^2 \leq .24$. [c] Large effect $f^2 \geq .40$.

for operationalizing ACEs—the individual risk approach, the DMAP approach, and the cumulative risk approach—to predict internalizing and externalizing symptoms at two timepoints in a large sample of youth in state custody. Results suggest that selection of an optimal approach is dependent on empirical objectives.

Statistically, we found individual risk to be the strongest approach for identifying youth at risk for developing psychopathology. Although our comparisons of incremental predictive ability a priori favored the fuller models, it was not predetermined that the fuller models would account for *significantly* more variance than the reduced models. Results revealed that individual risk accounted for significantly more variance in internalizing and externalizing symptoms than the DMAP and cumulative risk approaches, and DMAP accounted for significantly more variance in internalizing and externalizing symptoms than the cumulative risk approach, and these findings were replicated when all three models were reestimated in a validation sample. Of note, individual risk in the current study is a deviation from the approach used in some previous research (e.g., Cutajar et al., 2010; Taylor et al., 2018). We entered multiple ACEs simultaneously in a single model, which is an amalgam of the classic individual risk approach (i.e., examining ACEs in separate models) and the cumulative risk approach (i.e., collapsing ACEs into a single score in a single model).

Conceptually, the individual risk approach provided specific information about the association between ACEs and psychological symptoms, and when we combined ACEs to create cumulative risk scores, the effects of specific ACEs were lost. In the current sample of children in state custody, complete information proved to be important to understand nuanced associations between ACEs and symptoms. For example, at both T1 and T2, the cumulative risk approach indicated a small positive association between ACEs and antagonistic externalizing symptoms, concealing a negative association with caregiver substance abuse and minimizing the magnitude of the positive effects of other ACEs. Frameworks that organize experiences based on underlying dimensions that impact development offer promise for advancing research on the mechanisms by which ACEs affect development. In our test of DMAP, several heterogeneous effects were combined in the deprivation composite, obscuring unique associations between ACEs and psychological symptoms, but the threat composite preserved both the direction and the magnitude of effects. Taken together, DMAP may offer a useful middle ground between the individual and cumulative risk approaches, explaining more variance in psychological symptoms than the cumulative risk approach and providing a framework for understanding ACEs with distinct influences on child development. Nevertheless, more research is needed to understand associations between DMAP and psychopathology, and

consideration of additional dimensions for organizing ACEs and their effect on development is warranted.

Notwithstanding notable strengths, including conducting one of the first empirical confirmations of DMAP using a variety of ACEs in a large sample of children and adolescents in state custody, the current research has several limitations. First, the CANS consists of single items capturing domains of interest, and single-item measures should be interpreted with caution (Nunnally, 1967). Importantly, however, CANS service providers integrate information from multiple sources to rate each item, and reliability and validity have been supported in the literature (Anderson et al., 2003; Lyons et al., 2004). In Study 2, we used these single items to derive internalizing, disinhibited externalizing, and antagonistic externalizing symptom composites, which are consistent with the HiTOP model (Kotov et al., 2017). We used ACEs items both individually (i.e., individual risk model) and together (i.e., DMAP and cumulative risk model). Second, the neglect item represents exposure 30 days prior to the CANS assessment, which may have contributed to underestimations of lifetime exposure to neglect. Third, data on timing of ACEs were unavailable. Child age and developmental stage at the onset and offset of ACEs have important implications for risk for psychopathology (Thornberry et al., 2001). Fourth, 10 ACEs do not represent all of the adverse experiences a child might face during development, despite providing a larger and more diverse representation of ACEs than previous work. Research suggests that experiences such as parental criminal behavior, parental death, and parental divorce are also associated with risk for psychopathology (e.g., Burt et al., 2008; Kendler et al., 1992; Murray & Farrington, 2008). Finally, the characteristics of the study sample have implications for study findings. Multiple ACEs and comorbid psychological symptoms are more common in youth in state custody than the general population. Greater specificity between ACEs and outcomes may be observed in community-based samples (McMahon et al., 2003). Moreover, research suggests unique relations between prospective and retrospective reports of ACEs and psychopathology. Some research suggests a positive relation between DCS involvement and psychopathology (Bernard et al., 2015), whereas other studies have found the impact of ACEs on psychopathology to be stronger and longer lasting when assessed by retrospective report (Cohen et al., 2001). Unreported adversities could ultimately be more harmful, as they may continue or escalate without intervention (Kendall-Tackett & Becker-Blease, 2004). As such, the current research should be replicated, both with unique samples and by integrating multiple methods for assessing ACEs in youth into study designs. Generalization of the current findings may be limited to youth in DCS custody.

The findings of the current study may guide opportunities for future research. First, in addition to the contribution of ACEs on the development of psychopathology, some research suggests that preexisting psychological symptoms may put youth at risk for exposure to ACEs (Schaefer et al., 2018). Exposure to ACEs and psychopathology at multiple timepoints should be collected in future research so that reverse causality can be thoroughly tested. Second, additional empirical support is needed to confirm the structure of DMAP. The present study provides support for the face validity of DMAP, and Sheridan et al. (2020) provide an initial statistical confirmation using network analysis. However, subject matter experts in Study 1 did not achieve full consensus in categorizing ACEs as threat or deprivation, and neglect unexpectedly clustered with threat variables in Sheridan et al.'s (2020) data-driven approach. In addition, McLaughlin, Sheridan, and colleagues (McLaughlin et al., 2014; Sheridan & McLaughlin, 2014) describe ACEs as existing on a continuum (i.e., a dimensional model) of threat and deprivation, rather than exclusively being one or the other (Sheridan & McLaughlin, 2014, Figure 1). For example, caregivers with depression exhibit less warm and responsive, and more disengaged, parenting behaviors (deprivation) and more intrusive parenting behaviors (threat) than nondepressed parents (Lovejoy et al., 2000). Having a substance-abusing caregiver also likely predisposes youth to experiences of both threat and deprivation (Young et al., 2007). Future research exploring how ACEs may reflect both threat and deprivation to varying degrees will be important. Third, important differences in the direct effects of threat and deprivation on psychopathology emerged in the current research, but DMAP posits that threat and deprivation events have different mediators of similar outcomes (e.g., Miller et al., 2018; Platt et al., 2018). As such, future work should extend the current research to use an empirically derived DMAP model to examine potential mechanisms by which DMAP explains psychopathology, in addition to direct relations between ACEs and outcomes. Research examining other developmental correlates of DMAP may provide additional intervention targets for children exposed to threat and deprivation events (e.g., verbal abilities and executive function for youth exposed to deprivation; Sheridan & McLaughlin, 2016). Defining ACEs, operationalizing their measurement, and integrating this work with other burgeoning areas of ACEs research (e.g., passive and evocative gene-environment correlations; Pittner et al., 2019) are essential in understanding risk for psychopathology in children with ACEs (McLaughlin, 2016).

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