### **Special Issue Article**

### **Resilience in Development: Pathways to Multisystem Integration**

### Characterizing experiential elements of early-life stress to inform resilience: Buffering effects of controllability and predictability and the importance of their timing

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### Abstract

Key theoretical frameworks have proposed that examining the impact of exposure to specific dimensions of stress at specific developmental periods is likely to yield important insight into processes of risk and resilience. Utilizing a sample of N = 549 young adults who provided a detailed retrospective history of their lifetime exposure to numerous dimensions of traumatic stress and ratings of their current trauma-related symptomatology via completion of an online survey, here we test whether an individual's perception of their lifetime stress as either controllable or predictable buffered the impact of exposure on trauma-related symptomatology assessed in adulthood. Further, we tested whether this moderation effect differed when evaluated in the context of early childhood, middle childhood, adolescence, and young adulthood stress. Consistent with hypotheses, results highlight both stressor controllability and stressor predictability as buffering the impact of traumatic stress exposure on trauma-related symptomatology and suggest that the potency of this buffering effect varies across unique developmental periods. Leveraging dimensional ratings of lifetime stress exposure to probe heterogeneity in outcomes following stress – and, critically, considering interactions between dimensions of exposure and the developmental period when stress occurred – is likely to yield increased understanding of risk and resilience following traumatic stress.

Keywords: stress; adversity; traumatic stress; dimensional; controllability; predictability; trauma-related symptomatology

(Received 15 March 2023; revised 16 June 2023; accepted 18 June 2023; first published online 27 July 2023)

### Introduction

Though exposure to stress can have profound and lasting effects on the developing brain and behavior (Boyce, 2007; Shonkoff et al., 2012; VanTieghem & Tottenham, 2017), the effects of stress across development are neither ubiquitous nor uniform (Doom & Cicchetti, 2020; Gabbay et al., 2004). In an effort to further understand this multifinality, key theoretical frameworks have proposed that, rather than treating stress exposure as a unitary construct, examining correlates of specific dimensions of exposure – including experiential, environmental, and timing attributes – can yield a richer understanding of processes of risk and resilience (Belsky et al., 2012; Cohodes et al., 2021; Ellis et al., 2009, 2022; Gee & Casey, 2015; Kuhlman et al., 2017; Manly et al., 2001; Sheridan & McLaughlin, 2014). The controllability and predictability of a stressor are two such

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**Cite this article:** Cohodes, E. M., Sisk, L. M., Keding, T. J., Mandell, J. D., Notti, M. E., & Gee, D. G. (2023). Characterizing experiential elements of early-life stress to inform resilience: Buffering effects of controllability and predictability and the importance of their timing. *Development and Psychopathology* **35**: 2288–2301, https://doi.org/10.1017/S0954579423000822

features that are theorized to moderate the impact of exposure to stress on later functioning (Cohodes et al., 2021).

# Controllability as a proposed moderator of the impact of stress on later functioning

Decades of animal research document both short- and long-term resilience-promoting effects of exposure to stress that is controllable (Amat et al., 2010; Maier & Watkins, 2010; Seligman & Maier, 1967). Key findings from this rich animal literature highlight that exposure to controllable stress has the potential to buffer an individual from the potentially deleterious effects of exposure to subsequent stressors via persistent modulation of the neurocircuitry governing stress reactivity and regulation (e.g., Amat et al., 2010; Bravo-Rivera et al., 2015; Ramirez et al., 2015). Specifically, exposure to prior controllable stress may yield frontostriatal plasticity that facilitates prefrontal regulation of the amygdala even when an individual does not have control over a subsequent stressor (Amat et al., 2008, 2010), which over time may temper the negative impacts of stress exposure on wellbeing.

In humans, initial studies suggest that the ability to exert control over a stressor – in other words, an individual's ability to alter the intensity, duration, onset, or termination of a stressor – may

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engage frontolimbic circuitry to promote decreased stress reactivity and increased active coping behaviors when an individual is presented with subsequent stressors (Boeke et al., 2017; Collins et al., 2014). Thus, in contrast to the notion of stress as consistently harmful, exposure to controllable stress may actually render an individual more prepared to cope with subsequent stressors by promoting long-term resilience and facilitating adaptive coping and plasticity in behavioral responses to stress (Amat et al., 2010). Indeed, Moscarello and Hartley (2017) have theorized that an individual's history of interactions with the environment, and their resultant estimation of their ability to exert control over that environment, is likely to drive selection of behavioral strategies that are most likely to be adaptive in that context. For example, if an individual has previously been able to exert control over a particular environment or stressor, they may assume control during subsequent exposure to novel or uncertain environments and may therefore adopt adaptive coping strategies that are likely to promote resilience in the face of stress exposure. Recent empirical work supports this theory - exposure to controllable stress, relative to uncontrollable stress, was found to be associated with more exploratory behavior and greater reliance on an active approach to learning about a novel environment, underscoring greater exploration and adaptive estimation of environmental control as one such mechanism by which exposure to previous controllable stress may promote long-term resilience (Ligneul et al., 2020).

## Predictability as a proposed moderator of the impact of stress on later functioning

In addition, extensive animal studies and an emerging human developmental literature have highlighted that the degree to which a stressor is predictable – that is, the degree to which it occurs in a way that is both expected and reliable – may be a primary determinant of its impact on psychobiological outcomes (see Kuhlman et al., 2017 for a review). We note that, in contrast to the stressor controllability literature reviewed above, to date, the literature examining the role of exposure to predictability in the context of stress has not focused on examining whether the predictability of a stressor moderates the impact of that stressor on later functioning; rather empirical work has identified exposure to unpredictable caregiving and environmental inputs as a unique source of stress exposure in and of itself (Ellis et al., 2009; Glynn et al., 2018; Molet et al., 2016; Poggi Davis et al., 2017; Risbrough et al., 2018).

Animal studies testing the impact of exposure to predictability in the context of early-life stress have primarily employed models that manipulate the predictability of caregiver cues. Findings from this line of work suggest that exposure to fragmented care and unpredictable maternal cues among rodents is associated with cognitive and affective dysfunction among offspring (Baram et al., 2012; Brunson, 2005), with evidence that these changes may be driven by dysregulation of corticolimbic circuitry (Malter Cohen et al., 2013; Tsuda et al., 1989). Initial translation of animal work on predictability into human developmental samples also provides evidence that a higher degree of unpredictability in the context of caregiving relationships is associated with poorer cognitive outcomes for offspring. For example, higher rates of unpredictability of maternal cues in the first year of life (quantified by frequent switching between modalities of interaction and unpredictability of sensory cues) were found to be associated with poorer infant cognitive outcomes and language skills (Davis et al., 2019; Davis et al., 2017). One theoretical pathway by which predictability of caregiving cues may influence cognitive, social, and emotional development is via the association between appropriate and predictable cueing and the promotion of secure attachment (see Gee & Cohodes, 2021 for a review; Sroufe, 2005). Though the neurodevelopmental underpinnings of the impact of caregiver predictability on offspring functioning remain less clearly understood in human samples, evidence from rodent paradigms that manipulate predictability of maternal care has highlighted the impact of predictable care on corticolimbic circuitry central to emotion regulation (Guadagno et al., 2018; Malter Cohen et al., 2013). Though preliminary, these findings suggest that exposure to more predictable caregiving environments may scaffold normative development of corticolimbic circuitry to promote regulation which may, in turn, promote resilience following adversity.

In addition to extensive work documenting correlates of exposure to unpredictability in the context of caregiving, there is growing interest in examining the role of predictability more broadly in the context of generalized experience across development. For example, numerous recent studies suggest that exposure to unpredictability across social, emotional, and physical domains in both childhood and adolescence is associated with increased symptomatology reported in both adolescence and adulthood (Davis et al., 2019; Doom et al., 2016; McGinnis et al., 2022; Spadoni et al., 2022; Szepsenwol et al., 2022). These studies highlight that the degree to which life experiences are predictable across development - both in the context of caregiving unpredictability and more broadly across life experiences - is likely to have important implications for psychological functioning. These findings also suggest that assessment of predictability in the context of exposure to adversity across the life course is likely to facilitate new understanding of the ways in which predictability, as a characteristic of stress exposure, may promote resilient functioning.

### Developmentally-specific effects of stress exposure characterized by controllability and predictability

Building upon models of stress exposure that have highlighted theoretical effects of exposure to features of stress in isolation (Cicchetti & Toth, 1995; McCoy, 2013; Pynoos et al., 1999; Sheridan & McLaughlin, 2014), recent frameworks have proposed that the developmental timing of exposure to stress is likely a crucial backdrop for the estimation of effects of exposure to all other specific dimensions of stress (Cohodes et al., 2021; Gee & Casey, 2015; Kuhlman et al., 2017; Lupien et al., 2009; Tottenham & Sheridan, 2010). Given dynamic changes in the biological state of the developing brain (Casey et al., 2019; Gee et al., 2018), the degree to which an individual's exposure to a particular feature of stress has the potential to exacerbate or attenuate the impact of stress on functioning is likely to be driven by the developmental time point during which this exposure occurs (Cohodes et al., 2021).

Accordingly, though the ability to exert control over a stressor may buffer the impact of stress exposure across the life course, cross-species empirical evidence suggests that exposure to controllable stress may be particularly impactful during adolescence (Gamble, 1994; Kubala et al., 2012; Raab et al., 2022). Adolescence is characterized by increased plasticity (Larsen & Luna, 2018; Sisk & Gee, 2022; Sydnor et al., 2021), dramatic change in frontostriatal-amygdala circuitry, and, relatedly, motivated behavior (Casey et al., 2019; Herting et al., 2018; Luna et al., 2015; Silvers et al., 2017; van Duijvenvoorde et al., 2016). Given the centrality of this circuitry in the neural correlates of exposure to controllable stress observed in human imaging studies to date (Bhanji & Delgado, 2014; Boeke et al., 2017; Collins et al., 2014; Ligneul et al., 2020), the state of frontostriatal-amygdala circuitry in adolescence may be particularly sensitive to adaptive effects of being able to exert control over a stressor (Casey et al., 2019; Gee et al., 2018; Heller et al., 2016), which may promote active coping to a relatively greater degree when experienced during this developmental period

In addition, though exposure to unpredictability appears to have pronounced effects on functioning across development, early childhood has been proposed as a sensitive period for the biological embedding of predictable caregiving and environmental cues (Gee & Cohodes, 2021) given the centrality of these cues to primary developmental tasks of infancy and toddlerhood. It is therefore possible that early childhood may be a time when exposure to predictability in the context of traumatic stress exposure buffers the impact of that stress on later functioning to a greater degree relative to other developmental time periods. It is also possible that the buffering effect of predictability may be salient throughout all of childhood – rather than just in early childhood – particularly when it is considered as a potential feature of all traumatic stress exposures rather than solely in the context of caregiving-related predictability.

### The present study

There is considerable evidence that the degree to which a stressor is controllable or predictable may moderate the impact of that stressor on later functioning. Despite a rich cross-species literature that has examined the neurobehavioral correlates of stressor controllability and, separately, the impact of exposure to unpredictability as a standalone source of adversity, to our knowledge no empirical work to date has examined the role of either controllability or predictability of stress exposure as potential moderators of the impact of exposure to traumatic stress across the life course on mental health assessed in adulthood. Further, despite evidence that there may be age-related differences in the impact of exposure to controllable and predictable stress on coping and later functioning, to our knowledge, no empirical studies to date have examined age-related differences in the effects of exposure to controllable and predictable stress across human development. Finally, the extant literature has examined impacts of broad exposure to stressor controllability and predictability collapsing across both objective (e.g., induced) and subjective (e.g., perceived) constructs; to date, empirical investigation has not aimed to isolate the function of individuals' perception of these dimensions of experience in the context of considering the developmental timing of stress exposure.

The present study aimed to fill this gap in the literature by examining the potential of an individual's perception of a stressor – as controllable or predictable – to buffer the impact of overall exposure to traumatic stress across the life course on traumarelated symptomatology in adulthood. In addition, we examined whether the buffering effect of exposure to either controllable or predictable stress depends on whether the stress was experienced in early childhood, middle childhood, adolescence, and adulthood.

Consistent with recent theoretical models, we hypothesized that the degree to which an individual's lifetime exposure to traumatic stress is characterized by predictability or controllability would buffer the association between lifetime exposure to traumatic stress and current trauma-related symptomatology. We also hypothesized that this moderation effect would vary across development, such that the buffering effect of perceiving stressors to be controllable would be most salient during adolescence and the buffering effect of perceiving stressors to be predictable would be most salient in early childhood.

### Method

### Participants

A sample of workers with approval ratings over 90% was recruited via Amazon TurkPrime (Litman et al., 2017). The CloudResearch platform was used to manage participant recruitment. N = 856unique participants were recruited in response to an Amazon TurkPrime posting advertising a study titled "Life Experiences Survey" for individuals between the ages of 18 and 30 years of age living in the United States. Survey responses were considered eligible for inclusion in the present study if participants provided complete data for dimensional queries related to whether their exposures were characterized by controllability or predictability (i.e., participants had no missing data for these fields) on the electronic version of the Dimensional Inventory of Stress and Trauma Across the Lifespan-Electronic (Cohodes, McCauley et al., 2023; DISTAL-E) and if they also completed the Trauma Symptoms Checklist (TSC-40; Elliott & Briere, 1992) and demographic questions (N = 603). A subset of these participants (n = 54) were excluded for failing attention checks embedded in the DISTAL-E or TSC-40 and/or a single question at the end of the survey asking participants whether they thought their data should be eligible for inclusion in the study, yielding a final analyzable sample of N = 549.

#### Participant demographics

The majority of participants (60.8%) reported their sex assigned at birth to be female (n = 334), 39% of participants reported their sex assigned at birth to be male (n = 214), and 0.2% of participants reported their sex assigned at birth to be intersex male (n = 1). Participants' ages ranged from 18-30 years old (M = 25.48, SD = 2.78). 72.3% of the sample identified as White (n = 397), 7.7% identified as Asian (n = 42), 15.5% identified as Black or African American (n = 85), 1.5% identified as other (n = 8), and 0.4% stated that they preferred not to report their race or ethnicity (n = 2). 13.3% of the sample identified as Hispanic/Latinx (n = 73). Participants reported having completed an average of 14.70 years of education (SD = 2.20; range = 8–20 years) and reported an average annual income of \$61,396.39 (SD =\$52,070.21, range = \$0-\$600,000).

#### Procedure

All consent and assessment procedures were approved by the Institutional Review Board at Yale University. All study procedures were executed via distribution of a REDCap survey (Harris et al., 2009) on Amazon TurkPrime (managed via the Amazon CloudResearch platform). Participants provided informed consent prior to completing the DISTAL-E (electronic version of the DISTAL; Cohodes, Odriozola et al., 2023) and the TSC-40, and were thanked, debriefed, and compensated \$20 for completion of test procedures. Attention checks (e.g., "I have been paying attention") were embedded in each questionnaire that comprised the overall battery.

Recent methodological reviews have emphasized the importance of excluding low-reputation, inattentive workers from MTurk samples to maintain quality data (Hauser & Schwarz, 2016); therefore, several TurkPrime features were used to ensure the highest possible data quality, including automatic verification of worker country location, and automatic blocking of suspicious geocode locations and duplicate IP addresses. In addition, all workers were prescreened to be CloudResearch-approved participants (i.e., participants who systematically fail simple attention checks, provide "bot-like" responses, or have otherwise shown evidence that they are unwilling to follow study instructions across their history of study participation via the platform were excluded from the pool of possible study participants; see Hauser et al., 2022).

In addition to the DISTAL-E, participants also completed measures of demographics, socioeconomic status, and trauma-related symptomatology (TSC-40).

### **Materials**

### **Demographics**

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

# Dimensional Inventory of Stress and Trauma Across the Lifespan-Electronic (DISTAL-E)

The DISTAL-E is an electronic, self-administered adaptation of the Dimensional Inventory of Stress and Trauma Across the Lifespan (DISTAL; Cohodes, Odriozola et al., 2023). Like the clinical interview-based version of the measure, the structure of the DISTAL-E is broadly based on the structure of the University of California, Los Angeles, Post-Traumatic Stress Disorder Reaction Index (Pynoos & Steinberg, 2017; Steinberg et al., 2004). The DISTAL-E contains two subsections: a broad 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. Participants reported on their exposure to 24 distinct types of adversity events: serious accidental injury, illness/medical trauma, community violence, domestic violence, school violence or school emergency, physical assault, disaster, sexual abuse, physical abuse, neglect, psychological maltreatment and emotional abuse, impaired caregiving, sexual assault, kidnaping and abduction, terrorism, bereavement and witnessing death, caregiver separation, war and political violence, forced displacement, trafficking and sexual exploitation, bullying, attempted suicide, witnessing suicide, and verbal conflict.

Screening items for exposure to each type of adversity listed above were written to capture exposure to a particular type of adversity at the three levels of exposure of interest (i.e., directly experiencing, witnessing, and learning about the event happening to a close person; at least three screener questions were included for each type of event). For each type of event that was endorsed in the screening portion of the survey, participants were then asked to report on the cumulative list of ages at which they experienced a particular type of adversity (0-30, or 0 through the current age of participant, if current age less than 30). Following this comprehensive reporting of the list of ages at which a participant was exposed to a particular type of adversity, for each age of exposure that an individual endorsed, specific features of the exposure at a given age were queried (i.e., all dimensional queries were at the level of adversity type at a given age; e.g., serious accidental injury at age 5). All event-specific module questions were adapted from the DISTAL (see Cohodes, Odriozola et al., 2023, for details on the phrasing of specific module-level questions).

Of relevance to the present study, participants were asked to report whether they felt that there was an element of control inherent in their exposure to a particular type of adversity at a particular age (e.g., serious accidental injury at age 5; "Was there an element of control inherent in the serious accidental injury you experienced at age 5, for you?"). Participants were provided with the following definition of control: "Control is experienced when a person's actions have an impact on the outcome of a situation. A person is in control when they feel like they have the ability to do things that will affect what happens next in a given situation." Participants were specifically instructed to only consider themselves as the subject of control (i.e., to consider themselves, the participant, as the target subject when considering whether they experienced control, not whether any individual involved in the exposure experienced control). Participants were provided with a binary yes/no response choice (in addition to the option to decline to answer the question).

In addition, participants were asked to report whether they felt that there was an element of predictability inherent in their exposure to a particular type of adversity at a particular age (e.g., serious accidental injury at age 5; "Was there an element of predictability inherent in the serious accidental injury you experienced at age 5, for you?"). Participants were provided with the following definition of predictability: "An event is predictable when a person can make an accurate guess about what will happen next in a given situation, either based on previous experiences or knowledge of similar situations." Participants were specifically instructed to only consider themselves as the subject of predictability (i.e., to consider themselves, the participant, as the target subject when considering whether they experienced predictability, not whether any individual involved in the exposure experienced predictability). Participants were provided with a binary yes/no response choice (in addition to the option to decline to answer the question).

In order to further illustrate the measurement structure of the DISTAL-E, we outline a sample query of participants' exposure to serious accidental injuries. First, participants were asked to endorse whether they were exposed to serious accidental injury at the level of directly experiencing, witnessing, or learning about this type of event. Following this broad screener, participants were then asked to endorse all ages at which they were exposed to serious accidental injury at any of these levels of exposure. Following this age-specific screening, participants were then asked to report on the specific details of their exposure to serious accidental injury at each endorsed age (e.g., serious accidental injury at age 5), such as the degree to which this exposure was controllable or predictable, respectively.

In order to facilitate accurate self-administration of the DISTAL-E via an online platform, the REDCap survey used to administer the DISTAL-E had several features designed to minimize errors in reporting. The survey was coded so that participants were only presented with the opportunity to provide module-specific details (e.g., severity ratings) for adversity events that they endorsed experiencing. In addition, the survey response form was designed such that the adversity type and age of exposure to a particular event were piped into all module-specific questions such that participants responded to specific questions related to their experiences (e.g., "How many days of the year when you were age 5 were you exposed to serious accidental injury?"). When possible, field-specific validation (e.g., numeric ranges, text) was

enabled. Instructions for all module-specific questions were repeated at the top of every page of the survey so that they could be easily referenced. In order to minimize missing data, all fields were required to be completed prior to advancing the survey and a "Decline to answer" option was available for all questions.

### Trauma Symptoms Checklist

Participants completed the Trauma Symptoms Checklist (TSC-40; Elliott & Briere, 1992) to assess the relative frequency of distress arising from exposure to prior trauma. The TSC-40 is a self-report questionnaire consisting of 40 items representing trauma-related symptomatology (e.g., "Uncontrollable crying") rated on a 4-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. Because we were unable to monitor clinical risk for self-harm or suicidality given the online, anonymized nature of the study, the following item was removed from the study protocol: "26. Desire to physically hurt yourself." 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 = .94).

### Analytic strategy

### Derivation of dimensions of interest

Consistent with prior dimension derivation from the clinical interview-based DISTAL (Cohodes, McCauley et al., 2023), based on participants' endorsements of exposure to all types of adversity queried, an index of the total number of traumatic events experienced to date was created. In addition, to facilitate agespecific analyses, an index of participants' exposure to adversity was created for each developmental time point of interest: early childhood (0-5 years), middle childhood (6-12 years), adolescence (13-17 years), and adulthood (18-30 years or age of participant upon study entry). The total number of events characterized by controllability and/or predictability, respectively, were summed to yield indices reflecting prevalence of these elements of exposure in an individual's history of exposure to adversity, both overall, and in each developmental time period listed above (e.g., an index of the degree to which events experienced in early childhood were characterized by controllability).

### Treatment of missingness

Given that the focus of the present study was to examine the impact of exposure to traumatic stress characterized by controllability or predictability, respectively, on current functioning in adulthood, we limited the present sample to participants who provided a traumatic stress history with regard to these two dimensions (i.e., the degree to which all events were characterized by controllability or predictability was fully characterized).

### Modeling approach

We utilized a total of six models to test hypotheses for the present study. First, two *development-naïve* models were run to examine whether the degree to which an individual's lifetime history of exposure to traumatic stress was characterized as controllable (Model 1) or predictable (Model 2) moderated the association between their lifetime exposure to traumatic stress and traumarelated symptomatology assessed in adulthood. More specifically, models included main effects for the number of controllable and predictable traumatic stress exposures, respectively, total traumatic stress exposures, and an interaction between these two terms as predictors of trauma-related symptomatology assessed in adulthood. These models also included pertinent between-subject covariates (detailed below). A third development-naïve model (Model 3) examined both controllability- and predictabilityrelated interaction terms (and their main effects) within the same model to determine the unique roles of controllability and predictability accounting for the other.

Second, two development-informed models were run to examine whether stressor controllability (Model 4) and predictability (Model 5) differentially moderated the association between exposure to traumatic stress and trauma-related symptomatology assessed in adulthood when examined at distinct developmental stages. More specifically, models included main effects for the number of controllable and predictable stress exposures within each time period of interest (e.g., early childhood), total traumatic stress exposures during each time period of interest, and an interaction between these terms (with unique terms specific to early childhood, middle childhood, adolescence, and adulthood) predicting trauma-related symptomatology assessed in adulthood. Importantly, developmental stage was the only within-subject factor assessed, with retrospective controllability and predictability of stressors specific to each stage; however, due to analytic constraints outlined below, a purely between-subject implementation was used. Between-subject covariates were also included. Mirroring the development-naive approach, a third developmentinformed model (Model 6) examined all developmental stagespecific controllability- and predictability-related interaction terms in the same model to determine the unique roles of controllability and predictability accounting for the other.

### Model implementation and group-level analyses

All analyses were conducted in R (v. 4.2.2; R Core Team, 2022). Across all models, participant age, sex assigned at birth, yearly income, and years of education were included as covariates. Because the controllability and predictability of traumatic stress exposures were likely related, predictor collinearity was a potential concern; thus, the variance inflation factor (VIF; from the car [v. 3.1.1] package) was used to diagnose multicollinearity across all variables, excluding interaction terms. Multicollinearity was not a concern for any variables (VIF < 5 for all predictors; Akinwande et al., 2015). We additionally tested whether our variable representing trauma-related symptomatology was normally distributed using the Jarque-Bera test (from the tseries [v. 0.10-54] package). Results indicated that this variable was non-normally distributed, ( $X^2 = 84.59$ , p < .001). To address this, we applied a square-root transformation, and subsequent re-application of the Jarque-Bera test suggested this variable followed a normal distribution ( $X^2 = 4.01$ , p = .135). The transformed trauma-related symptomatology measure was used in all subsequent analyses. The *lm* function from the *stats* package (v. 3.6.2) was used to fit coefficients (B) for all main effects and interactions, which were then standardized ( $\beta$ ) using the lm.beta package. Finally, contrasts with corresponding t, p, and effect size (Cohen's d; generated with the *t\_to\_d* function in the *effectsize* [v. 0.8.3] package) statistics

| Table 1. | Descriptive | statistics | for | main | study | variables |
|----------|-------------|------------|-----|------|-------|-----------|
|----------|-------------|------------|-----|------|-------|-----------|

| Variable   | Mean (SD)    | Median | Range    | Variance |
|--|--------------|--------|----------|----------|
| Number of witnessed events                           | 3.71 (4.11)  | 3      | 0.0-30.0 | 16.88    |
| Number of events that were learned about             | 3.91 (3.58)  | 3      | 0.0-21.0 | 12.8     |
| Number of directly experienced events                | 6.02 (6.72)  | 4      | 0.0-47.0 | 45.21    |
| Number of controllable events<br>in early childhood  | 0.14 (0.65)  | 0      | 0.0-10.0 | 0.42     |
| Number of predictable events<br>in early childhood   | 0.14 (0.61)  | 0      | 0.0-7.0  | 0.37     |
| Number of total events in<br>early childhood         | 0.59 (1.22)  | 0      | 0.0-10.0 | 1.49     |
| Number of controllable events<br>in middle childhood | 0.63 (1.71)  | 0      | 0.0-18.0 | 2.93     |
| Number of predictable events<br>in middle childhood  | 0.85 (2.10)  | 0      | 0.0-21.0 | 4.41     |
| Number of total events in<br>middle childhood        | 2.96 (4.08)  | 2      | 0.0-37.0 | 16.66    |
| Number of controllable events in adolescence         | 0.81 (1.80)  | 0      | 0.0-16.0 | 3.25     |
| Number of predictable events in adolescence          | 0.86 (1.78)  | 0      | 0.0-15.0 | 3.18     |
| Number of total events in adolescence                | 3.54 (3.53)  | 3      | 0.0-23.0 | 12.47    |
| Number of controllable events in adulthood           | 1.34 (2.31)  | 0      | 0.0-13.0 | 5.34     |
| Number of predictable events in adulthood            | 1.27 (2.25)  | 0      | 0.0-14.0 | 5.08     |
| Number of total events in adulthood                  | 5.47 (4.39)  | 4      | 0.0-23.0 | 19.23    |
| Number of total controllable events                  | 2.92 (4.94)  | 1      | 0.0-45.0 | 24.43    |
| Number of total predictable events                   | 3.12 (5.22)  | 1      | 0.0-50.0 | 27.25    |
| Number of total events                               | 12.55 (9.36) | 11     | 0.0-59.0 | 87.55    |

SD = standard deviation.

were generated and reported. Because group-level analyses examined related hypotheses with multiple statistical tests, the rate of false positive results was likely inflated; thus, the Benjamini-Hochberg (Benjamini & Hochberg, 1995) method, implemented with the *p.adjust* function in the *stats* package, was used further limit statistical significance. All *p*-values were adjusted across models and significant main effects and interactions were retained at a threshold of corrected p < .05. Significant interactions were probed and visualized with the interactive data visualization tool (McCabe et al., 2018).

### Results

Table 1 presents descriptive statistics for all study variables and Table 2 presents zero-order correlations among study variables.

### Development-naive models

In the development-naive model solely examining the potential moderating role of controllability of lifetime traumatic stress exposure (henceforth referred to as lifetime stressor controllability) on the association between lifetime traumatic stress exposure (henceforth referred to as lifetime stress) and trauma-related symptomatology (Model 1; adj.  $R^2 = 0.30$ , p < .001), there was a significant main effect of lifetime stress ( $\beta = 0.39$ , d = 0.67,  $p_{\rm FDR} < .001$ ) and of lifetime stressor controllability ( $\beta = 0.49$ , d = 0.48,  $p_{\rm FDR} < .001$ ). In addition, consistent with hypotheses, the interaction between lifetime stress and lifetime stressor controllability emerged as a significant predictor of trauma-related symptomatology ( $\beta = -0.38$ , d = -0.37,  $p_{\rm FDR} < .001$ ), see Table 3. As is depicted in Figure 1, the association between lifetime stress and trauma-related symptomatology was attenuated among individuals who reported higher lifetime stressor controllability (i.e., a relatively higher percentage of lifetime traumatic stress exposures characterized by controllability).

In addition, in the development-naive model solely examining the potential moderating role of predictability of lifetime traumatic stress (henceforth referred to as lifetime stressor predictability) on the association between lifetime traumatic stress exposure and trauma-related symptomatology (Model 2; adj.  $R^2 = 0.27$ , p < .001), there was a significant main effect of lifetime stress ( $\beta = 0.43$ , d = 0.76,  $p_{FDR} < .001$ ) and of lifetime stressor predictability ( $\beta = 0.49$ , d = 0.47,  $p_{FDR} < .001$ ). In line with hypotheses, there was a significant interaction between lifetime stress and lifetime stressor predictability on trauma-related symptomatology  $(\beta = -0.53, d = -0.49, p_{FDR} < .001)$ , see Table 3. As is depicted in Figure 1, the association between lifetime stress and trauma-related symptomatology was attenuated among individuals who reported higher lifetime stressor predictability (i.e., a relatively higher percentage of lifetime traumatic stress exposures characterized by predictability).

When both controllability- and predictability-related terms were included in the same model (Model 3; adj.  $R^2 = 0.30$ , p < .001), significant main effects emerged for both lifetime stress ( $\beta = 0.39$ , d = 0.69,  $p_{\rm FDR} < .001$ ) and lifetime stressor predictability ( $\beta = 0.41$ , d = 0.31,  $p_{\rm FDR} < .001$ ). Only the interaction between lifetime stress and lifetime stressor predictability emerged as a significant predictor of trauma-related symptomatology ( $\beta = -0.58$ , d = -0.38,  $p_{\rm FDR} < .001$ ); see Figure 1.

### Development-informed models

Building upon the development-naive models described above, our development-informed models aimed to identify whether the moderating effects of stressor controllability and stressor predictability on the association between lifetime stress and current trauma-related symptomatology varied across developmental stages. Model 4 (adj.  $R^2 = 0.278$ , p < .001) examined whether an individual's characterization of their history of exposure to traumatic stress as controllable during each of the following developmental time periods moderated the association between exposure to traumatic stress in each of these periods and traumarelated symptomatology assessed in adulthood: early childhood, middle childhood, adolescence, and adulthood. Significant main effects emerged for early childhood stress exposure ( $\beta = 0.12$ , d = 0.21,  $p_{FDR} = .033$ ), middle childhood stressor controllability  $(\beta = 0.28, d = 0.26, p_{FDR} = .006)$ , adolescent stress exposure  $(\beta = 0.12, d = 0.20, p_{FDR} = .042)$ , adulthood stressor controllability  $(\beta = 0.34, d = 0.27, p_{FDR} = .004)$ , and adulthood stress exposure  $(\beta = 0.25, d = 0.40, p_{FDR} < .001)$ . In addition, there was a significant interaction between middle childhood stress and middle childhood stressor controllability on trauma-related symptomatology ( $\beta = -0.20$ , d = -0.20,  $p_{FDR} = .043$ ), as well as a

Table 2. Zero-order correlations among all study variables

|  | Controllable exposures in early childhood | Controllable exposures in middle childhood | Controllable exposures in adolescence | Controllable exposures in adulthood | Predictable exposures in early childhood | Predictable exposures in middle childhood | Predictable exposures in adolescence | Predictable exposures in adulthood | Total exposures in early childhood | Total exposures in middle childhood | Total exposures in adolescence | Total exposures in adulthood | _ 1  |
|--|---|--|---------------------------------------|-------------------------------------|--|---|--------------------------------------|------------------------------------|------------------------------------|-------------------------------------|--------------------------------|------------------------------|------|
| Controllable exposures in early childhood  | 1.00                                      | 0.39                                       | 0.26                                  | 0.20                                | 0.77                                     | 0.34                                      | 0.21                                 | 0.15                               | 0.60                               | 0.16                                | 0.12                           | 0.09                         |      |
| Controllable exposures in middle childhood | 0.39                                      | 1.00                                       | 0.55                                  | 0.37                                | 0.34                                     | 0.71                                      | 0.43                                 | 0.30                               | 0.27                               | 0.46                                | 0.26                           | 0.17                         | 0.8  |
| Controllable exposures in adolescence      | 0.26                                      | 0.55                                       | 1.00                                  | 0.49                                | 0.22                                     | 0.37                                      | 0.62                                 | 0.35                               | 0.15                               | 0.22                                | 0.52                           | 0.24                         | -0.6 |
| Controllable exposures in adulthood        | 0.20                                      | 0.37                                       | 0.49                                  | 1.00                                | 0.19                                     | 0.23                                      | 0.35                                 | 0.59                               | 0.15                               | 0.12                                | 0.23                           | 0.61                         | -0.4 |
| Predictable exposures in early childhood   | 0.77                                      | 0.34                                       | 0.22                                  | 0.19                                | 1.00                                     | 0.41                                      | 0.26                                 | 0.26                               | 0.66                               | 0.21                                | 0.13                           | 0.12                         | 0.2  |
| Predictable exposures in middle childhood  | 0.34                                      | 0.71                                       | 0.37                                  | 0.23                                | 0.41                                     | 1.00                                      | 0.49                                 | 0.42                               | 0.35                               | 0.57                                | 0.29                           | 0.19                         | - 0  |
| Predictable exposures in adolescence       | 0.21                                      | 0.43                                       | 0.62                                  | 0.35                                | 0.26                                     | 0.49                                      | 1.00                                 | 0.53                               | 0.15                               | 0.31                                | 0.60                           | 0.27                         |      |
| Predictable exposures in adulthood         | 0.15                                      | 0.30                                       | 0.35                                  | 0.59                                | 0.26                                     | 0.42                                      | 0.53                                 | 1.00                               | 0.15                               | 0.22                                | 0.29                           | 0.59                         | -0.2 |
| Total exposures in early childhood         | 0.60                                      | 0.27                                       | 0.15                                  | 0.15                                | 0.66                                     | 0.35                                      | 0.15                                 | 0.15                               | 1.00                               | 0.40                                | 0.16                           | 0.19                         | -0.4 |
| Total exposures in middle childhood        | 0.16                                      | 0.46                                       | 0.22                                  | 0.12                                | 0.21                                     | 0.57                                      | 0.31                                 | 0.22                               | 0.40                               | 1.00                                | 0.49                           | 0.17                         | -0.6 |

0.26 0.52 0.23

0.24 0.61

Table 3. Coefficients for development-naïve models

Total exposures in adolescence

Total exposures in adulthood

| Model                                    | Effect/interaction                        | β     | Std. β | t     | Cohen's d | р      | Corr. p |
|--|---|-------|--------|-------|-----------|--------|---------|
| Controllability (Model 1)                | Controllable Stress*                      | 0.20  | 0.49   | 5.60  | 0.48      | <.0001 | <.0001  |
|  | Total Stress*                             | 0.07  | 0.39   | 7.76  | 0.67      | <.0001 | <.0001  |
|  | Controllable Stress × Total Stress*       | -0.00 | -0.38  | -4.27 | -0.37     | <.0001 | .0001   |
| Predictability (Model 2)                 | Predictable Stress*                       | 0.20  | 0.49   | 5.42  | 0.47      | <.0001 | <.0001  |
|  | Total Stress*                             | 0.10  | 0.43   | 8.84  | 0.76      | <.0001 | <.0001  |
|  | Predictable Stress $\times$ Total Stress* | -0.01 | -0.53  | -5.73 | -0.49     | <.0001 | <.0001  |
| Controllability/Predictability (Model 3) | Controllable Stress                       | 0.08  | 0.19   | 1.71  | 0.15      | .088   | .1437   |
|  | Predictable Stress*                       | 1.64  | 0.41   | 3.63  | 0.31      | .0003  | .0008   |
|  | Total Stress*                             | 0.09  | 0.39   | 8.05  | 0.69      | <.0001 | <.0001  |
|  | Controllable Stress × Total Stress        | 0.00  | 0.03   | 0.27  | 0.02      | .789   | .8085   |
|  | Predictable Stress $\times$ Total Stress* | -0.01 | -0.58  | -4.40 | -0.38     | <.0001 | <.0001  |

0.29 0.60 0.29

0.27 0.59

0.49 1.00 0.34

0.17 0.34 1.00

0.8

-1

\**p* < .05.

significant interaction between adulthood stress and adulthood stressor controllability on trauma-related symptomatology ( $\beta = -0.33$ , d = -0.27,  $p_{FDR} = .005$ ), see Table 4. As is depicted in Figure 2, the association between traumatic stress exposure in

adulthood and trauma-related symptomatology was attenuated among individuals who reported a higher degree of controllability. With regard to predictability, Model 5 (adj.  $R^2 = 0.30$ , p < .001) examined whether an individual's characterization of their history



of exposure to traumatic stress as predictable during each of the following developmental time periods moderated the association between exposure to traumatic stress in each of these periods and trauma-related symptomatology assessed in adulthood: early childhood, middle childhood, adolescence, and adulthood. Significant main effects emerged for middle childhood stressor predictability ( $\beta = 0.39$ , d = 0.35,  $p_{FDR} < .001$ ), adolescence stress  $(\beta = 0.16, d = 0.25, p_{FDR} = .008)$ , adulthood stressor predictability  $(\beta = 0.24, d = 0.20, p_{FDR} = .044)$ , and adulthood stress  $(\beta = 0.32, p_{FDR} = .044)$ d = 0.53,  $p_{\rm FDR} < .001$ ). Additionally, three interactions – (1) the interaction between middle childhood stress and middle childhood stressor predictability ( $\beta = -0.34$ , d = -0.32,  $p_{FDR} < .001$ ), (2) the interaction between adolescence stress and adolescence stressor predictability ( $\beta = -0.20$ , d = -0.21,  $p_{FDR} = .029$ ), and (3) the interaction between adulthood stress and adulthood stressor predictability ( $\beta = -0.35$ , d = -0.29,  $p_{FDR} = .003$ ) – emerged as significant predictors of trauma-related symptomatology, see Table 4. As is depicted in Figure 2, the association between middle childhood stress and trauma-related symptomatology was attenuated among individuals who reported higher middle childhood stressor predictability (i.e., a higher degree of predictability of their traumatic stress exposures in middle childhood).

When developmentally-informed controllability- and predictability-related terms were included in the same model (Model 6; adj.  $R^2 = 0.31$ ,  $p_{\rm FDR} < .001$ ), significant main effects emerged for middle childhood stressor predictability ( $\beta = 0.41$ , d = 0.31,  $p_{\rm FDR} < .001$ ), adolescence stress ( $\beta = 0.17$ , d = 0.25,  $p_{\rm FDR} = .008$ ), adulthood stressor controllability ( $\beta = 0.30$ , d = 0.21,  $p_{\rm FDR} = .033$ ), and adulthood stress ( $\beta = 0.29$ , d = 0.44,  $p_{\rm FDR} < .001$ ). Finally, only one interaction emerged as significant – the interaction between middle childhood stress and middle childhood stressor predictability ( $\beta = -0.51$ , d = -0.33,  $p_{\rm FDR} < .001$ ; see Figure 2 and Table 4.

### Discussion

produced using the interActive data visualization tool (McCabe et al., 2018)

The present study provides an empirical test of theorized contributions of dimensional models of early adversity, particularly with regard to understanding resilience. Specifically, by examining an individual's perception of the degree to which their lifetime history of exposure to traumatic stressors was either controllable or predictable, we tested whether controllability and predictability buffered the impact of exposure to traumatic stress on mental health symptoms in adulthood. The controllability and predictability of traumatic stressors have been proposed to be important dimensions of experience (Cohodes et al., 2021; Gee & Cohodes, 2021), and both features are the subject of cross-species literature that provides support for their potential to attenuate the impact of stress (e.g., Baram et al., 2012; Davis et al., 2019; Kubala et al., 2012; Moscarello & Hartley, 2017). However, despite the theorized centrality of these features, to our knowledge, previous studies have not examined whether an individual's perception of stressors as controllable or predictable moderates the association between lifetime exposure to stress and clinical symptomatology.

Results suggest that the degree to which an individual's lifetime history of stress exposure is characterized by controllability or predictability, respectively, moderates the impact of stress exposure on trauma-related symptomatology in adulthood. Among individuals who reported a higher degree of controllability or predictability in their lifetime stress exposure to date, respectively, there was a relatively weaker association between lifetime exposure to traumatic stress and present symptomatology. Consistent with hypotheses, these findings suggest that an

### Table 4. Coefficients for development-informed models

| Model                                    | Developmental stage | Effect/Interaction                         | β     | Std. $\beta$ | t     | Cohen's d | р       | Corr. p  |
|--|---------------------|--|-------|--------------|-------|-----------|---------|----------|
| Controllability (Model 4)                | Early Childhood     | Controllable Stress                        | 0.32  | 0.10         | 1.05  | 0.09      | .2932   | .3955    |
|  | ,                   | Total Stress*                              | 0.21  | 0.12         | 2.40  | 0.21      | .0169   | .0334    |
|  |                     | Controllable Stress $\times$ Total Stress  | -0.04 | -0.11        | -1.24 | -0.11     | .2140   | .3191    |
|  | Middle Childhood    | Controllable Stress*                       | 0.35  | 0.28         | 3.01  | 0.26      | .0027   | .0061    |
|  |                     | Total Stress                               | 0.03  | 0.06         | 1.13  | 0.10      | .2589   | .3730    |
| _  |                     | Controllable Stress $\times$ Total Stress* | -0.02 | -0.20        | -2.28 | -0.20     | .0229   | .0433    |
|  | Adolescence         | Controllable Stress                        | 0.07  | 0.06         | 0.64  | 0.06      | .5257   | .6122    |
|  |                     | Total Stress*                              | 0.07  | 0.12         | 2.30  | 0.20      | .0216   | .0417    |
| _  |                     | Controllable Stress $\times$ Total Stress  | -0.00 | -0.07        | -0.79 | -0.07     | .4273   | .5421    |
|  | Adulthood           | Controllable Stress*                       | 0.31  | 0.34         | 3.17  | 0.27      | .0016   | 0.0038   |
|  |                     | Total Stress*                              | 0.12  | 0.25         | 4.66  | 0.40      | <.0001  | <0.0001  |
|  |                     | Controllable Stress $\times$ Total Stress* | -0.02 | -0.33        | -3.09 | -0.27     | 0.0021  | 0.0049   |
| Predictability (Model 5)                 | Early Childhood     | Predictable Stress                         | 0.54  | 0.16         | 1.83  | 0.16      | 0.0682  | 0.1137   |
|  |                     | Total Stress                               | 0.17  | 0.10         | 1.87  | 0.16      | 0.0623  | 0.1060   |
|  |                     | Predictable Stress $\times$ Total Stress   | -0.05 | -0.11        | -1.27 | -0.11     | 0.2034  | 0.3143   |
|  | Middle Childhood    | Predictable Stress*                        | 0.39  | 0.39         | 4.05  | 0.35      | 0.0001  | 0.0002   |
|  |                     | Total Stress                               | 0.04  | 0.07         | 1.38  | 0.12      | 0.1683  | 0.2699   |
|  |                     | Predictable Stress $\times$ Total Stress*  | -0.02 | -0.34        | -3.73 | -0.32     | 0.0002  | 0.0006   |
|  | Adolescence         | Predictable Stress                         | 0.13  | 0.11         | 1.25  | 0.11      | 0.2110  | 0.3191   |
|  |                     | Total Stress*                              | 0.10  | 0.16         | 2.91  | 0.25      | 0.0038  | 0.0081   |
|  |                     | Predictable Stress $\times$ Total Stress*  | -0.02 | -0.20        | -2.46 | -0.21     | 0.0141  | 0.0292   |
|  | Adulthood           | Predictable Stress*                        | 0.22  | 0.24         | 2.26  | 0.20      | 0.0240  | 0.0444   |
|  |                     | Total Stress*                              | 0.16  | 0.32         | 6.14  | 0.53      | <0.0001 | < 0.0001 |
|  |                     | Predictable Stress × Total Stress*         | -0.02 | -0.35        | -3.30 | -0.29     | 0.0010  | 0.0025   |
| Controllability/predictability (Model 6) | Early Childhood     | Controllable Stress                        | 0.25  | 0.08         | 0.66  | 0.06      | 0.5069  | 0.5984   |
|  |                     | Predictable Stress                         | 0.10  | 0.03         | 0.24  | 0.02      | 0.8115  | 0.8211   |
|  |                     | Total Stress                               | 0.19  | 0.11         | 2.02  | 0.18      | 0.0442  | 0.0782   |
|  |                     | Controllable Stress × Total Stress         | -0.08 | -0.20        | -1.20 | -0.10     | 0.2322  | 0.3403   |
|  |                     | Predictable Stress $\times$ Total Stress   | 0.06  | 0.12         | 0.67  | 0.06      | 0.5065  | 0.5984   |
|  | Middle Childhood    | Controllable Stress                        | 0.05  | 0.04         | 0.33  | 0.03      | 0.7415  | 0.7687   |
|  |                     | Predictable Stress*                        | 0.41  | 0.41         | 3.58  | 0.31      | 0.0004  | 0.0009   |
|  |                     | Total Stress                               | 0.04  | 0.07         | 1.32  | 0.12      | 0.1874  | 0.2950   |
|  |                     | Controllable Stress × Total Stress         | 0.01  | 0.14         | 1.06  | 0.09      | 0.2908  | 0.3955   |
|  |                     | Predictable Stress $\times$ Total Stress*  | -0.03 | -0.51        | -3.73 | -0.33     | 0.0002  | 0.0006   |
| -  | Adolescence         | Controllable Stress                        | -0.00 | -0.00        | -0.03 | -0.00     | 0.9746  | 0.9746   |
|  |                     | Predictable Stress                         | 0.06  | 0.05         | 0.56  | 0.05      | 0.5767  | 0.6535   |
|  |                     | Total Stress*                              | 0.10  | 0.17         | 2.91  | 0.25      | 0.0038  | 0.0081   |
|  |                     | Controllable Stress × Total Stress         | 0.00  | 0.05         | 0.47  | 0.04      | 0.6403  | 0.7068   |
|  |                     | Predictable Stress × Total Stress          | -0.02 | -0.21        | -2.18 | -0.19     | 0.0301  | 0.0544   |
| -  | Adulthood           | Controllable Stress*                       | 0.28  | 0.30         | 2.42  | 0.21      | 0.0160  | 0.0325   |
|  |                     | Predictable Stress                         | 0.09  | 0.10         | 0.80  | 0.07      | 0.4257  | 0.5421   |
|  |                     | Total Stress*                              | 0.14  | 0.29         | 5.00  | 0.44      | <0.0001 | <0.0001  |
|  |                     | Controllable Stress × Total Stress         | -0.02 | -0.28        | -1.98 | -0.17     | 0.0488  | 0.0847   |
|  |                     | Predictable Stress $\times$ Total Stress   | -0.01 |              | -1.11 | -0.10     | 0.2677  | 0.3793   |

\**p* < .05.

-evel of Moderator (Middle childhood stressor predictability)



Figure produced using the interActive data -igure 2. Stressor controllability and stressor predictability differentially moderate the association between developmental stage-specific stress exposure and trauma-related symptoms according to developmental stage. Respectively each model. interaction effects for Models 4, 5, and 6 are shown here. Given that multiple interaction effects were significant for Models 4 and 5, a selected interaction effect is visualized for visualization tool (McCabe et al., 2018).

individual's perception of a stressor as controllable or predictable may attenuate the impact of that stressor.

In addition, we harnessed rich phenotyping of participants' lifetime histories of traumatic stress exposure to assess whether the moderating effect of perception of a stressor as controllable or predictable varied by developmental period. Based on the broad translational literature documenting buffering effects of exposure to stressor controllability, we hypothesized that adolescence would emerge as the developmental stage during which self-reported perception of traumatic stress events as controllable would be most impactful. However, inconsistent with hypotheses, middle childhood and adulthood emerged as the periods during which control appeared to moderate the impact of traumatic stress on later symptomatology to the greatest degree. In addition, though we predicted that exposure to predictability in the context of traumatic stress might be most important in early childhood, results suggest that middle childhood, adolescence, and adulthood are the developmental time periods when predictability may most strongly buffer the negative impacts of traumatic stress. As this study represents an initial test of the interplay between timing and key experiential features of stress exposure, additional empirical work in large-scale datasets is needed to further test the potency of these moderating effects across time and in other diverse samples.

Of note, when both controllability and predictability were examined within a single model, the degree to which an individual's exposure to stress during middle childhood was characterized by predictability together with the degree to which an individual's exposure to stress during adulthood was characterized by controllability appeared to be the most salient predictors of current trauma-related symptomatology, suggesting that perceptions of predictability and controllability may differentially buffer against negative impacts of exposure to traumatic stress depending on developmental stage. While both aspects meaningfully moderated the association between total traumatic stress exposure and trauma-related symptoms in adulthood, predictability displayed larger effect sizes when directly compared with controllability in both development-naive and developmentinformed models. Results of the present study suggest that exposure to predictability may have a more potent buffering impact than controllability during earlier development, while controllability may become increasingly protective in adulthood. However, due to the individualized and complex nature of traumatic stress exposures, there are likely important interactions between both controllability and predictability at an event level, and dimensional aspects of stress other than developmental stage of exposure that play an important role in shaping symptomatology (e.g., type of stress, caregiver involvement in stress). Additional work that aims to disentangle these interactive effects will continue to shed light on the specific ways in which these two dimensions attenuate the impact of stress on later functioning.

In addition to the significant interaction effects described above, the combined model testing interactions between both controllability and predictability with exposure to traumatic stress across early childhood, middle childhood, adolescence, and adulthood displayed developmentally-specific main effects of exposure to traumatic stress during middle childhood, adolescence, and adulthood. Though interactions between timing and other specific features of exposure appear to be critically important in predicting symptomatology, in line with past theoretical and empirical work documenting the importance of timing of exposure (Cowell et al., 2015; Fox et al., 2010; Gee & Casey, 2015; Lupien et al., 2009; Manly et al., 2001; Tottenham & Sheridan, 2010), the

present study provides evidence for considerable dissociable effects of exposure to traumatic stress at specific developmental stages as well.

The present study represents a substantial deviation from the extant literature, in that here we examined participants' perceptions of stressors as characterized by controllability or predictability, rather than defining exposure categories a-priori, experimentally manipulating the degree to which an aversive exposure was controllable (Boeke et al., 2017; Cohodes, Odriozola et al., 2023; Hartley et al., 2014) or predictable (Baram et al., 2012), or, alternatively, examining broader exposure to controllability (Meyers & Wong, 1988) or unpredictability (Glynn et al., 2018) of life experiences or broader environments. Subjective perception of the impact of traumatic stress has been proposed to be a crucial factor influencing associations between exposure to traumatic stress and mental health symptoms (Danese & Widom, 2021; Smith & Pollak, 2021); therefore, a novel line of research investigating perceptions of dimensional characterization of stress exposure has the potential to further elucidate mechanisms by which stress exposure affects later functioning.

Finally, fine-grained developmental inquiry into the impact of exposure to particular experiential elements of stress reported here was facilitated by recent advances in dimensional assessment of lifetime traumatic stress exposure (Cohodes, Odriozola et al., 2023). While several previous assessment tools facilitate accurate capture of the timing of exposure to various types of traumatic stress exposures (e.g., Teicher & Parigger, 2015), the DISTAL (the electronic version of which was utilized in the present study) is the first psychometrically-validated tool that affords examination of the developmental timing of exposure to a broad range of features of stress exposure, including controllability and predictability (Cohodes, Odriozola et al., 2023). Utilizing assessment tools that afford testing of dimension- and developmental time periodspecific questions about the impact of traumatic stress on subsequent functioning will continue to be essential to field-wide efforts to improve understanding of the nuanced ways in which traumatic stress affects the developing brain and behavior.

### Limitations and future directions

The present study examined the impact of exposure to aspects of traumatic stress across development on mental health in adulthood. While this study design facilitates empirical testing of how key aspects of traumatic stress at distinct developmental stages may moderate the pernicious impacts of stress on well-being in adulthood, further work should explore whether stressor controllability and predictability exert a similar moderating effect on more proximal symptomatology (i.e., assessed during the developmental time period during which stress exposure occurred). Since the present study relied on cross-sectional, retrospective reports of exposure to traumatic stress across the lifespan, it is possible that participants' capacity to accurately recollect and document the degree to which exposures were characterized by specific features (i.e., endorsement of a stressor as either controllable or predictable) is related to the developmental time period during which that stressor occurred (with participants experiencing more difficulty reporting on earlier stressors that occurred during infancy and toddlerhood, e.g., (Williams, 1994). This effect is likely to be further exacerbated by developmental shifts in perceptions of specific features of stressors (e.g., Raab et al., 2022). These limitations are reflective of the known issues with retrospective reports in the sphere of traumatic stress assessment (Baldwin et al., 2019; Brewin et al., 1993; Hardt & Rutter, 2004). Prospective, longitudinal studies that query children's exposure to stressors – and the dimensions that characterize them – as they occur, in addition to children's trauma-related symptomatology at that specific developmental timepoint, are likely to play an important role in elucidating the timing-specific effects of exposure to various features of adversity over the course of development. In addition, the present study design allowed participants to mark traumatic events as being characterized by both predictability and controllability. While this design more accurately represents the complex nature of real-world stressors, definitively parsing the relative contributions of controllability and predictability will require further study.

Though the developmentally-specific hypotheses for the present study were largely rooted in the translational psychobiological literature, the present study only examined self-reported trauma-related symptomatology as a dependent variable given online administration of the study protocol. Future studies will build upon this initial work by examining how stressor controllability and predictability, respectively, interact with exposure to traumatic stress across the life course to predict clinical interview-derived indices of clinical functioning and symptomatology. In addition, the present study examined the impact of cumulative exposure to events that were controllable or predictable, respectively, at three levels of exposure (i.e., directly experienced, witnessed, and learned about) and to events that differed on numerous additional dimensions (e.g., persons involved, severity, event type; see Table 1). Though results of the present study suggest a buffering impact of exposure to stressors that are perceived to be controllable or predictable, with the potency of this effect differing by developmental stage, it is possible that this effect differs as a function of other critical factors such as event type and level of exposure. For example, perceptions of controllability in the context of exposure to events that are directly experienced, that are classified as maltreatment, and that are perpetrated by a caregiver may actually exacerbate the impact of exposure to stress on later functioning given the likelihood that this constellation may reflect an individual's tendency to blame themselves for such an event (i.e., given that they perceived their ability to control the event but were not indeed able to prevent it from happening). These are complex interactions that warrant substantial additional investigation.

Finally, future studies should examine associations with neurobiological outcomes in an effort to clarify the mechanisms by which exposure to controllability and predictability of adversity across development buffer against risk for negative impacts of traumatic stress on wellbeing. Incorporation of assessment tools that capture how exposure to stressor controllability and predictability varies across development in longitudinal neuroimaging studies (e.g., Hoffman et al., 2019) will also allow for more fine-grained assessment of the proximal neural signatures of exposure to these features of stress, as well as better mechanistic understanding of how exposure to stress and its features across development ultimately affects individual wellbeing in adulthood. We note that the sample utilized in the present study was predominantly White, educated, and with adequate income, and results may not generalize to a broader population; future studies utilizing online tools to query dimensional exposure to traumatic stress exposure should aim to recruit a broader, more diverse population.

In conclusion, this study provides empirical evidence for longstanding theories, validated in animal models, that controllability and predictability may buffer against the negative consequences of exposure to traumatic stress. Further, we examine specific effects of controllability and predictability during development, providing novel evidence that perceived predictability in particular may buffer traumatic stress exposure in a developmentally-specific manner that attenuates trauma-related symptoms experienced in adulthood. These findings pave the way for future longitudinal work to examine the influence of predictability and controllability on developmental trajectories, and demonstrate the promise of dimensional approaches in informing novel prevention and intervention efforts for youth exposed to traumatic stress.

Acknowledgments. The authors gratefully acknowledge individuals who facilitated data collection via the REDCap and MTurk platforms, including Samuel Haller, Christine James, Sui Tsang, and Theresa DeLuca; research assistants who facilitated database development and survey testing: Mary Margaret Schroeder and Rachel Ababio; and past lab members who made critical contributions to the development of the original DISTAL interview upon which the electronic version described here is based: Sarah McCauley, H. R. Hodges, and Jasmyne Pierre.

Funding statement. This work was supported by the National Science Foundation (NSF) CAREER Award (BCS-2145372) to D.G.G., 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 Institutes of Health Director's Early Independence Award (DP5OD021370) to D.G.G.; NSF Graduate Research Fellowship Program award (NSF DGE-1752134), 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, and a Scholar Award granted by the International Chapter of the Philanthropic Educational Organization (P.E.O. Foundation) to E.M.C.; NSF GRFP award (NSF DGE-1752134) to L.M.S.; and the Yale Child Study Center Postdoctoral T32 (Postdoctoral Research in Childhood Neuropsychiatric Disorders: T32-MH01826837) to T.J.K.

Competing interests. None.

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