Check for updates

Received: 29 August 2022 | Revised: 16 November 2022 | Accepted: 4 January 2023

DOI: 10.1002/dev.22372

Developmental Psychobiology WILEY

RESEARCH ARTICLE

Development and validation of the Dimensional Inventory of Stress and Trauma Across the Lifespan (DISTAL): A novel assessment tool to facilitate the dimensional study of psychobiological sequelae of exposure to adversity

Emily M. Cohodes[1] | Sarah McCauley[1] | Jasmyne C. Pierre[1] | H. R. Hodges[1] | Jason T. Haberman[1] | Isabel Santiuste[1] | Marisa K. Rogers[1] | Jenny Wang[1] | Jeffrey D. Mandell[2] | Dylan G. Gee[1]

[1]Department of Psychology, Yale University, New Haven, Connecticut, USA

[2]Program in Computational Biology and Bioinformatics, Yale University, New Haven, Connecticut, USA

Correspondence
Emily M. Cohodes and Dylan G. Gee, Department of Psychology, Yale University, 2 Hillhouse Avenue, New Haven, CT 06520, USA.
Email: emily.cohodes@yale.edu; dylan.gee@yale.edu

Funding information
National Institutes of Health; Brain and Behavior Research Foundation; Jacobs Foundation; National Science Foundation; The Society for Clinical Child and Adolescent Psychology; Society for Research in Child Development; American Association of University Women; American Psychological Association; Philanthropic Educational Organization

Abstract

Decades of research underscore the profound impact of adversity on brain and behavioral development. Recent theoretical models have highlighted the importance of considering specific features of adversity that may have dissociable effects at distinct developmental timepoints. However, existing measures do not query these dimensions in sufficient detail to support the proliferation of this approach. The Dimensional Inventory of Stress and Trauma Across the Lifespan (DISTAL) was developed with the aim to thoroughly and retrospectively assess the timing, severity (of exposure and reaction), type, persons involved, controllability, predictability, threat, deprivation, proximity, betrayal, and discrimination inherent in an individual's exposure to adversity. Here, we introduce this instrument, present descriptive statistics drawn from a sample of $N = 187$ adults who completed the DISTAL, and provide initial information about its psychometric properties. This novel measure facilitates the expansion of research focused on assessing the relative impact of exposure to key dimensions of adversity on the brain and behavior across development.

KEYWORDS
adolescence, adversity, childhood, frontolimbic circuitry, infancy, multidimensional, trauma screening, traumatic stress

1 | INTRODUCTION

Decades of research inform our understanding of the pernicious effects of exposure to environmental adversity for individuals across the lifespan. The extant literature underscores a pathway from exposure to adversity to increased risk for developing cognitive, social, emotional, and physical health problems (Boyce, 2007; Shonkoff et al., 2012), as well as altered neurobiological development (McLaughlin et al., 2019). Exposure to adversity is also widespread, with an estimated 70% of adults experiencing a traumatic event that satisfies criterion A of the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5) definition of posttraumatic disorder (PTSD; American Psychiatric Association, 2013; Kessler et al., 2017).

To date, the majority of studies assessing the effects of exposure to adversity on the brain and behavior have employed a categorical approach (i.e., classifying individuals as either trauma-exposed or non-trauma-exposed and assessing group differences in neural structure and function or clinical symptomatology). However, exposure to adversity does not have a universal effect on all individuals across development (Gabbay et al., 2004), which may be due, in part, to the

 © 2023 Wiley Periodicals LLC.

vast heterogeneity in adversity exposure (Cohodes et al., 2021). As such, though foundational research has demonstrated the profound and lasting consequences of exposure to early-life adversity on the developing brain and behavior (see VanTieghem & Tottenham, 2017 for a review), researchers have increasingly advocated for novel multivariate, multidimensional approaches to assessing the effects of early-life adversity that allow us to capture this complexity (Cohodes et al., 2021; Ellis et al., 2009, 2022; Manly et al., 2001; McLaughlin & Sheridan, 2016; McLaughlin et al., 2021; Sheridan & McLaughlin, 2014).

Specifically, recent dimensional approaches have focused on highlighting experiential, environmental, and temporal factors that may substantially affect the association between exposure to adversity and subsequent vulnerability across multiple domains of functioning (e.g., Herzog et al., 2020). For example, theoretical models propose that the developmental timing and presence—or absence—of elements of threat and/or deprivation, caregiver involvement, predictability, and controllability may moderate the association between exposure to adversity and both clinical and neurodevelopmental outcomes (Cohodes et al., 2021; Ellis et al., 2022; Fox et al., 2010; Gee & Casey, 2015; Glynn & Baram, 2019; Hambrick et al., 2019; Luby et al., 2019; Manly et al., 2001; McLaughlin & Sheridan, 2016; McLaughlin et al., 2014; Sheridan & McLaughlin, 2014; Teicher et al., 2016; Tottenham & Sheridan, 2010). To highlight one particular model that has been the subject of extensive empirical research to date, the Dimensional Model of Adversity and Psychopathology (DMAP; McLaughlin & Sheridan, 2016) proposes a clear distinction between the neurodevelopmental sequelae of exposure to adverse early experiences characterized by threat (i.e., exposure to stressors that may cause harm to an individual's physical integrity) versus those characterized by deprivation (i.e., exposure to stressors that are characterized by the absence of species-expected inputs in the environment). McLaughlin et al. (2021) have proposed that exposure to threat is more likely to be associated with alterations in fear learning, whereas exposure to deprivation is more likely to impact neural proliferation and pruning.

Despite considerable empirical support for the importance of dimensional conceptualization of adversity, there has been recent discourse about the utility of such an approach. Most notably, Smith and Pollak (2021) have cautioned against reliance on "specificity models," arguing that it is frequently challenging to delineate boundaries between subtypes of exposure, that co-occurrence of specific types of adversity confounds understanding of isolated effects of exposure to specific dimensions of experience, and that there is currently limited evidence for differentiated outcomes following exposure to specific types of adversity. Though fruitful debate in this area is ongoing, we believe that precise assessment of these multilevel factors that may moderate the impact of adversity on neurodevelopmental and clinical outcomes is a critical step in understanding the etiology of stress-related psychopathology, and, further, that several of the challenges related to dimensional models that have been identified in the extant literature may stem from limited measurement specificity (e.g., lack of clear and comprehensive screener questions to define exposure to a particular type of event). Novel assessment tools that sufficiently capture exposure to specific elements of adversity are required to facilitate this line of research, and, further, to generate empirical evidence that is likely to advance the important scientific discussion regarding the utility of dimensional models.

1.1 | A brief review of existing assessment tools

Given burgeoning interest in studying the neural and behavioral sequelae of exposure to adversity, assessment of exposure to adversity has become increasingly common. However, existing assessment tools are limited in their capacity to adequately assess the dimensionality of experience that may be needed to delineate nuanced associations between adversity exposure and neurodevelopmental and clinical outcomes. The adverse childhood experiences (ACE) study (Felitti et al., 1998), a landmark epidemiological study demonstrating a dose-dependent association between the number of adverse childhood experiences and detrimental outcomes across multiple domains of development, contributed to increased societal awareness about the impact of exposure to maltreatment and other forms of childhood adversity. The ACE study spawned many additional studies examining associations between ACE scores and numerous outcomes across the lifespan (see Hughes et al., 2017 for a review). An individual's ACE score is derived from responses to a series of binary questions that query an individual's exposure to abuse and neglect during childhood, including emotional, physical, and sexual abuse, as well as exposure to substance abuse, mental illness, violent treatment of an individual's family member, incarceration for criminality, parental separation, divorce, and death. This score has proven to be a simple index of multiplicity (number of adversity exposures) that is a salient predictor of health outcomes across the life course (e.g., Anda et al., 2006). While questionnaire-based assessment of individuals' exposure to ACEs remains a frequently utilized methodological approach to capturing early-life adversity exposure, sole reliance on this construct presents several important methodological issues, including assessment of a restricted range of exposures and a lack of timing or severity-related information (K. E. Smith & Pollak, 2020; Teicher & Parigger, 2015). Though longitudinal studies have demonstrated that ACE scores are associated with group-level differences in health outcomes, this lack of dimensional specificity is likely to substantially limit the degree to which ACE scores can accurately predict health outcomes on an individual level (Baldwin et al., 2021), therefore drawing attention to the need for more specific measurement tools.

Several other questionnaire-based assessments of early-life adversity, including the Childhood Trauma Questionnaire (CTQ; D. P. Bernstein et al., 1997) and the Childhood Experience of Care and Abuse Questionnaire (CECA-Q; N. Smith et al., 2002), aim to capture the severity—rather than multiplicity—of exposure to one or multiple types of adversity. The CTQ is a widely used self-report measure that queries individuals' history of exposure to abuse and neglect across five subtypes—emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect—and assesses the relative severity of exposure to each type. The CTQ yields scores representing the

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

severity of an individual's exposure to several types of maltreatment during childhood, which can also be summed to create a composite score. The CECA-Q is a self-report assessment of lack of parental care, as well as exposure to physical and sexual abuse, which yields subscale scores reflecting the severity of an individual's exposure to each type of aforementioned adversity. Though these measures provide an important metric of the severity of an individual's exposure to specific types of adversity, neither of these adult self-report instruments captures timing-related information.

Various additional questionnaire-based measures fill this gap by specifically assessing the degree to which an individual's exposure to multiple different types of adversity varies across the course of development (e.g., the Life Stressors Checklist; LSC; Wolfe et al., 1996; the University of California Los Angeles Posttraumatic Stress Disorder Reaction Index; UCLA PTSD RI; Steinberg et al., 2004; Pynoos & Steinberg, 2017; the Traumatic Life Events Questionnaire; TLEQ; Kubany et al., 2000; the Stressful Life Events Screening Questionnaire; SLESQ; Goodman et al., 1998; the Life Events Checklist; LEC; Weathers et al., 2013; and the Maltreatment and Abuse Chronology of Exposure Scale; MACE; Teicher & Parigger, 2015). The UCLA PTSD RI, the MACE, and the SLESQ represent important progress in the development of assessment tools that facilitate the dimensional study of the effects of adversity on brain and behavior in that they capture timing-related information for several types of adversity across development. Further, the UCLA PTSD RI and the LEC capture an individual's level of exposure to adversity (i.e., directly experienced, witnessed, learned about), and the MACE yields both a multiplicity and severity score for several types of adversity exposure.

Several additional interview-based assessment tools afford collection of more detailed information regarding an individual's life history of exposure to adversity. Of particular relevance to the present study, the Dimensions of Stressful Events Rating Scale (DOSE; Spilsbury et al., 2008), for example, assesses several key aspects of focal adversities identified by a child, including the degree to which the child felt like they had control over the event or suffered lasting losses due to the event. As another example, the UCLA Life Stress Interview (see Hammen et al., 1985) queries the occurrence of specific types of events, developmental timing, severity, and independence of the event (i.e., whether the event was related to characteristics of the individual), and involves detailed reliability coding by a team of trained interviewers and raters following data collection. Though these measures are indeed more dimensional assessments of exposure to adversity in that they query specific features of adversity, to date, no retrospective report-based measure of exposure to adversity across the lifespan adequately and simultaneously assesses the degree to which an individual was exposed to key dimensions of interest that have been theorized to contribute to variability in neurodevelopmental and clinical outcomes following exposure to trauma. The need for an assessment tool to capture heterogeneity in exposure to adversity motivated the development of the Dimensional Inventory of Stress and Trauma Across the Lifespan (DISTAL), a novel adaptation of the UCLA PTSD RI (Pynoos & Steinberg, 2017).

1.2 | Selection of dimensions of interest for a novel assessment tool

The goal of the present study was to develop a novel, retrospective measure of exposure to adversity that would systematically query multiple dimensions of exposure. Development of this measure was motivated by our desire to facilitate research focused on parsing heterogeneity in exposure to adversity to better understand the neurodevelopmental and behavioral sequelae of exposure across development. Therefore, the selection of dimensions of interest to be queried in the DISTAL was based on a comprehensive review of both animal and human studies that have aimed to identify specific dimensions of adversity that may confer risk or resilience across development. When possible, we focused on literature examining effects of exposure to adversity on frontolimbic circuitry due to the sensitivity of this circuitry to the effects of stress, as well as literature suggesting that alterations in frontolimbic circuitry may mediate associations between stress exposure and increased risk for psychopathology (see VanTieghem & Tottenham, 2017 for a review). Based on this review of the literature (summarized briefly below and more comprehensively in Cohodes et al., 2021), the following factors were selected: timing of adversity (i.e., age of onset of adversity, duration of adversity, chronicity of adversity), type of adversity (including whether there was an element of threat or deprivation inherent in exposure), severity of adversity, persons involved in adversity, controllability of adversity, predictability of adversity, whether there was perceived betrayal inherent in adversity (and, if so, the specific type of betrayal experienced: personal, caregiver, or systemic), whether there was perceived discrimination inherent in adversity, and an individual's proximity to adversity (both physical and psychological).

These dimensions represent important features of exposure to adversity that may either exacerbate, or conversely, attenuate the effects of adversity on the developing brain and behavior, as is briefly reviewed below. Although we review dimensions separately—and despite considerable interest in delineating the neurodevelopmental sequelae of exposure to specific features of stress, in isolation—we underscore that multidimensional assessment of exposure to adversity across development facilitates more complex analyses of probable interactions among multiple different features of exposure to adversity. It is important to note that we hypothesize that the *interaction* between multiple dimensions of exposure is likely to be most salient in the prediction of neurobiological change following exposure to adversity, and therefore we purposefully refrain from describing certain factors as buffering (i.e., positive) or exacerbating (i.e., negative).

1.3 | Type of adversity

Although relatively less is known about the impact of specific types of adversity on neurobiological outcomes, extensive prior research has compared behavioral and clinical outcomes associated with different types of adversity (e.g., sexual abuse vs. physical abuse) to test whether

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

specific exposures may be associated with differential outcomes (e.g., Cicchetti & Toth, 1995; Estrada et al., 2021; Gomez et al., 2017; Khan et al., 2015; McCoy, 2013). Relatedly, as previously described, an emerging line of research suggests that exposures characterized by *threat* versus *deprivation* may have differential effects on neurobiological (e.g., Colich et al., 2020) and clinical outcomes (e.g., Miller et al., 2018). Taken together, the extant literature suggests that assessment of both exposure to relatively broad categories of adversity (e.g., sexual abuse vs. community violence), as well as exposures characterized by specific features that denote exposure to a particular type of adversity (e.g., threat vs. deprivation) will yield insight into the mechanisms by which specific types of adversity exposure differentially affect the developing brain.

1.4 | Timing of adversity

Frontolimbic circuitry undergoes dynamic changes across development (Casey et al., 2019; Gabard-Durnam et al., 2014; Gee et al., 2013, 2018; Hare et al., 2008; M. Wu et al., 2016), and current theory posits that the specific developmental phase, or phases, during which an individual is exposed to adversity likely impacts both short- and long-term outcomes (Eiland & Romeo, 2013; Gee & Casey, 2015; Gee & Cohodes, 2021). Indeed, a growing body of research suggests that adversity that occurs at different times during development can have different effects on the structure and function of frontolimbic circuitry, as well as on behavioral and psychiatric outcomes (Andersen et al., 2008; Cameron, 2001; Russotti et al., 2021; Sabatini et al., 2007; Schalinski et al., 2016). In addition to capturing specific ages at which exposures to adversity occur across development, additional indices such as the chronicity and duration of exposure (i.e., number of days, months, years during which an individual was exposed to a specific type of adversity, or to any type of adversity) may be important factors to consider when assessing the impact of exposure to adversity on frontolimbic circuitry (for a detailed review see Andersen et al., 2008; Doom & Cicchetti, 2020; Gee & Casey, 2015; Tottenham & Sheridan, 2010). Naturally occurring variability in the duration of exposure to adversity has allowed researchers to begin to test the long-term effects of exposure to chronic adversity that occurs early in life. Several studies suggest that the number of years that an individual is exposed to adversity is linearly related to neurobiological outcomes (De Bellis & Kuchibhatla, 2006; De Bellis et al., 1999; McCrory et al., 2013; Tupler & De Bellis, 2006).

1.5 | Severity of adversity

The severity of exposure to adversity has frequently been studied in relation to neural and clinical outcomes following exposure. To date, research has found that higher severity of childhood exposure to adversity is associated with reduced gray matter volumes across the limbic system and higher amygdala activation to negatively valenced emotional stimuli (Dannlowski et al., 2012; Fan et al., 2014; Graham et al., 2015; Pechtel et al., 2014; Veer et al., 2015). Additionally, an emerging line of research suggests that perceptions of severity of adversity across development may exert unique effects on neural outcomes (J. Wu et al., 2018). To date, many studies have relied on either an index of the number of exposures to adversity reported by an individual as a proxy for severity, or, alternatively, a single metric of self-reported subjective severity, in line with a broader literature examining associations between subjective stress severity and psychological outcomes (Espejo et al., 2011). Recent research suggests that distinct indices of adversity severity may differentially relate to the development of psychopathology following exposure (Baldwin et al., 2019; Danese & Widom, 2020, 2021). Therefore, assessment of multiple distinct constructs of severity—including assessment of both the subjective severity of exposure to an event versus subjective severity of an individual's reaction to the event—may shed light on dissociable mechanisms by which exposure to severe adversity may impact neural outcomes.

1.6 | Persons involved in adversity, including caregiver involvement in adversity

Both extensive animal and limited human studies of exposure to adversity involving caregivers (either in the form of caregiver-absent or caregiver-perpetrated events) point to the deleterious effect of caregiver-related exposure to adversity on the development of frontolimbic circuitry (for a comprehensive review, see Callaghan et al., 2019). The majority of human studies of the effects of caregiver involvement in adversity on neurobiological outcomes in offspring focus on exposure to parental deprivation, which represents a major deviation from species-expected caregiving (Nelson et al., 2007; Tottenham, 2012). Parental deprivation has been theorized to trigger a developmental cascade (Masten & Cicchetti, 2010) that is associated with alterations in the development of the HPA axis, amygdala hyperactivity (Tottenham et al., 2011) and larger amygdala volumes (Mehta et al., 2009; Tottenham et al., 2010), reduced gray matter volumes (Sheridan et al., 2012), and early maturation of frontoamygdala circuitry (Gee et al., 2013; Herzberg et al., 2021). In parallel, evidence also suggests that parental presence during exposure to adversity—in a non-perpetrating role—may serve to buffer the neurobiological effects of adversity by reducing amygdala reactivity (Gee et al., 2014; Hostinar et al., 2015). Beyond caregiver involvement, research suggests that the presence of peers (e.g., Peters et al., 2011) during exposure to adversity may affect the relative impact of a given event, motivating the assessment of involvement of a host of individuals in exposure to adversity.

1.7 | Controllability of adversity

Counter to the conventional notion of stress as universally harmful, studies of stressor controllability in rodents and human adults provide evidence for the idea that the experience of controllable stress may buffer some individuals against the negative effects of subsequent stress exposure (Amat et al., 2010; Hartley et al., 2014; Maier & Watkins, 2010). The ability to exert control over a stressor appears to moderate the effects of stress on frontolimbic circuitry (Amat et al.,

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

2008; Boeke et al., 2017; Maier & Watkins, 2010), such that exposure to controllable stress facilitates adaptive coping and promotes long-term resilience. Though this literature is in its infancy, an emerging line of research on the neural mechanisms of stressor controllability in humans suggests that the ability to exert control over an adverse experience appears to engage ventral striatum, medial prefrontal cortex, and lateral and basal nuclei of the amygdala to promote reduced physiological reactivity and increased adaptive coping behavior during exposure to subsequent stress (Boeke et al., 2017; Collins et al., 2014).

1.8 | Predictability of adversity

Based on a rich translational literature and a limited but growing human developmental literature, the predictability of early life experience has been proposed as a key determinant of the development of frontolimbic circuitry, as well as mental health outcomes (Baram et al., 2012). Exposure to fragmented care and higher rates of unpredictability in maternal cues early in development is associated with cognitive and affective dysfunction in rodent offspring (Baram et al., 2012; Brunson, 2005). From a neurobiological perspective, rodents exposed to unpredictable care showed greater c-Fos expression in the basolateral amygdala, relative to animals raised in typical conditions without exposure to unpredictable maternal care (Malter Cohen et al., 2013). Rodents exposed to unpredictable shock exhibited higher levels of stress hormones in the hypothalamus, amygdala, and thalamus, relative to rodents who had been exposed to predictable shock (Tsuda et al., 1989). Taken together, these findings delineate possible biological mechanisms by which exposure to more unpredictable environments may alter brain development.

1.9 | Betrayal inherent in adversity

Though exposure to betrayal, specifically, has yet to be mapped onto neurodevelopmental outcomes, the broader clinical literature suggests that exposure to betrayal inherent in adversity may confer risk for adverse developmental and health outcomes (e.g., Edwards et al., 2012). Betrayal trauma theory (Freyd, 1994, 1997) and attachment theory (Bowlby, 1958) assert that a child's formation and maintenance of attachment relationships to caregivers is critical to their emotional and physical wellbeing, as well as survival (R. Bernstein & Freyd, 2014). Betrayal trauma theory proposes that because children are compelled to align themselves with caregivers, children form attachment relationships to caregivers regardless of the quality of care that they receive (Delker et al., 2018; Freyd, 1994). However, although the ability to form an attachment despite a caregiver's betrayal cues may be adaptive to an infant given their biological need to attach to a caregiver for safety and survival, the development of non-secure attachment relationships to caregivers as a result may be less adaptive later in life. In addition to caregiver betrayal, the extant clinical literature highlights exposure to systemic betrayal as a potent predictor of deleterious sequelae of exposure to adversity (see Goldsmith et al., 2014; C. P. Smith & Freyd, 2014).

1.10 | Discrimination inherent in adversity

Initial human studies highlight that exposure to adversity during which an individual perceives discrimination may impact psychobiological responses to adversity (Allen et al., 2019; Clark et al., 2018; Currie et al., 2019; Matheson et al., 2016, 2019). Exposure to racial discrimination has been found to be robustly associated with poorer mental health outcomes (Elias & Paradies, 2016; Priest et al., 2013), and further, a nascent body of literature has begun to examine the neurobiological sequelae of exposure to adversity characterized by discrimination. Though the neurobiological impacts of discrimination may vary depending on the specific nature of discrimination experienced, the neural effects of exposure to racial discrimination appear to be similar to those of chronic social stress, with evidence for associations between exposure to racial discrimination and alterations in functioning of the HPA axis, and specific brain regions implicated in emotion regulation such as the ventromedial prefrontal cortex (Fani et al., 2021) and anterior cingulate cortex (see Berger & Sarnyai, 2015 for a comprehensive review).

1.11 | Proximity to adversity

Finally, a growing body of research suggests that an individual's physical proximity to adversity impacts their short- and long-term likelihood of developing PTSD following exposure, particularly among events that are directly experienced (Frans et al., 2018; May & Wisco, 2016). Further, though preliminary, several studies lend initial evidence for an association between proximity to adversity and neurobiological processes underlying fear acquisition (Faul et al., 2020; Ganzel et al., 2007).

2 | MEASURE DEVELOPMENT AND STRUCTURE

The DISTAL contains two subsections that are broadly modeled as a novel adaptation of the UCLA PTSD RI: a screener for potential exposure to multiple types of adversity at three levels of exposure (directly experiencing, witnessing, and learning about the event happening to a close person), and event-specific modules that query additional details about each event endorsed. Screener questions were developed with the goal of capturing a comprehensive list of exposures to adversity; screener questions were only included if they had the potential to result in report of adversity (i.e., "normative" stressors were not the subject of screening questions). As is reviewed in further detail below, screening questions were administered to query individuals' exposure to 24 distinct types of adversity at multiple levels of exposure. For each type of adversity that was endorsed on the screening form, participants were then asked to report on the cumulative list of ages at which they experienced this particular type of adversity. For each age that individuals reported experiencing a particular type of adversity, specific timing-related, experiential, and environmental features of the exposure were queried.

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

See Supporting Information for details of screening and event-specific module questions.

3 | THE PRESENT STUDY

The present study provides an overview of the development of the DISTAL and presents descriptive data from the first $N = 187$ adult participants to be administered this novel measure. In addition, we describe initial psychometric properties of this tool by assessing content and convergent validity in subsamples of participants who completed additional target measures (see Section 5.6).

With regard to tests of content validity, we hypothesized a general pattern whereby severity of exposure to adversity in childhood (both in general and within a given type of exposure [e.g., physical abuse]) as assessed by the DISTAL would be significantly positively associated with severity of exposure to adversity in childhood as assessed by the CTQ. Further, we expected that exposure to a higher number of adversity events across the life course—as well as a greater degree of exposure to each novel dimension of interest—would be significantly positively associated with trauma-related symptomatology.

4 | METHOD

4.1 | Participants

The present study utilized a community sample of $N = 187$ participants recruited as part of a broader study using flyers and online advertisements in the New Haven, Connecticut area. To be included in the current study, all participants had to be between the ages of 18 to 30 years and to have completed the DISTAL (see Supporting Information for exclusion criteria). As presented in Table 1, 61.5% of participants reported their sex assigned at birth to be female ($n = 115$), and 37.4% of participants reported their sex assigned at birth to be male ($n = 70$); information about participant sex was missing for $n = 2$ participants. Participants' ages ranged from 18.05 to 30.96 years ($M = 23.01$, $SD = 3.45$). 45.3% of the sample identified as Non-Hispanic White ($n = 77$), 20.6% identified as Asian ($n = 35$), 14.7% identified as Black or African American ($n = 25$), 10.6% identified as Hispanic or Latinx ($n = 18$), 7.1% identified as Other ($n = 12$), and 1.8% reported that they preferred not to report their race or ethnicity ($n = 3$). Participants reported having completed an average of 15.03 years of education (SD = 2.53; range = 0–20 years). A subset of participants ($n = 165$) also completed the CTQ (target of content validity), and a subset of participants ($n = 152$) also completed the Trauma Symptoms Checklist (TSC-40; target of convergent validity).

TABLE 1 Demographic information

Variables		(N = 187)
Age	Mean ± SD	23.01 ± 3.45
	Min-Max	18.05–30.96
	Median (IQR)	22.33 (5.78)
	Missing	1 (.5%)
Sex	Male	70 (37.4%)
	Female	115 (61.5%)
	Missing	2 (1.1%)
Race/ethnicity	Non-Hispanic White	77 (45.3%)
	Hispanic/Latinx	18 (10.6%)
	Black/African American	25 (14.7%)
	Asian	35 (20.6%)
	Other/not listed	12 (7.1%)
	Prefer not to answer	3 (1.8%)
	Missing	17 (9.1%)
Income	<$5,000	9 (5.9%)
	$5,000–$11,999	8 (5.3%)
	$12,000–$15,999	6 (3.9%)
	$16,000–$24,999	16 (10.5%)
	$25,000–$34,999	13 (8.6%)
	$35,000–$49,999	14 (9.2%)
	$50,000–$74,999	31 (20.4%)
	$75,000–$99,999	7 (4.6%)
	>$100,000	48 (31.6%)
	Missing	34 (18.3%)
Years of education	Mean ± SD	15.03 ± 2.53
	Min-Max	0–20
	Median (IQR)	15 (3)
	Missing	15 (8.0%)

4.2 | Administration procedure

The Institutional Review Board at Yale University approved all study procedures, and all participants provided written informed consent prior to engaging in study procedures. During an in-person laboratory session, the DISTAL was administered by trained doctoral students and research assistants supervised by a clinical psychologist. Given the complexity of this data collection, all interviewers were trained extensively via in-vivo observations. Participants also completed a demographic survey as well as a questionnaire battery including targets for establishing content and convergent validity. Participants were compensated $25/hour for their time.

5 | Materials

5.1 | Demographics

Participants were asked to report on their age, sex assigned at birth, race/ethnicity, and level of education.

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

5.2 | Dimensional Inventory of Stress and Trauma Across the Lifespan

Participants completed the DISTAL to assess exposure to multiple dimensions of adversity across the lifespan. As described above, the DISTAL systematically queries participants' exposure to 24 different broad categories of exposure to adversity (e.g., physical abuse, domestic violence, serious accidental injury, forced displacement) and contains modules designed to assess specific dimensions of exposure to adversity, including timing-related factors, severity, person involvement, and whether exposures were characterized by threat, deprivation, controllability, predictability, betrayal, or discrimination. For both event and reaction severity ratings, participants were asked to rate the severity of the event and their reaction to the event, respectively, on a scale of 0 (not at all) to 8 (very, very much). Participants were guided through a detailed description of the rating scale, and the following anchors were provided: 0 (not at all), 1, 2 (a little bit), 3, 4 (some), 5, 6 (a lot), 7, and 8 (very, very much). In addition, sample event descriptions were provided for 0 and 8.

5.3 | Childhood Trauma Questionnaire

Participants completed the CTQ (Bernstein et al., 1994) to assess exposure to abuse in childhood. The CTQ is a self-report questionnaire consisting of 40 items that assess the degree to which an individual was exposed to sexual, physical, and emotional abuse, as well as emotional and physical neglect, during childhood that has been shown to have good internal consistency, stability over time, and criterion validity (Bernstein et al., 1994). Participants rated each item (e.g., "People in my family said hurtful or insulting things to me") on a scale ranging from *Never True* to *Very Often True*. The present study utilized all five severity subscale scores (sexual abuse, physical abuse, emotional abuse, emotional neglect, and physical neglect), as well as the total severity score for the CTQ. All five subscales, as well as the total score, demonstrated good internal consistency (Cronbach's alpha for the sexual abuse, physical abuse, emotional abuse, emotional neglect, and physical neglect subscales were .67, .92, .85, .86, and .65, respectively, and Cronbach's alpha for the total score was .77).

5.4 | Trauma Symptoms Checklist

Participants completed the TSC-40 (Elliott & Briere, 1992) to assess the relative frequency of distress arising from exposure to trauma in both childhood and adulthood. The TSC-40 is a self-report questionnaire consisting of 40 items representing trauma-related symptomatology (e.g., "Uncontrollable crying") rated on a four-point Likert scale from 0 (*Never*) to 3 (*Often*). The instrument is unique in that it does not assess diagnostic status related to PTSD, but rather it assesses broad trauma-related symptomatology including posttraumatic stress symptoms, mood-related symptoms, and interpersonal difficulties in the previous two months. The TSC-40 has been shown to have good internal consistency and validity. In addition to a total score representing the frequency of global symptomatology related to past trauma, the TSC-40 produces six subscales (dissociation, anxiety, depression, trauma history, sleep disturbances, and sexual problems). The present study utilized the total score (Cronbach's alpha = .90).

5.5 | Quality assurance of data

Given the complexity of this phenotypic collection, to assure the highest standards of data quality, a team of trained undergraduate research assistants performed systematic quality assurance on all data generated by the DISTAL. First, following completion of interviews by a team of trained evaluators, research assistants reviewed all interview data in detail to ensure correct categorization of all exposures, and to verify concordance of all data within a given module. As necessary, following a detailed coding manual (to be made available upon request) and in conference with the first, second, or last authors, a team of highly trained research assistants recoded data to ensure that all interviewers were reliably categorizing exposures and accurately endorsing specific elements of exposure. Of note, in order to preserve the original clinical interview documentation, coders did not alter the original interview data (paper interview form) and instead created an exact copy that was marked up to document changes. Following creation of this "update" form, a final, clean version of the interview—reflecting coding-related changes—was created for entry. Following completion of the coding phase, all interview data were entered by two separate research assistants (masked to one another's entries). All discrepancies between the two entries were resolved by a third enterer, in consultation with the first or second author. Following entry, all coding changes and documentation, as well as all three rounds of entry and error resolution, were reviewed in a final round of quality assurance by a trained research assistant.

5.6 | Analytic strategy

5.6.1 | Derivation of dimensions of interest

The multivariate nature of the DISTAL affords the derivation of numerous, specific dimensions of interest based on researcher preference and specific research question. Here, we present descriptive data for several indices of interest to illustrate the range of dimensional variables made accessible by this novel instrument.

Based on participants' endorsements of screener questions, the number of distinct types of adversity to which an individual was exposed was summed as an index of *type* of adversity exposure. In order to derive three separate metrics of *chronicity*, the number of ages, months, and days, respectively, that an individual was exposed to any type of adversity, was summed. The *age of onset* of an individual's exposure to adversity was calculated by identifying the earliest age at which a participant reported exposure to any type of adversity. Averages of *average event severity, worst event severity, average*

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

reaction severity, and *worst reaction severity* were computed by averaging these specific scores across all completed modules. *Physical proximity* was calculated by summing the number of events during which an individual was either close enough to sense (e.g., hear, see, smell) specific aspects of a given event or during which they reported being in the same house or room as the person to whom this event occurred (including themselves). *Caregiver involvement* was operationalized as the number of exposures during which a caregiver was involved as a perpetrator and/or affected caregiver. The number of events characterized by *threat*, *deprivation*, *controllability*, *predictability*, *betrayal (both generalized and caregiver-specific)*, and *discrimination* were summed to create indices reflecting prevalence of these elements of exposure in an individual's history of exposure to adversity.

5.6.2 | Assessment of content and convergent validity

In addition to the primary DISTAL subscales described above, several additional DISTAL indices were calculated to facilitate the assessment of content validity via associations between the DISTAL and the CTQ (Bernstein et al., 1994), an established measure of childhood exposure to abuse and neglect, among the subset of participants who completed both the DISTAL and CTQ ($n = 165$). Pearson correlations were calculated to examine relations between DISTAL subscales and CTQ subscale and total scores. Specifically, the following DISTAL subscale scores were created to mirror CTQ subscale scores: severity of sexual abuse exposure in childhood, severity of physical abuse exposure in childhood, severity of emotional abuse exposure in childhood, severity of neglect exposure in childhood[1], and severity of abuse and neglect exposure in childhood (a composite severity rating of all aforementioned adversity categories). Note that only exposures to adversity that participants reported directly experiencing on the DISTAL were considered in the creation of DISTAL subscale scores (i.e., exposures that participants witnessed or learned about were not included) in order to parallel the assessment of exposure to adversity via the CTQ.

A Pearson correlation was also calculated to examine the relation between total number of lifetime traumatic exposures, as assessed by the DISTAL, and a theoretically relevant construct to establish convergent validity with trauma-related symptomatology (assessed via the TSC-40; Elliott & Briere, 1992) among the subset of participants who completed both the DISTAL and the TSC-40 ($n = 152$). In addition, in order to assess the utility of examining isolated dimensions of exposure (assessed by the DISTAL), a Pearson correlation was calculated to examine the relation between each dimension of interest (e.g., number of ages of exposure, number of events characterized by predictability) and trauma-related symptomatology.

[1] Due to the fact that the DISTAL does not query emotional and physical neglect separately, distinct emotional and physical neglect subscale scores were not created.

6 | RESULTS

6.1 | Descriptive statistics for all dimensions of interest

Table 2 presents descriptive statistics for all dimensions of interest: type, chronicity, age of onset, average event severity, worst event severity, average reaction severity, worst reaction severity, physical proximity, caregiver involvement, threat, deprivation, controllability, predictability, betrayal (both generalized and caregiver-specific), and discrimination.

Participants reported experiencing an average of 20.05 exposures to adversity events across their lifetime to date (SD = 17.49; range = 0–104), and an average exposure to 8.41 distinct types of events (e.g., sexual abuse, serious accidental injury; SD = 4.37; range = 0–21). With regard to chronicity of exposure, participants reported an average of 9.22 distinct ages during which they were exposed to adversity (SD = 5.50; range = 0–29), for an average of 692.41 days across their lifetime (SD = 1298.45; range = 0–7656), and for an average of 86.55 months across their lifetime (SD = 127.35; range = 0–854). The average onset of exposure to adversity—across all adversity types queried—was 8.01 years of age (SD = 5.14; range = 0–26).

With regard to participants' report of the severity of their exposure to adversity, the average event severity of all events reported across participants' lifetimes to date was 4.83 (SD = 1.24; range = 0–8), and the average worst event severity of all events was 5.34 (SD = 1.38; range = 0–8). The average reaction severity of all events was 4.55 (SD = 1.24; range = 1.80–7.44), and the average worst reaction severity was 5.17 (SD = 1.36; range = 1.80–8).

Participants reported direct physical proximity to an average of 16.65 exposures to adversity (SD = 25.39; range = 0–189). Participants reported exposure to an average of 4.73 exposures (SD = 11.17; range = 0–70) that involved a caregiver in a perpetrating role.

Participants reported exposure to an average of 1.94 events (SD = 3.63; range = 0–21) that they perceived to be characterized by controllability, an average of 3.45 events (SD = 7.10; range = 0–41) that they perceived to be characterized by predictability, an average of 4.57 events (SD = 8.26; range = 0–48) that they perceived to be characterized by betrayal, an average of 1.67 events (SD = 5.07; range = 0–34) that they perceived to be characterized by caregiver betrayal, and an average of 1.42 events (SD = 3.60; range; 0–26) that they perceived to be characterized by discrimination. The average number of events that participants perceived to be characterized by threat was 4.34 (SD = 6.66; range = 0–50), and the average number of events that they perceived to be characterized by deprivation was 2.60 (SD = 7.45; range = 0–53).

6.2 | Preliminary evidence of content and convergent validity of selected DISTAL subscales

Correlations between DISTAL subscales (i.e., severity of sexual abuse exposure in childhood, severity of physical abuse exposure in

10992302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

TABLE 2 Descriptive statistics for Dimensional Inventory of Stress and Trauma Across the Lifespan (DISTAL) variables of interest

Variable	Description of variable	Descriptive statistics ($N = 187$)	
Exposure to adversity	Number of lifetime exposures to adversity	Mean ± SD	20.05 ± 17.49
		Min-Max	0–104
		Median (IQR)	15.00 (20)
		Missing or N/A	0 (0%)
Exposure to adversity event types	Number of distinct types of adversity to which participant was exposed	Mean ± SD	8.41 ± 4.37
		Min-Max	0–21
		Median (IQR)	8 (6)
		Missing or N/A	0 (0%)
Chronicity (ages/years)	Number of distinct ages during which participant was exposed to adversity	Mean ± SD	9.22 ± 5.50
		Min-Max	0–29
		Median (IQR)	9 (7)
		Missing or N/A	0 (0%)
Chronicity (days)	Number of days during which participant was exposed to adversity	Mean ± SD	692.41 ± 1,298.45
		Min-Max	0–7656
		Median (IQR)	163 (683)
		Missing or N/A	0 (0%)
Chronicity (months)	Number of months during which participant was exposed to adversity	Mean ± SD	86.55 ± 127.35
		Min-Max	0–854
		Median (IQR)	38 (88)
		Missing or N/A	0 (0%)
Age of onset	Age at which participant experienced first exposure to adversity	Mean ± SD	8.01 ± 5.14
		Min-Max	0–26
		Median (IQR)	7 (6)
		Missing or N/A	1 (0.5%)*
Average event severity	Mean severity of all exposures to adversity (rated by participant)	Mean ± SD	4.83 ± 1.24
		Min-Max	0–8
		Median (IQR)	5 (1.77)
		Missing or N/A	6 (3.2%)
Worst event severity	Mean worst severity of all exposures to adversity (rated by participant)	Mean ± SD	5.34 ± 1.38
		Min-Max	0–8
		Median (IQR)	5.58 (1.72)
		Missing or N/A	18 (9.6%)
Average reaction severity	Mean severity of reaction to all exposures to adversity (rated by participant)	Mean ± SD	4.55 ± 1.24
		Min-Max	1.80–7.44
		Median (IQR)	4.51 (1.85)
		Missing or N/A	18 (9.6%)
Worst reaction severity	Mean worst severity of reaction to all exposures to adversity (rated by participant)	Mean ± SD	5.17 ± 1.36
		Min-Max	1.80–8
		Median (IQR)	5.14 (1.94)
		Missing or N/A	22 (11.8%)

(Continues)

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

TABLE 2 (Continued)

Variable	Description of variable	Descriptive statistics ($N = 187$)	
Physical proximity	Number of exposures to adversity during which participant was in the same room/house as victim and/or was close enough to see/hear/smell aspects of the event as it happened (rated by participant)	Mean ± SD	16.65 ± 25.39
		Min-Max	0–189
		Median (IQR)	10 (18)
		Missing or N/A	2 (1.1%)
Caregiver involvement	Number of exposures to adversity perpetrated by a caregiver and/or during which a caregiver was involved as an affected caregiver (rated by participant)	Mean ± SD	4.73 ± 11.17
		Min-Max	0–70
		Median (IQR)	0 (4)
		Missing or N/A	0 (0%)
Controllability	Number of exposures to adversity characterized by controllability (rated by participant)	Mean ± SD	1.94 ± 3.63
		Min-Max	0–21
		Median (IQR)	0 (3)
		Missing or N/A	38 (20.3%)
Predictability	Number of exposures to adversity characterized by predictability (rated by participant)	Mean ± SD	3.45 ± 7.10
		Min-Max	0–41
		Median (IQR)	1 (3)
		Missing or N/A	46 (24.6%)
Traumatic exposures characterized by betrayal	Number of exposures to adversity characterized by betrayal (rated by participant)	Mean ± SD	4.57 ± 8.26
		Min-Max	0–48
		Median (IQR)	0 (5)
		Missing or N/A	28 (15.0%)
Caregiver betrayal	Number of exposures to adversity characterized by caregiver betrayal (rated by participant)	Mean ± SD	1.67 ± 5.07
		Min-Max	0–34
		Median (IQR)	0 (0)
		Missing or N/A	0 (0%)
Discrimination	Number of exposures to adversity characterized by discrimination (rated by participant)	Mean ± SD	1.42 ± 3.60
		Min-Max	0–26
		Median (IQR)	0 (1)
		Missing or N/A	16 (8.6%)
Threat	Number of exposures to adversity characterized by threat (rated by participant)	Mean ± SD	4.34 ± 6.66
		Min-Max	0–50
		Median (IQR)	2 (6)
		Missing or N/A	28 (15%)
Deprivation	Number of exposures to adversity characterized by deprivation (rated by participant)	Mean ± SD	2.60 ± 7.45
		Min-Max	0–53
		Median (IQR)	0 (1)
		Missing or N/A	28 (15%)

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

TABLE 3 Bivariate correlations among Dimensional Inventory of Stress and Trauma Across the Lifespan (DISTAL)-derived and Childhood Trauma Questionnaire (CTQ)-derived indices of childhood abuse and neglect ($n = 165$)

	Factor	2	3	4	5	6	7	8	9	10	11
1.	Severity of childhood sexual abuse (DISTAL)	0.10	0.22**	−0.01	0.40**	0.38**	0.11	0.22**	0.13	0.10	0.26**
2.	Severity of childhood physical abuse (DISTAL)	–	0.10	0.83**	0.82**	0.00	0.27**	0.25**	0.32**	0.45**	0.34**
3.	Severity of childhood emotional abuse (DISTAL)	–	–	0.10	0.57**	0.21**	0.24**	0.60**	0.34**	0.21**	0.46**
4.	Severity of childhood neglect (DISTAL)	–	–	–	0.78**	0.03	0.26**	0.30**	0.34**	0.56**	0.34**
5.	Severity of childhood abuse and neglect (DISTAL)	–	–	–	–	0.21**	0.35**	0.53**	0.45**	0.52**	0.57**
6.	Severity of childhood sexual abuse (CTQ)	–	–	–	–	–	0.33**	0.36**	0.20**	0.22**	0.52**
7.	Severity of childhood physical abuse (CTQ)	–	–	–	–	–	–	0.52**	0.39**	0.46**	0.68**
8.	Severity of childhood emotional abuse (CTQ)	–	–	–	–	–	–	–	0.62**	0.53**	0.86**
9.	Severity of childhood emotional neglect (CTQ)	–	–	–	–	–	–	–	–	0.61**	0.82**
10.	Severity of childhood physical neglect (CTQ)	–	–	–	–	–	–	–	–	–	0.76**
11.	Severity of childhood abuse and neglect (CTQ)	–	–	–	–	–	–	–	–	–	–

*$p < .05$; **$p < .01$; ***$p < .001$.

TABLE 4 Correlations between dimensions of exposure (DISTAL) and trauma-related symptomatology (TSC-40)

Dimension of exposure (DISTAL)	Trauma-related symptomatology (TSC-40)
1. Number of exposures	0.30***
2. Unique event exposures	0.28***
3. Number of ages of exposure	0.24**
4. Number of months of exposure	0.17*
5. Average reaction severity	0.19*
6. Worst reaction severity	0.19*
7. Caregiver involvement	0.18*
8. Predictability	0.29**
9. Controllability	0.18*
10. Betrayal	0.27**
11. Caregiver betrayal	0.19*
12. Threat	0.32***
13. Deprivation	0.28**

*$p < .05$; **$p < .01$; ***$p < .001$.

childhood, severity of emotional abuse exposure in childhood, severity of neglect exposure in childhood, and severity of abuse and neglect exposure in childhood [a composite severity rating of all aforementioned adversity categories]) and CTQ subscales (i.e., severity of sexual abuse in childhood, severity of physical abuse in childhood, severity of emotional abuse in childhood, severity of emotional neglect in childhood, severity of physical neglect in childhood) and total score were broadly in line with our expectations, thus supporting the convergent validity of the DISTAL (see Table 3 for full correlation matrix).

In addition, consistent with hypotheses, the total number of lifetime exposures to adversity was significantly positively associated with trauma-related symptomatology, $r(151) = 0.30$, $p < .001$ (see Table 4). As is also depicted in Table 4, specific dimensions of adversity exposure were significantly positively correlated with trauma-related symptomatology. Specifically, the total number of unique event exposures (i.e., type) was significantly positively correlated with trauma-related symptomatology, $r(152) = 0.28$, $p < .001$. Two separate indices of chronicity—the number of ages at which a participant was exposed to adversity and the number of months during which a participant was exposed to adversity—were significantly positively associated with trauma-related symptomatology, $r(152) = 0.24$, $p = .003$, and $r(152) = 0.17$, $p = .042$, respectively. Two indices of the severity of participants' reactions to events were also associated with trauma-related symptomatology: average reaction severity and worst reaction severity were both associated with trauma-related symptomatology, $r(147) = 0.19$, $p = .025$, and $r(144) = 0.19$, $p = .026$, respectively. The degree to which a participant's exposure to adversity was characterized by caregiver involvement was significantly positively associated with trauma-related symptomatology, $r(152) = 0.18$, $p = .026$, as was the degree to which a participant's exposure to adversity was associated with each of the following elements: predictability ($r(122) = 0.29$, $p = .001$), controllability ($r(128) = 0.18$, $p = .041$), betrayal ($r(137) = 0.27$, $p = .002$), caregiver betrayal ($r(152) = 0.19$, $p = .019$), threat ($r(137) = 0.32$, $p < .001$), and deprivation ($r(137) = 0.28$, $p = .001$).

7 | DISCUSSION

The present study provides initial descriptive and psychometric information for the DISTAL, a novel measure that assesses whether exposures to adversity are characterized by specific dimensions. Preliminary data collected from $N = 187$ adult participants who completed the DISTAL suggest that this novel instrument is poised to capture substantial variability in the degree to which exposures to adversity are characterized by dimensional indices of interest: type, chronicity, age of onset, severity, proximity, caregiver involvement,

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

controllability, predictability, betrayal, threat, and deprivation. In addition, initial psychometric analyses suggest that the DISTAL demonstrates excellent content and convergent validity via associations between DISTAL subscales and established indices of childhood trauma exposure, in addition to associations between DISTAL subscales and trauma-related symptomatology.

The DISTAL can facilitate research that has the potential to greatly expand upon our current understanding of the ways in which stress and adversity affects the developing brain and mental health across development. Though there has been increasing focus on the utility of employing dimensional models to probe the lifelong effects of exposure to adversity, the majority of research to date has either utilized a categorical approach or has focused on one or two isolated dimensions of adversity. Moreover, existing research on dimensions of adversity has often relied on researcher-defined types of adversity, rather than querying participants about their own experiences with a given experiential element of adversity or empirically deriving key dimensions of exposure (Pollak & K. E. Smith, 2021). Advances in the field's understanding of the dynamic associations between exposure to specific indices of adversity and both brain and behavioral development are contingent upon the development of measurement tools that systematically query the presence of numerous dimensions of adversity and that better assess experiential elements and individuals' subjective experiences. To our knowledge, the DISTAL is the first such measure to query a broad range of facets of exposure to adversity across the lifespan.

We note that the specific dimensions presented here represent a subset of the variables of interest that could be derived from the extremely rich phenotypic data generated by the DISTAL. Though there is likely important information to be gained from analyzing associations between exposure to an isolated feature of adversity and a specific outcome of interest, major breakthroughs in our understanding of the ways in which stress and adversity "gets under the skin" will likely require multivariate and data-driven approaches that examine the relative importance of exposure to specific clusters of features of adversity (i.e., interactions between specific dimensions of interest). For example, though it is of great interest, here we do not present age- or development-specific dimensions (e.g., number of adversity exposures characterized by caregiver involvement in early childhood), and future studies that utilize the DISTAL will be well positioned to branch into this critical area of investigation. We also note that the DISTAL was developed with the intention for expansion and adaptation of this tool to encompass new dimensions that emerge as potentially relevant in the ever-growing literature examining associations between exposure to adversity and neurodevelopment. It is our intention and hope that researchers will adapt the current version of the DISTAL to query further dimensions of interest that may be specific to targeted protocols.

In initially determining how to optimally structure the DISTAL, we made several methodological decisions that should be noted here as they reflect an opportunity for flexible use of the measure by other research groups. First, we decided to query the presence of specific dimensions of interest at the level of exposure to a particular type of adversity at a given age. For example, the DISTAL assesses the degree to which an individual's exposure to a particular type of adversity (e.g., physical abuse) at a particular age (e.g., age 5) was characterized by a particular feature (e.g., predictability). Following this example, the DISTAL documents whether an individual's exposure to physical abuse at age 5 was characterized by predictability. This was a specific determination, reflecting our belief that developmental stage during which an individual is exposed to a given stressor is of critical importance, as well as our desire to develop a measure that produces a dataset with rich age-related information. We piloted several alternative versions of the DISTAL that utilized different approaches such as querying the relative frequency of dimensions of interest at each instance of exposure within a given age; we ultimately determined that the version presented here reflected the most parsimonious balance between interview time, resources, and richness of data. Future studies that aim to examine the role of exposure to specific features of adversity at an even more granular developmental level (e.g., in early childhood) may choose to adapt the current structure of the DISTAL to facilitate this aim. Second, we made the intentional decision to only present participants with screener questions that had the potential to result in reports of exposure to adversity that would satisfy Criterion A of the DSM-5's criteria for PTSD (American Psychiatric Association, 2013). As such, we did not include screening questions that only had the potential to result in reports of "normative" stressors (e.g., regarding divorce, unemployment). We acknowledge that the utility of Criterion A in determining the relative severity of a given exposure—or, more specifically, whether an exposure to stress should be considered traumatic (and a viable "seed" for the development of PTSD) or "normative" in nature—has garnered significant criticism (see Friedman, 2013; Pai et al., 2017). As such, the specific protocol for determining which events reported in response to DISTAL screening questions are considered "traumatic" versus "normative" is the responsibility of the research group utilizing this measure and should be based on specific research questions and aims of a given project. Third, though the basis of our motivation for developing the DISTAL was indeed rooted in our interest in examining the impact of exposure to early-life adversity characterized by specific dimensions, we are also interested in examining patterns of exposure across the lifespan (i.e., into early adulthood) in order to better understand how exposure to specific aspects of adversity early in life has the potential to affect later patterns of exposure and resulting symptomatology, and to test whether associations between adversity and psychobiological sequelae are specific to or stronger for adversity experienced at a given stage of development. As such, we decided to examine exposure to adversity through age 30 (or through the participant's current age, if younger than 30) in the present sample; however, the DISTAL is designed to be flexible and can be adapted to fit the exact needs of a given study. For example, researchers could choose to only examine exposure to adversity up until a particular age (e.g., age 18 if a study were solely focused on the impact of exposure to dimensions of adversity in childhood and adolescence).

A core motivation in developing the DISTAL was to more accurately capture the complexity of exposure to adversity; therefore, we chose

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

to multiply count exposures that satisfied the criteria for more than one distinct type of traumatic exposure. For example, an individual's report of exposure to domestic violence in childhood that resulted in separation from a primary caregiver due to incarceration would be multiply counted as an exposure to both domestic violence and caregiver separation. Individual researchers utilizing the measure may choose to, alternatively, employ a "best fit" approach in which events are only counted once and researchers determine which "type" of event best captures a given exposure.

We acknowledge that, though it generates unparalleled dimensional data, the DISTAL requires significant time and clinical resources to complete. The resources required to yield rich dimensional data regarding an individual's exposure to adversity reflect a field-wide trade-off between ease of assessment and complexity of datasets derived from such data collection. Administration of the current version of the DISTAL presented here is likely to be appropriate for research groups with the clinical resources necessary to train and supervise a team of interviewers to query an individual's complex exposures in sufficient detail, and to provide appropriate clinical resources and supervision. Future research should focus on developing a range of both interview-based and self-report tools that assess a range of informative dimensions of exposure (including, but not limited to, those presented here) in order to facilitate dimensional assessment of exposure to adversity across a range of research settings. In addition, over time, empirical work that utilizes tools such as the DISTAL will likely yield improved understanding of which dimensions of adversity exposure may be most indicative of later functioning; such findings will certainly inform revisions of such instruments to minimize participant and researcher burden.

Finally, here we present initial validation evidence for the adult version of the DISTAL, which is based entirely on adults' retrospective report of exposure to adversity across the lifespan. Recent studies have identified low rates of concordance between retrospective and prospective reports of exposure to adversity, though concordance has been found to be higher for studies employing interviews, rather than questionnaires (Baldwin et al., 2019). Our group is currently in the process of examining initial descriptive statistics for versions of the DISTAL developed for use with children and adolescents, and their parents, respectively. Validation of these additional versions, and ultimately, their use in prospective, longitudinal studies will yield additionally rich information about the ways in which exposure to stress and adversity influences the developing brain. We also note that the present sample included a sample of adults who were relatively highly educated. Further, the broader study from which the present sample was drawn excluded participants on the basis of a range of factors that may be associated with exposure to adversity (e.g., suicidality, active substance use, use of psychotropic medication), thus limiting the degree to which the current validation effort may be broadly applicable to an unrestricted sample of adults. Future validation efforts of the DISTAL should aim to include a more socioeconomically diverse sample with a broader range of psychiatric profiles and fewer exclusion criteria in order to assess the utility of dimensional assessment in a general sample.

In conclusion, here we present initial descriptive statistics for the DISTAL, a newly developed instrument that queries the presence of specific attributes of adversity that the extant literature suggests may be highly relevant for the developing brain and behavior. It is our hope that this measure facilitates a new wave of highly dimensional research on the neurodevelopmental and behavioral sequelae of exposure to specific features of adversity. This work has the potential to fundamentally shift the way in which we conceptualize trajectories of risk following exposure to adversity, as well as to inform the development of targeted prevention and intervention efforts for individuals affected by adversity.

ACKNOWLEDGMENTS

This work was supported by the National Institutes of Health (NIH) Director's Early Independence Award (DP5OD021370), Brain & Behavior Research Foundation (National Alliance for Research on Schizophrenia and Depression; NARSAD) Young Investigator Award, Jacobs Foundation Early Career Research Fellowship, The Society for Clinical Child and Adolescent Psychology (Division 53 of the American Psychological Association) Richard "Dick" Abidin Early Career Award and Grant, and National Science Foundation (NSF) CAREER Award (BCS-2145372) to Dylan G. Gee; and NSF GRFP Award (NSF DGE1752134), The Society for Clinical Child and Adolescent Psychology (Division 53 of the American Psychological Association) Donald Routh Dissertation Grant, the American Psychological Foundation Elizabeth Munsterberg Koppitz Child Psychology Graduate Fellowship, a Dissertation Funding Award from the Society for Research in Child Development, a Dissertation Research Award from the American Psychological Association, an American Dissertation Fellowship from the American Association of University Women (AAUW), and a Scholar Award granted by the International Chapter of the Philanthropic Educational Organization (P.E.O. Foundation) to Emily M. Cohodes. The authors gratefully acknowledge current and former lab members who assisted with data collection: Paola Odriozola, Sahana Kribakaran, Sadie Zacharek, and Camila Caballero; research assistants who contributed to coding interviews and data management: Lindiwe Mayinja, Reta Behnam, Nathalie Eid, Elizabeth Kitt, Jordan Foster, Cristina Nardini, Georgia Spurrier, Brandon Lopez, and Sterling Strother; and Mary Margaret Schroeder, who contributed to table creation for the manuscript.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Research data are not shared due to sensitive nature of data derived from clinical interviews.

ORCID

Emily M. Cohodes https://orcid.org/0000-0002-0167-3392
Sarah McCauley https://orcid.org/0000-0003-4619-0156
Jasmyne C. Pierre https://orcid.org/0000-0003-2010-6278
H. R. Hodges https://orcid.org/0000-0001-9228-6365

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Marisa K. Rogers https://orcid.org/0000-0001-7939-3561
Jeffrey D. Mandell https://orcid.org/0000-0002-3839-2543
Dylan G. Gee https://orcid.org/0000-0002-3685-2710

REFERENCES

Allen, A. M., Thomas, M. D., Michaels, E. K., Reeves, A. N., Okoye, U., Price, M. M., Hasson, R. E., Syme, S. L., & Chae, D. H. (2019). Racial discrimination, educational attainment, and biological dysregulation among midlife African American women. *Psychoneuroendocrinology*, *99*, 225–235. https://doi.org/10.1016/j.psyneuen.2018.09.001

Amat, J., Aleksejev, R. M., Paul, E., Watkins, L. R., & Maier, S. F. (2010). Behavioral control over shock blocks behavioral and neurochemical effects of later social defeat. *Neuroscience*, *165*(4), 1031–1038. https://doi.org/10.1016/j.neuroscience.2009.11.005

Amat, J., Paul, E., Watkins, L. R., & Maier, S. F. (2008). Activation of the ventral medial prefrontal cortex during an uncontrollable stressor reproduces both the immediate and long-term protective effects of behavioral control. *Neuroscience*, *154*(4), 1178–1186. https://doi.org/10.1016/j.neuroscience.2008.04.005

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C. H., Perry, B. D., Dube, S. R., & Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European Archives of Psychiatry and Clinical Neuroscience*, *256*(3), 174–186.

Andersen, S. L., Tomada, A., Vincow, E. S., Valente, E., Polcari, A., & Teicher, M. H. (2008). Preliminary evidence for sensitive periods in the effect of childhood sexual abuse on regional brain development. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *20*(3), 292–301. https://doi.org/10.1176/jnp.2008.20.3.292

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: Diagnostic and statistical manual of mental disorders* (5th ed.). American Psychiatric Association.

Baldwin, J. R., Caspi, A., & Meehan, A. J. (2021). Population vs individual prediction of poor health from results of adverse childhood experiences screening. *JAMA Pediatrics*, *175*(4), 385–393.

Baldwin, J. R., Reuben, A., Newbury, J. B., & Danese, A. (2019). Agreement between prospective and retrospective measures of childhood maltreatment: A systematic review and meta-analysis. *JAMA Psychiatry*, *76*(6), 584–593. https://doi.org/10.1001/jamapsychiatry.2019.0097

Baram, T. Z., Davis, E. P., Obenaus, A., Sandman, C. A., Small, S. L., Solodkin, A., & Stern, H. (2012). Fragmentation and unpredictability of early-life experience in mental disorders. *American Journal of Psychiatry*, *169*(9), 907–915. https://doi.org/10.1176/appi.ajp.2012.11091347

Berger, M., & Sarnyai, Z. (2015). "More than skin deep": Stress neurobiology and mental health consequences of racial discrimination. *Stress (Amsterdam, Netherlands)*, *18*(1), 1–10. https://doi.org/10.3109/10253890.2014.989204

Bernstein, D. P., Fink, L., Handelsman, L., Foote, J., Lovejoy, M., Wenzel, K., Sapareto, E., & Ruggiero, J. (1994). Initial reliability and validity of a new retrospective measure of child abuse and neglect. *American Journal of Psychiatry*, *151*, 1132–1136.

Bernstein, D. P., Ahluvalia, T., Pogge, D., & Handelsman, L. (1997). Validity of the childhood trauma questionnaire in an adolescent psychiatric population. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(3), 340–348. https://doi.org/10.1097/00004583-199703000-00012

Bernstein, R., & Freyd, J. (2014). Trauma at home: How betrayal trauma and attachment theories understand the human response to abuse by an attachment figure. *Attachment*, *8*(1), 18–41.

Boeke, E. A., Moscarello, J. M., LeDoux, J. E., Phelps, E. A., & Hartley, C. A. (2017). Active avoidance: Neural mechanisms and attenuation of Pavlovian conditioned responding. *The Journal of Neuroscience*, *37*(18), 4808–4818. https://doi.org/10.1523/JNEUROSCI.3261-16.2017

Bowlby, J. (1958). The nature of the child's tie to his mother. *International Journal of Psychoanalysis*, *39*, 350–373.

Boyce, W. T. (2007). A biology of misfortune: Stress reactivity, social context, and the ontogeny of psychopathology in early life. In A. Masten (Ed.), Minnesota Symposium on Child Development: Vol. 34. *Multilevel dynamics in developmental psychopathology: Pathways to the future* (pp. 45–82). Minneapolis, MN: University of Minnesota.

Brunson, K. L. (2005). Mechanisms of late-onset cognitive decline after early-life stress. *Journal of Neuroscience*, *25*(41), 9328–9338. https://doi.org/10.1523/JNEUROSCI.2281-05.2005

Callaghan, B., Meyer, H., Opendak, M., Van Tieghem, M., Harmon, C., Li, A., Lee, F., Sullivan, R., & Tottenham, N. (2019). Using a developmental ecology framework to align fear neurobiology across species. *Annual Review of Clinical Psychology*, *15*, 345–369. https://doi.org/10.1146/annurev-clinpsy-050718-095727

Cameron, J. L. (2001). Critical periods for social attachment: Deprivation and neural systems in rhesus monkeys. *Society for Research in Child Development*, *2*, 054.

Casey, B. J., Heller, A. S., Gee, D. G., & Cohen, A. O. (2019). Development of the emotional brain. *Neuroscience Letters*, *693*, 29–34. https://doi.org/10.1016/j.neulet.2017.11.055

Cicchetti, D., & Toth, S. L. (1995). A developmental psychopathology perspective on child abuse and neglect. *Journal of the American Academy of Child & Adolescent Psychiatry*, *34*(5), 541–565. https://doi.org/10.1097/00004583-199505000-00008

Clark, U. S., Miller, E. R., & Hegde, R. R. (2018). Experiences of discrimination are associated with greater resting amygdala activity and functional connectivity. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *3*(4), 367–378. https://doi.org/10.1016/j.bpsc.2017.11.011

Cohodes, E. M., Kitt, E. R., Baskin-Sommers, A., & Gee, D. G. (2021). Influences of early-life stress on frontolimbic circuitry: Harnessing a dimensional approach to elucidate the effects of heterogeneity in stress exposure. *Developmental Psychobiology*, *63*(2), 153–172. https://doi.org/10.1002/dev.21969

Colich, N. L., Rosen, M. L., Williams, E. S., & McLaughlin, K. A. (2020). Biological aging in childhood and adolescence following experiences of threat and deprivation: A systematic review and meta-analysis. *Psychological Bulletin*, *146*(9), 721–764. https://doi.org/10.1037/bul0000270

Collins, K. A., Mendelsohn, A., Cain, C. K., & Schiller, D. (2014). Taking action in the face of threat: Neural synchronization predicts adaptive coping. *Journal of Neuroscience*, *34*(44), 14733–14738. https://doi.org/10.1523/JNEUROSCI.2152-14.2014

Currie, C. L., Copeland, J. L., & Metz, G. A. (2019). Childhood racial discrimination and adult allostatic load: The role of Indigenous cultural continuity in allostatic resiliency. *Social Science & Medicine*, *241*, 112564. https://doi.org/10.1016/j.socscimed.2019.112564

Danese, A., & Widom, C. S. (2020). Objective and subjective experiences of child maltreatment and their relationships with psychopathology. *Nature Human Behaviour*, *4*, 811–818. https://doi.org/10.1038/s41562-020-0880-3

Danese, A., & Widom, C. S. (2021). The subjective experience of childhood maltreatment in psychopathology. *JAMA Psychiatry*, *78*(12), 1307–1308. https://doi.org/10.1001/jamapsychiatry.2021.2874

Dannlowski, U., Stuhrmann, A., Beutelmann, V., Zwanzger, P., Lenzen, T., Grotegerd, D., Domschke, K., Hohoff, C., Ohrmann, P., & Bauer, J. (2012). Limbic scars: Long-term consequences of childhood maltreatment revealed by functional and structural magnetic resonance imaging. *Biological Psychiatry*, *71*(4), 286–293.

De Bellis, M. D., Keshavan, M. S., Clark, D. B., Casey, B. J., Giedd, J. N., Boring, A. M., Frustaci, K., & Ryan, N. D. (1999). Developmental traumatology part II: Brain development. *Biological Psychiatry*, *45*, 1271–1284.

De Bellis, M. D., & Kuchibhatla, M. (2006). Cerebellar volumes in pediatric maltreatment-related posttraumatic stress disorder. *Biological Psychiatry*, *60*(7), 697–703.

Delker, B. C., Smith, C. P., Rosenthal, M. N., Bernstein, R., & Freyd, J. J. (2018). When home is where the harm is: Family betrayal and posttraumatic outcomes in young adulthood. *Journal of Aggression, Maltreatment &*

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Trauma, *27*(7), 720–743. https://doi.org/10.1080/10926771.2017.1382639

Doom, J. R., & Cicchetti, D. (2020). *The developmental psychopathology of stress exposure in childhood*. The Oxford Handbook of Stress and Mental Health. https://doi.org/10.1093/oxfordhb/9780190681777.013.12

Edwards, V. J., Freyd, J. J., Dube, S. R., Anda, R. F., & Felitti, V. J. (2012). Health outcomes by closeness of sexual abuse perpetrator: A test of betrayal trauma theory. *Journal of Aggression, Maltreatment & Trauma*, *21*(2), 133–148.

Eiland, L., & Romeo, R. D. (2013). Stress and the developing adolescent brain. *Neuroscience*, *249*, 162–171. https://doi.org/10.1016/j.neuroscience.2012.10.048

Elias, A., & Paradies, Y. (2016). Estimating the mental health costs of racial discrimination. *BMC Public Health*, *16*(1), 1205. https://doi.org/10.1186/s12889-016-3868-1

Elliott, D. M., & Briere, J. (1992). Sexual abuse trauma among professional women: Validating the Trauma Symptom Checklist-40 (TSC-40). *Child Abuse & Neglect*, *16*(3), 391–398.

Ellis, B. J., Figueredo, A. J., Brumbach, B. H., & Schlomer, G. L. (2009). Fundamental dimensions of environmental risk. *Human Nature*, *20*(2), 204–268.

Ellis, B. J., Sheridan, M. A., Belsky, J., & McLaughlin, K. A. (2022). Why and how does early adversity influence development? Toward an integrated model of dimensions of environmental experience. *Development and Psychopathology*, *34*(2), 447–471.

Espejo, E. P., Ferriter, C. T., Hazel, N. A., Keenan-Miller, D., Hoffman, L. R., & Hammen, C. (2011). Predictors of subjective ratings of stressor severity: The effects of current mood and neuroticism. *Stress and Health*, *27*(1), 23–33. https://doi.org/10.1002/smi.1315

Estrada, S., Gee, D. G., Bozic, I., Cinguina, M., Joormann, J., & Baskin-Sommers, A. (2021). Individual and environmental correlates of childhood maltreatment and exposure to community violence: Utilizing a latent profile and a multilevel meta-analytic approach. *Psychological Medicine*, *2*, 1–17.

Fan, Y., Herrera-Melendez, A. L., Pestke, K., Feeser, M., Aust, S., Otte, C., Pruessner, J. C., Böker, H., Bajbouj, M., & Grimm, S. (2014). Early life stress modulates amygdala-prefrontal functional connectivity: Implications for oxytocin effects: Early life stress and amygdala functional connectivity. *Human Brain Mapping*, *35*(10), 5328–5339. https://doi.org/10.1002/hbm.22553

Fani, N., Carter, S. E., Harnett, N. G., Ressler, K. J., & Bradley, B. (2021). Association of racial discrimination with neural response to threat in black women in the US exposed to trauma. *JAMA Psychiatry*, *78*(9), 1005–1012. https://doi.org/10.1001/jamapsychiatry.2021.1480

Faul, L., Stjepanović, D., Stivers, J. M., Stewart, G. W., Graner, J. L., Morey, R. A., & LaBar, K. S. (2020). Proximal threats promote enhanced acquisition and persistence of reactive fear-learning circuits. *Proceedings of the National Academy of Sciences of the United States of America*, *117*(28), 16678–16689. https://doi.org/10.1073/pnas.2004258117

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine*, *14*(4), 245–258. https://doi.org/10.1016/S0749-3797(98)00017-8

Fox, S. E., Levitt, P., & Nelson, III, C. A. (2010). How the timing and quality of early experiences influence the development of brain architecture. *Child Development*, *81*(1), 28–40.

Frans, Ö., Ahs, J., Bihre, E., & Ahs, F. (2018). Distance to threat and risk of acute and posttraumatic stress disorder following bank robbery: A longitudinal study. *Psychiatry Research*, *267*, 461–466.

Freyd, J. J. (1994). Betrayal trauma: Traumatic amnesia as an adaptive response to childhood abuse. *Ethics & Behavior*, *4*(4), 307–329. https://doi.org/10.1207/s15327019eb0404_1

Freyd, J. J. (1997). II. Violations of power, adaptive blindness and betrayal trauma theory. *Feminism & Psychology*, *7*(1), 22–32.

Friedman, M. J. (2013). Finalizing PTSD in DSM-5: Getting here from there and where to go next. *Journal of Adversity*, *26*(5), 548–556. https://doi.org/10.1002/jts.21840

Gabard-Durnam, L. J., Flannery, J., Goff, B., Gee, D. G., Humphreys, K. L., Telzer, E., Hare, T., & Tottenham, N. (2014). The development of human amygdala functional connectivity at rest from 4 to 23 years: A cross-sectional study. *Neuroimage*, *95*, 193–207. https://doi.org/10.1016/j.neuroimage.2014.03.038

Gabbay, V., Oatis, M. D., Silva, R. R., & Hirsch, G. (2004). Posttraumatic stress disorder in children and adolescents: Epidemiological aspects of PTSD in children and adolescents. https://www.uptodate.com/contents/posttraumatic-stress-disorder-in-children-and-adolescents-epidemiology-pathogenesis-clinical-manifestations-course-assessment-and-diagnosis#disclaimerContent

Ganzel, B., Casey, B. J., Glover, G., Voss, H. U., & Temple, E. (2007). The aftermath of 9/11: Effect of intensity and recency of trauma on outcome. *Emotion (Washington, D.C.)*, *7*(2), 227–238. https://doi.org/10.1037/1528-3542.7.2.227

Gee, D. G., Bath, K. G., Johnson, C. M., Meyer, H. C., Murty, V. P., Bos, W. v. d., & Hartley, C. A. (2018). Neurocognitive development of motivated behavior: Dynamic changes across childhood and adolescence. *Journal of Neuroscience*, *38*(44), 9433–9445. https://doi.org/10.1523/JNEUROSCI.1674-18.2018

Gee, D. G., & Casey, B. J. (2015). The impact of developmental timing for stress and recovery. *Neurobiology of Stress*, *1*, 184–194. https://doi.org/10.1016/j.ynstr.2015.02.001

Gee, D. G., & Cohodes, E. M. (2021). Influences of caregiving on development: A sensitive period for biological embedding of predictability and safety cues. *Current Directions in Psychological Science*, *30*(5), 376–383.

Gee, D. G., Gabard-Durnam, L., Telzer, E. H., Humphreys, K. L., Goff, B., Shapiro, M., Flannery, J., Lumian, D. S., Fareri, D. S., Caldera, C., & Tottenham, N. (2014). Maternal buffering of human amygdala-prefrontal circuitry during childhood but not during adolescence. *Psychological Science*, *25*(11), 2067–2078. https://doi.org/10.1177/0956797614550878

Gee, D. G., Gabard-Durnam, L. J., Flannery, J., Goff, B., Humphreys, K. L., Telzer, E. H., Hare, T. A., Bookheimer, S. Y., & Tottenham, N. (2013). Early developmental emergence of human amygdala-prefrontal connectivity after maternal deprivation. *Proceedings of the National Academy of Sciences*, *110*(39), 15638–15643. https://doi.org/10.1073/pnas.1307893110

Glynn, L. M., & Baram, T. Z. (2019). The influence of unpredictable, fragmented parental signals on the developing brain. *Frontiers in Neuroendocrinology*, *53*, 100736. https://doi.org/10.1016/j.yfrne.2019.01.002

Goldsmith, R. E., Martin, C. G., & Smith, C. P. (2014). Systemic trauma. *Journal of Trauma & Dissociation*, *15*(2), 117–132. https://doi.org/10.1080/15299732.2014.871666

Gomez, S. H., Tse, J., Wang, Y., Turner, B., Millner, A. J., Nock, M. K., & Dunn, E. C. (2017). Are there sensitive periods when child maltreatment substantially elevates suicide risk? Results from a nationally representative sample of adolescents. *Depression and Anxiety*, *34*(8), 734–741. https://doi.org/10.1002/da.22650

Goodman, L. A., Corcoran, C., Turner, K., Yuan, N., & Green, B. L. (1998). Assessing traumatic event exposure: General issues and preliminary findings for the Stressful Life Events Screening Questionnaire. *Journal of Traumatic Stress: Official Publication of The International Society for Traumatic Stress Studies*, *11*(3), 521–542.

Graham, A. M., Pfeifer, J. H., Fisher, P. A., Carpenter, S., & Fair, D. A. (2015). Early life stress is associated with default system integrity and emotionality during infancy. *Journal of Child Psychology and Psychiatry*, *56*(11), 1212–1222.

Hambrick, E. P., Brawner, T. W., Perry, B. D., Brandt, K., Hofmeister, C., & Collins, J. O. (2019). Beyond the ACE score: Examining relationships between timing of developmental adversity, relational health and devel-

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

opmental outcomes in children. *Archives of Psychiatric Nursing*, *33*(3), 238–247. https://doi.org/10.1016/j.apnu.2018.11.001

Hammen, C., Marks, T., Mayol, A., & DeMayo, R. (1985). Depressive self-schemas, life stress, and vulnerability to depression. *Journal of Abnormal Psychology*, *94*(3), 308.

Hare, T. A., Tottenham, N., Galvan, A., Voss, H. U., Glover, G. H., & Casey, B. J. (2008). Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. *Biological Psychiatry*, *63*(10), 927–934.

Hartley, C. A., Gorun, A., Reddan, M. C., Ramirez, F., & Phelps, E. A. (2014). Stressor controllability modulates fear extinction in humans. *Neurobiology of Learning and Memory*, *113*, 149–156. https://doi.org/10.1016/j.nlm.2013.12.003

Herzberg, M. P., McKenzie, K. J., Hodel, A. S., Hunt, R. H., Mueller, B. A., Gunnar, M. R., & Thomas, K. M. (2021). Accelerated maturation in functional connectivity following early life stress: Circuit specific or broadly distributed? *Developmental Cognitive Neuroscience*, *48*, 100922. https://doi.org/10.1016/j.dcn.2021.100922

Herzog, J. I., Thome, J., Demirakca, T., Koppe, G., Ende, G., Lis, S., Rausch, S., Priebe, K., Müller-Engelmann, M., & Steil, R. (2020). Influence of severity of type and timing of retrospectively reported childhood maltreatment on female amygdala and hippocampal volume. *Scientific Reports*, *10*(1), 1–10.

Hostinar, C. E., Johnson, A. E., & Gunnar, M. R. (2015). Parent support is less effective in buffering cortisol stress reactivity for adolescents compared to children. *Developmental Science*, *18*(2), 281–297.

Hughes, K., Bellis, M. A., Hardcastle, K. A., Sethi, D., Butchart, A., Mikton, C., Jones, L., & Dunne, M. P. (2017). The effect of multiple adverse childhood experiences on health: A systematic review and meta-analysis. *The Lancet Public Health*, *2*(8), e356–e366. https://doi.org/10.1016/S2468-2667(17)30118-4

Kessler, R. C., Aguilar-Gaxiola, S., Alonso, J., Benjet, C., Bromet, E. J., Cardoso, G., Degenhardt, L., Girolamo, G., Dinolova, R. V., Ferry, F., Florescu, S., Gureje, O., Haro, J. M., Huang, Y., Karam, E. G., Kawakami, N., Lee, S., Lepine, J.-P., Levinson, D., … Koenen, K. C. (2017). Trauma and PTSD in the WHO world mental health surveys. *European Journal of Psychotraumatology*, *8*(sup5), 1353383. https://doi.org/10.1080/20008198.2017.1353383

Kubany, E. S., Leisen, M. B., Kaplan, A. S., Watson, S. B., Haynes, S. N., Owens, J. A., & Burns, K. (2000). Development and preliminary validation of a brief broad-spectrum measure of trauma exposure: the Traumatic Life Events Questionnaire. *Psychological Assessment*, *12*(2), 210.

Khan, A., McCormack, H. C., Bolger, E. A., McGreenery, C. E., Vitaliano, G., Polcari, A., & Teicher, M. H. (2015). Childhood maltreatment, depression, and suicidal ideation: Critical importance of parental and peer emotional abuse during developmental sensitive periods in males and females. *Frontiers in Psychiatry*, *6*, 42. https://doi.org/10.3389/fpsyt.2015.00042

Luby, J. L., Tillman, R., & Barch, D. M. (2019). Association of timing of adverse childhood experiences and caregiver support with regionally specific brain development in adolescents. *JAMA Network Open*, *2*(9), e1911426–e1911426.

Maier, S. F., & Watkins, L. R. (2010). Role of the medial prefrontal cortex in coping and resilience. *Brain Research*, *1355*, 52–60. https://doi.org/10.1016/j.brainres.2010.08.039

Malter Cohen, M., Jing, D., Yang, R. R., Tottenham, N., Lee, F. S., & Casey, B. J. (2013). Early-life stress has persistent effects on amygdala function and development in mice and humans. *Proceedings of the National Academy of Sciences*, *110*(45), 18274–18278. https://doi.org/10.1073/pnas.1310163110

Manly, J. T., Kim, J. E., Rogosch, F. A., & Cicchetti, D. (2001). Dimensions of child maltreatment and children's adjustment: Contributions of developmental timing and subtype. *Development and Psychopathology*, *13*(4), 759–782.

Masten, A. S., & Cicchetti, D. (2010). Developmental cascades. *Development and Psychopathology*, *22*(3), 491–495.

Matheson, K., Foster, M. D., Bombay, A., McQuaid, R. J., & Anisman, H. (2019). Traumatic experiences, perceived discrimination, and psychological distress among members of various socially marginalized groups. *Frontiers in Psychology*, *10*, 416. https://doi.org/10.3389/fpsyg.2019.00416

Matheson, K., McQuaid, R. J., & Anisman, H. (2016). Group identity, discrimination, and well-being: Confluence of psychosocial and neurobiological factors. *Current Opinion in Psychology*, *11*, 35–39. https://doi.org/10.1016/j.copsyc.2016.05.005

May, C. L., & Wisco, B. E. (2016). Defining trauma: How level of exposure and proximity affect risk for posttraumatic stress disorder. *Psychological Trauma: Theory, Research, Practice, and Policy*, *8*(2), 233.

McCoy, D. C. (2013). Early violence exposure and self-regulatory development: A bioecological systems perspective. *Human Development*, *56*(4), 254–273. https://doi.org/10.1159/000353217

McCrory, E. J., De Brito, S. A., Kelly, P. A., Bird, G., Sebastian, C. L., Mechelli, A., Samuel, S., & Viding, E. (2013). Amygdala activation in maltreated children during pre-attentive emotional processing. *British Journal of Psychiatry*, *202*(04), 269–276. https://doi.org/10.1192/bjp.bp.112.116624

McLaughlin, K. A., & Sheridan, M. A. (2016). Beyond cumulative risk: A dimensional approach to childhood adversity. *Current Directions in Psychological Science*, *25*(4), 239–245. https://doi.org/10.1177/0963721416655883

McLaughlin, K. A., Weissman, D., & Bitrán, D. (2019). Childhood adversity and neural development: A systematic review. *Annual Review of Developmental Psychology*, *1*, 277–312.

McLaughlin, K. A., Sheridan, M. A., Humphreys, K. L., Belsky, J., & Ellis, B. J. (2021). The value of dimensional models of early experience: Thinking clearly about concepts and categories. *Perspectives on Psychological Science*, *16*(6), 1463–1472. https://doi.org/10.1177/1745691621992346

McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity and neural development: Deprivation and threat as distinct dimensions of early experience. *Neuroscience and Biobehavioral Reviews*, *47*, 578–591. https://doi.org/10.1016/j.neubiorev.2014.10.012

Mehta, M. A., Golembo, N. I., Nosarti, C., Colvert, E., Mota, A., Williams, S. C. R., Rutter, M., & Sonuga-Barke, E. J. S. (2009). Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: The English and Romanian adoptees study pilot. *Journal of Child Psychology and Psychiatry*, *50*(8), 943–951. https://doi.org/10.1111/j.1469-7610.2009.02084.x

Miller, A. B., Sheridan, M. A., Hanson, J. L., McLaughlin, K. A., Bates, J. E., Lansford, J. E., Pettit, G. S., & Dodge, K. A. (2018). Dimensions of deprivation and threat, psychopathology, and potential mediators: A multi-year longitudinal analysis. *Journal of Abnormal Psychology*, *127*(2), 160.

Nelson, C. A., Zeanah, C. H., Fox, N. A., Marshall, P. J., Smyke, A. T., & Guthrie, D. (2007). Cognitive recovery in socially deprived young children: The Bucharest Early Intervention Project. *Science*, *318*(5858), 1937–1940. https://doi.org/10.1126/science.1143921

Pai, A., Suris, A., & North, C. (2017). Posttraumatic stress disorder in the DSM-5: Controversy, change, and conceptual considerations. *Behavioral Sciences*, *7*(4), 7. https://doi.org/10.3390/bs7010007

Pechtel, P., Lyons-Ruth, K., Anderson, C. M., & Teicher, M. H. (2014). Sensitive periods of amygdala development: The role of maltreatment in preadolescence. *Neuroimage*, *97*, 236–244.

Peters, E., Riksen-Walraven, J. M., Cillessen, A. H., & de Weerth, C. (2011). Peer rejection and HPA activity in middle childhood: Friendship makes a difference. *Child Development*, *82*(6), 1906–1920.

Pollak, S. D., & Smith, K. E. (2021). Thinking clearly about biology and childhood adversity: Next steps for continued progress. *Perspectives on Psychological Science*, *16*, 1473–1477. https://doi.org/10.1177/17456916211031539

Priest, N., Paradies, Y., Trenerry, B., Truong, M., Karlsen, S., & Kelly, Y. (2013). A systematic review of studies examining the relationship between

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

reported racism and health and wellbeing for children and young people. *Social Science & Medicine*, *95*, 115–127.

Pynoos, R. S., & Steinberg, A. M. (2017). *The University of California, Los Angeles, Post-traumatic Stress Disorder Reaction Index (UCLA PTSD Index) for the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5)*. Los Angeles: University of California.

Russotti, J., Warmingham, J. M., Duprey, E. B., Handley, E. D., Manly, J. T., Rogosch, F. A., & Cicchetti, D. (2021). Child maltreatment and the development of psychopathology: The role of developmental timing and chronicity. *Child Abuse & Neglect*, *120*, 105215. https://doi.org/10.1016/j.chiabu.2021.105215

Sabatini, M. J., Ebert, P., Lewis, D. A., Levitt, P., Cameron, J. L., & Mirnics, K. (2007). Amygdala gene expression correlates of social behavior in monkeys experiencing maternal separation. *Journal of Neuroscience*, *27*(12), 3295–3304.

Schalinski, I., Teicher, M. H., Nischk, D., Hinderer, E., Müller, O., & Rockstroh, B. (2016). Type and timing of adverse childhood experiences differentially affect severity of PTSD, dissociative and depressive symptoms in adult inpatients. *BMC Psychiatry*, *16*(1), 1–15.

Sheridan, M. A., Fox, N. A., Zeanah, C. H., McLaughlin, K. A., & Nelson, C. A. (2012). Variation in neural development as a result of exposure to institutionalization early in childhood. *Proceedings of the National Academy of Sciences*, *109*(32), 12927–12932.

Sheridan, M. A., & McLaughlin, K. A. (2014). Dimensions of early experience and neural development: Deprivation and threat. *Trends in Cognitive Sciences*, *18*(11), 580–585. https://doi.org/10.1016/j.tics.2014.09.001

Shonkoff, J. P., Siegel, B. S., Dobbins, M. I., Earls, M. F., Garner, A. S., McGuinn, L., Pascoe, J., & Wood, D. L., The Committee on Psychosocial Aspects of Child and Family Health, Committee on Early Childhood Adoption, and Dependent Care, and Section on Developmental and Behavioral Pediatrics. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, *129*(1), e232–e246. https://doi.org/10.1542/peds.2011-2663

Smith, C. P., & Freyd, J. J. (2014). Institutional betrayal. *American Psychologist*, *69*(6), 575–587. https://doi.org/10.1037/a0037564

Smith, K. E., & Pollak, S. D. (2020). Early life stress and development: Potential mechanisms for adverse outcomes. *Journal of Neurodevelopmental Disorders*, *12*(1), 34. https://doi.org/10.1186/s11689-020-09337-y

Smith, K. E., & Pollak, S. D. (2021). Rethinking concepts and categories for understanding the neurodevelopmental effects of childhood adversity. *Perspectives on Psychological Science*, *16*(1), 67–93. https://doi.org/10.1177/1745691620920725

Smith, N., Lam, D., Bifulco, A., & Checkley, S. (2002). Childhood Experience of Care and Abuse Questionnaire (CECA.Q). Validation of a screening instrument for childhood adversity in clinical populations. *Social Psychiatry and Psychiatric Epidemiology*, *37*(12), 572–579. https://doi.org/10.1007/s00127-002-0589-9

Spilsbury, J. C., Fletcher, K. E., Creeden, R., & Friedman, S. (2008). Psychometric properties of the dimensions of stressful events rating scale. *Traumatology*, *14*(4), 116–130. https://doi.org/10.1177/1534765608321072

Steinberg, A. M., Brymer, M. J., Decker, K. B., & Pynoos, R. S. (2004). The University of California at Los Angeles Post-Traumatic Stress Disorder Reaction Index. *Current Psychiatry Reports*, *6*(2), 96–100. https://doi.org/10.1007/s11920-004-0048-2

Teicher, M. H., & Parigger, A. (2015). The 'Maltreatment and abuse chronology of exposure' (MACE) scale for the retrospective assessment of abuse and neglect during development. *PLoS One*, *10*(2), e0117423.

Teicher, M. H., Samson, J. A., Anderson, C. M., & Ohashi, K. (2016). The effects of childhood maltreatment on brain structure, function and connectivity. *Nature Reviews Neuroscience*, *17*(10), 652–666. https://doi.org/10.1038/nrn.2016.111

Tottenham, N. (2012). Human amygdala development in the absence of species-expected caregiving. *Developmental Psychobiology*, *54*(6), 598–611.

Tottenham, N., Hare, T. A., Millner, A., Gilhooly, T., Zevin, J. D., & Casey, B. J. (2011). Elevated amygdala response to faces following early deprivation. *Developmental Science*, *14*(2), 190–204.

Tottenham, N., & Sheridan, M. A. (2010). A review of adversity, the amygdala and the hippocampus: A consideration of developmental timing. *Frontiers in Human Neuroscience*, *3*, 68.

Tsuda, A., Ida, Y., Satoh, H., Tsujimaru, S., & Tanaka, M. (1989). Stressor predictability and rat brain noradrenaline metabolism. *Pharmacology Biochemistry and Behavior*, *32*(2), 569–572.

Tupler, L. A., & De Bellis, M. D. (2006). Segmented hippocampal volume in children and adolescents with posttraumatic stress disorder. *Biological Psychiatry*, *59*(6), 523–529.

VanTieghem, M. R., & Tottenham, N. (2017). Neurobiological programming of early life stress: Functional development of amygdala-prefrontal circuitry and vulnerability for stress-related psychopathology. In E. Vermetten, D. G. Baker, & V. B. Risbrough (Eds.), *Behavioral neurobiology of PTSD* (vol. 38, pp. 117–136). Springer International Publishing. https://doi.org/10.1007/7854_2016_42

Veer, I. M., Oei, N. Y., van Buchem, M. A., Spinhoven, P., Elzinga, B. M., & Rombouts, S. A. (2015). Evidence for smaller right amygdala volumes in posttraumatic stress disorder following childhood trauma. *Psychiatry Research: Neuroimaging*, *233*(3), 436–442.

Weathers, F. W., Blake, D. D., Schnurr, P. P., Kaloupek, D. G., Marx, B. P., & Keane, T. M. (2013). The life events checklist for DSM-5 (LEC-5). Instrument available from the National Center for PTSD. Retrieved January 31, 2023.

Wolfe, J., Kimerling, R., Brown, P. J., Chrestman, K. R., & Levin, K. (1996). Psychometric review of the life stressor checklist-revised. *Measurement of Stress, Trauma, and Adaptation*, 198–201.

Wu, J., Geng, X., Shao, R., Wong, N. M., Tao, J., Chen, L., Chan, C. C., & Lee, T. M. (2018). Neurodevelopmental changes in the relationship between stress perception and prefrontal-amygdala functional circuitry. *NeuroImage: Clinical*, *20*, 267–274.

Wu, M., Kujawa, A., Lu, L. H., Fitzgerald, D. A., Klumpp, H., Fitzgerald, K. D., Monk, C. S., & Phan, K. L. (2016). Age-related changes in amygdala–frontal connectivity during emotional face processing from childhood into young adulthood. *Human Brain Mapping*, *37*(5), 1684–1695. https://doi.org/10.1002/hbm.23129

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Cohodes, E. M., McCauley, S., Pierre, J. C., Hodges, H. R., Haberman, J. T., Santiuste, I., Rogers, M. K., Wang, J., Mandell, J. D., & Gee, D. G. (2023). Development and validation of the Dimensional Inventory of Stress and Trauma Across the Lifespan (DISTAL): A novel assessment tool to facilitate the dimensional study of psychobiological sequelae of exposure to adversity. *Developmental Psychobiology*, *65*, e22372. https://doi.org/10.1002/dev.22372

10982302, 2023, 4, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/dev.22372 by Yale University, Wiley Online Library on [15/05/2023]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License