

## Regular Article

# Chronic early trauma impairs emotion recognition and executive functions in youth; specifying biobehavioral precursors of risk and resilience

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### Abstract

Exposure to chronic early trauma carries lasting effects on children's well-being and adaptation. Guided by models on resilience, we assessed the interplay of biological, emotional, cognitive, and relational factors in shaping two regulatory outcomes in trauma-exposed youth: emotion recognition (ER) and executive functions (EF). A unique war-exposed cohort was followed from early childhood to early adolescence. At preadolescence (11–13 years), ER and EF were assessed and respiratory sinus arrhythmia (RSA), biomarker of parasympathetic regulation, was quantified. Mother–child dyadic reciprocity, child's avoidance symptoms, and cortisol (CT) were measured in early childhood. Trauma-exposed youth displayed impaired ER and EF abilities. Conditional process analysis described two differential indirect paths leading from early trauma to regulatory outcomes. ER was mediated by avoidance symptoms in early childhood and modulated by cortisol, such that this path was evident only for preadolescents with high, but not low, CT. In comparison, EF was mediated by the degree of dyadic reciprocity experienced in early childhood and modulated by RSA, observed only among youth with lower RSA. Findings pinpoint trauma-related disruptions to key regulatory support systems in preadolescence as mediated by early-childhood relational, clinical, and physiological factors and highlight the need to specify biobehavioral precursors of resilience toward targeted early interventions.

**Keywords:** trauma, emotion recognition, executive functions, resilience, dyadic reciprocity, RSA, longitudinal studies

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Exposure to chronic early trauma carries negative effects on multiple domains of adaptation (Feldman & Vengrober, 2011; Halevi, Djalovski, Vengrober, & Feldman, 2016; Scheeringa, Zeanah, Myers, & Putnam, 2005) and these impact overall functioning across childhood and up until adulthood (Carr, Martins, Stingel, Lemgruber, & Jurueña, 2013; Enoch, 2011; Pechtel & Pizzagalli, 2011). To date, most studies on the long-term impact of early trauma have focused on psychopathology and findings consistently demonstrate marked increases in internalizing and externalizing symptoms, prevalence of psychiatric disorders, and posttraumatic symptoms (Feldman & Vengrober, 2011; Heleniak, Jenness, Vander Stoep, McCauley, & McLaughlin, 2016; Herringa et al., 2013; Joshi & O'Donnell, 2003; Pérez-Fuentes et al., 2013; Yehuda et al., 2015). Yet, in the context of chronic early trauma, which leads to excessive activation of the stress response across extended spans of brain maturation, the consolidation of a distinct psychiatric disorder likely reflects an endpoint of impairments in cognitive, social, and emotional

regulatory support systems (Shonkoff, 2016). Consistent with this view, it has been suggested that the study of psychopathology following early trauma should be complemented by disseminating the global posttraumatic symptomatology into its distinct clusters (Sullivan, Fehon, Andres-Hyman, Lipschitz, & Grilo, 2006; Tursich et al., 2015) and assessing the maturation of regulatory functions across domains (Cicchetti, 2013; Feldman, 2015a).

Conceptual models on risk and resilience underscore the need to specify modular systems and functional processes that underpin well-being (Luthar, Cicchetti, & Becker, 2000; Masten, 2007; Rutter, 2013); for instance, attachment and caregiving, self-regulation, and allostasis and stress management are considered core components of resilience following adversity (Masten, 2007; Masten & Narayan, 2012; Southwick, Bonanno, Masten, Panter-Brick, & Yehuda, 2014). However, the pathways by which risk and protective factors operate across childhood to shape the maturation of specific regulatory functions are unclear and require a longitudinal approach (Rutter, 2013). Longitudinal studies have shown that the quality of parenting is a key predictor of psychopathology following early trauma (Betancourt, McBain, Newnham, & Brennan, 2015; Halevi et al., 2016; Koenen, Moffitt, Poulton, Martin, & Caspi, 2007; Scheeringa & Zeanah, 2001; Yirmiya, Djalovski, Motsan, Zagoory-Sharon, & Feldman, 2018). Still, while authors have suggested that resilience is not defined by a mere lack of psychopathology but also indexes systems that enable plasticity, flexibility, and adaptation (Feldman, 2020;

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Southwick *et al.*, 2014), very few studies focused on specific regulatory systems that support adaptive functioning.

In the current study, we targeted two key regulatory functions that sustain adaptive development; emotion recognition (ER) and executive functions (EF) in youth exposed to chronic early trauma since birth. We utilized a unique cohort of war-exposed children who were followed from early childhood to early adolescence. To specify pathways of risk and resilience, we focused on the avoidance cluster of posttraumatic stress disorder (PTSD) and on mother–child reciprocity in early childhood as precursors of regulatory outcomes. In addition, we tested the role of two stress biomarkers, cortisol (CT) and respiratory sinus arrhythmia (RSA), in shaping pathways to regulatory outcomes. Overall, our goal was to illuminate potential longitudinal pathways that enhance or buffer the effects of trauma on the child's regulatory capacities.

### Emotion recognition and trauma exposure: The mediating role of posttraumatic avoidance

The ability to perceive and accurately label emotions in others is a key aspect of humans' adaptation to their surroundings, which Darwin (1872) identified as innate and critical for adaptation. Studies have shown that ER – the ability to correctly detect emotions in others – is essential for typical development and pinpointed childhood as a critical period for ER development. By the end of the first year, infants can discriminate several emotions from facial and vocal cues (Caron, Caron, & Maclean, 1988) and by late childhood they recognize most emotions as precisely as adults (Durand, Gally, Seigneuric, Robichon, & Baudouin, 2007). Recognition of some emotions, such as disgust and anger, gradually mature across childhood and adolescence (Rodger, Vizioli, Ouyang, & Caldara, 2015), when sensitivity to subtle changes in emotions is acquired (Thomas, De Bellis, Graham, & LaBar, 2007).

ER provides the foundation for more complex emotional abilities and the ability to recognize emotions predicts positive social interactions, whereas deficits in ER are associated with behavioral and learning problems (Izard *et al.*, 2001). ER plays a key role in emotion regulation; it enables children to identify internal states, attach feelings to events, and practice the regulation of distinct emotions (Eisenberg, Sadovsky, & Spinrad, 2005). Deficits in ER, manifested by difficulty to identify and understand emotions, impair social skills, increase negative affect, and lead to psychopathology (Trentacosta & Fine, 2010), and ER deficits predict externalizing and internalizing psychopathologies across childhood and adolescence (Easter *et al.*, 2005; Trentacosta & Fine, 2010). Studies have shown that recognition of emotions in others is a precursor of emotion regulation (Di Maggio, Zappulla, & Pace, 2016; Izard, Stark, Trentacosta, & Schultz, 2008b) and, following emotion-based treatment, improvements in emotion regulation were mediated by change in ER (Izard, King, *et al.*, 2008).

Early exposure to trauma is associated with either augmented or suppressed ER abilities. Studies indicated decreased ability to recognize emotions following early adversity (Dvir, Ford, Hill, & Frazier, 2014; Shipman, Edwards, Brown, Swisher, & Jennings, 2005); others assessing ER from an affect-specific perspective suggested that type of adversity shapes recognition of specific emotions. While preschoolers with a history of neglect exhibited suppressed ER abilities, those with a history of physical abuse showed response bias for angry faces (Pollak, Cicchetti, Hornung, & Reed, 2000). Furthermore, children with a history of abuse required fewer physiological cues to recognize facial expression of anger and this enhanced recognition correlated

with parental anger (Pollak, Messner, Kistler, & Cohn, 2009), and it is suggested that physically abused children learned to over-tune their perception of anger cues (Pollak, 2008). However, adversity of nonfamilial nature may follow a different course. To date, studies have mainly focused on maltreated/abused children and none, to our knowledge, examined ER in nonfamilial trauma where the family must face the trauma together. In adults, some studies showed reduced ER in war veterans (Poljac, Montagne, & de Haan, 2011), whereas others found increase in ER in earthquake survivors, both with and without PTSD, and suggested that the traumatic exposure, not the presence of PTSD, is predictive of ER difficulties (Bell *et al.*, 2017). Research in adults that tested the differential effects of various PTSD clusters on ER found that avoidance symptoms, but not other posttraumatic symptom clusters, predicted ER impairment (Mazza *et al.*, 2012; Schmidt & Zachariae, 2009). Such inconsistencies underscore the need for longitudinal studies that may illuminate the developmental course of ER following trauma, taking into consideration the different posttraumatic symptom clusters.

Avoidance symptoms define one of four core symptoms in early childhood PTSD (Zero To Three, 2005) and one of three symptom clusters in adolescents and adults (American Psychiatric Association, 2013). Longitudinal studies pinpointed avoidance symptoms in early childhood as the most notable risk pathway in PTSD chronicity (Feldman & Vengrober, 2011; Feldman, Vengrober, & Ebstein, 2014; Perkonig *et al.*, 2005) and avoidant behavior is related to ER deficits (Schultz, Izard, Ackerman, & Youngstrom, 2001; Trentacosta & Fine, 2010). A study from the current cohort showed that early avoidance symptoms predicted disruptions to default mode network (DMN) connectivity in adolescence (Zeev-Wolf, Levy, Ebstein, & Feldman, 2020) and these findings are important as the DMN plays a crucial role in ER (Li, Mai, & Liu, 2014). These studies raise the possibility that early childhood avoidance may mediate the relations between early-onset trauma and ER deficits in adolescence.

### Executive functions and trauma exposure: The mediating role of parent–child reciprocity

EF are a set of core cognitive processes that facilitate the ability to restrain habitual response, detect errors, and learn by adapting behavior. Three related processes are considered core components of EF: inhibition, working memory, and cognitive flexibility; among these, inhibition is considered the key process that underlies the global EF (Gillie & Thayer, 2014; Miyake & Friedman, 2012; Valian, 2015) and studies have suggested that of the three core aspects, inhibition is the most central (Hall & Fong, 2015). Specifically, the stop-signal task, which is often used to estimate inhibitory capacities, has been suggested to reflect a common EF that underpin EF development (Chevalier, 2015; Friedman & Miyake, 2017). Furthermore, inhibition has shown to be critical for regulating emotional and cognitive arousal (Diamond, 2013). Overall, EF plays a key role in cognitive, social, and psychological development and impacts multiple dimensions of well-being, from physical and mental health to academic achievement and social competence (Diamond, 2013).

The development of EF is most rapid during early childhood and rates of growth decelerate in late childhood and adolescence (Best, Miller, & Naglieri, 2011). Global EF, and response inhibition in particular, involve activation of select prefrontal cortex (PFC) regions (Ridderinkhof, Van Den Wildenberg, Segalowitz, & Carter, 2004) and due to its prolonged maturation and

increased plasticity, the PFC is highly sensitive to both favorable and adverse rearing conditions (Kolb et al., 2012). Animal studies indicate that early-life adversities lead to PFC abnormalities, which, in turn, cause impaired EF (Hedges & Woon, 2011). In humans, EF deficits are linked with psychopathologies and impaired functioning (Fairchild et al., 2009; Penadés et al., 2007; Taylor Tavares et al., 2007) and early life stress predicts decreased EF skills across childhood (De Bellis, Hooper, Spratt, & Woolley, 2009; Hostinar, Stellern, Schaefer, Carlson, & Gunnar, 2012; McDermott et al., 2013) and adolescence (Mueller et al., 2010). Adolescents exposed to chronic trauma exhibited EF deficiency, particularly inhibition, which correlated with PTSD symptoms and functional impairment (op den Kelder, Ensink, Overbeek, Maric, & Lindauer, 2017). Given its key role in adaptation, it is important to assess EF in youth exposed to early trauma (Blair & Diamond, 2008).

Early parent–child relationship quality is a well-known predictor of later EF (Bernier, Carlson, Deschênes, & Matte-Gagné, 2012; Conway & Stifter, 2012; Cuevas et al., 2014; Deater-Deckard, 2014; Lucassen et al., 2015), apart from its role in overall well-being and adaptation. Exposure to early adversity impairs the parent–child relationship (Halevi et al., 2016; Scheeringa & Zeanah, 2001), particularly the dimensions of synchrony, reciprocity, and attunement (Feldman & Vengrober, 2011; Halevi et al., 2017; Levy, Yirmiya, Goldstein, & Feldman, 2019). Dyadic reciprocity indexes the degree of coordination, synchrony, and mutual responsiveness in the Mother×Child interaction, is individually stable from infancy to adolescence (Feldman, 2010), and mediates the links between early adverse conditions, such as maternal depression, on EF abilities in late childhood (Priel, Zeev-Wolf, Djalovski, & Feldman, 2020), suggesting that mother–child reciprocity may mediate the effects of trauma on EF.

### Risk and resilience biomarkers: Respiratory sinus arrhythmia and cortisol

Models on resilience emphasize the need to employ longitudinal designs, include theoretically based biomarkers, and test condition-specific factors that tilt children toward a resilient trajectory (Feldman, 2020; Rutter, 2013; Southwick et al., 2014). Furthermore, cognitive, emotional, and parent–child factors should be integrated with biological markers of allostasis and stress regulation (Masten, 2007). We measured two biomarkers consistently linked with risk and resilience; hypothalamic–pituitary–adrenal (HPA) axis function indexed by CT and parasympathetic control marked by RSA.

The parasympathetic nervous system, a branch of the autonomic nervous system (ANS), plays an important role in regulatory functions, supporting sustained attention and social engagement (Porges, 2001, 2007), and RSA is a reliable marker of parasympathetic activity (Porges, 1995). High resting RSA indexes flexible adaptation to changing environmental condition, a key feature of resilience (Beauchaine, 2001; Feldman, 2020). In contrast, low resting RSA is associated with high-risk rearing, troubled mother–child relationship, and functional impairments (Beauchaine & Thayer, 2015; Feldman, 2006; Shahrestani, Stewart, Quintana, Hickie, & Guastella, 2014).

Parasympathetic activity plays a role in allostasis via its connection with PFC activity and its inhibitory function, and RSA has been linked with response inhibition (Thayer, 2006; Thayer & Sternberg, 2006). Response inhibition, a core component of EF, gauges context-dependent responses that enable anticipatory,

resource-sensitive (i.e. allostatic) regulation based on ongoing parasympathetic inputs (Gillie & Thayer, 2014; Thayer, 2006). Due to the negative effects of adversity on parenting quality and the associations between parenting and EF, it is possible that child RSA may serve a protective role in the pathway leading from early trauma to EF through its impact on the link between dyadic reciprocity and EF. This is supported by a study showing that baseline RSA modulated the link between sensitive parenting in the toddler years and EF at 5 years (Gueron-Sela et al., 2017).

CT is the end product of the HPA axis, the body central stress-management system (Hellhammer, Wüst, & Kudielka, 2009; Tsigos & Chrousos, 2002). Early trauma leads to significant alterations in HPA axis activity and CT levels, including cases of abuse and neglect (Cicchetti & Rogosch, 2001; Cicchetti, Rogosch, Gunnar, & Toth, 2010; De Bellis & Zisk, 2014) or war-related trauma (Feldman, Vengrober, Eidelman-Rothman, & Zagoory-Sharon, 2013; Yirmiya et al., 2018). However, findings on CT and PTSD are mixed; some studies found that children with PTSD show attenuated basal CT (Feldman, Vengrober, et al., 2013), others showed that elevated basal CT predicted the development of PTSD following trauma (Pervanidou et al., 2007), while still others found no difference in basal CT between trauma-exposed children with and without PTSD (Carrion et al., 2002). Assessment of the different PTSD clusters showed specific associations of CT and the avoidance cluster (Goenjian et al., 2003; Yehuda et al., 1995) and this is supported by a study showing higher basal CT in healthy children with avoidant behavior (Blair, Peters, & Granger, 2004).

To further understand the inconsistencies in the associations of CT and trauma, one solution is to view the role of HPA system activity within a complex net of mutual influences leading from trauma exposure to regulatory outcomes. CT has shown to function as risk indicator in the longitudinal course leading from trauma and PTSD symptoms to various aspect of development (Dunlop & Wong, 2019; Kamin & Kertes, 2017; Olf & van Zuiden, 2017; van Ast et al., 2013). These studies suggest that CT may have a modulating role in the complex pathways leading from early trauma to the maturation of ER and EF through its effect on avoidance symptoms. For instance, under conditions of chronic stress both avoidance and CT were related to ER processing (Roelofs, Elzinga, & Rotteveel, 2005). Similarly, CT moderated the links between early adversity and children's emotion regulation (Von Klitzing et al., 2012), which is closely related to ER (Eisenberg et al., 2005), and CT interacted with social withdrawal in predicting ER processing (van Peer, Spinhoven, Dijk, & Roelofs, 2009). Finally, early-life stress interacted with genes implicated in HPA axis functioning in predicting ER in healthy adults (Hartling et al., 2019). These studies support the hypothesis that CT may impact the pathway leading from early trauma to later ER abilities through its interaction with avoidance symptomatology in early childhood.

### The current study

We addressed the effects of early and chronic exposure to war-related trauma on children's EF and ER at the transition to adolescence, focusing on two regulatory outcomes critical for well-being and adaptive functioning. While studies have indicated that early adversity impairs EF and ER, the mechanisms and pathways underlying these associations are mainly unknown. Utilizing a unique cohort of war-exposed preadolescents exposed to the same external trauma, we were able to utilize a unique "natural experiment" to test these longitudinal pathways. Our goal was

to assess the interplay of biological, relational, and clinical components as they shape each other over time toward a more comprehensive model, consistent with models that emphasize the need to study resilience from a longitudinal, biobehavioral perspective (Feldman, 2020; Masten, 2007; Rutter, 2013; Southwick et al., 2014).

Three hypotheses were formulated. First, we expected that preadolescents exposed to chronic war-related trauma would show impaired EF and ER compared to non-exposed controls. Second, we expected that the association between trauma exposure and EF would be mediated by the degree of reciprocity experienced in early childhood and that this indirect pathway may be modulated by the child's RSA. Finally, we hypothesized that avoidance symptomatology in early childhood would mediate the association between early exposure and preadolescence ER and this cascade may be moderated by child's CT levels. Overall, our goal was to test pathways from chronic early trauma to ER and EF in preadolescence as mediated by the parent-child relationship, avoidance symptoms, CT, and RSA to specify mechanisms of risk and resilience.

## Method

### Participants

Participants were recruited in early childhood and followed four times from early childhood to early adolescence. Here, we used data collected in early childhood (T1) and early adolescence (T4).

#### Early childhood (T1)

A total of 232 war-exposed and control children-mother dyads were initially recruited ( $M = 2.76$  years,  $SD = 0.91$ ). The war-exposed group included 148 children and mothers living in Sderot, a small Israeli town located near the Gaza border whose residents experienced frequent and unpredictable rockets and missile attacks for nearly 20 years, with periods of exacerbation occurring every few months. During these years, dozens of civilians were killed, and thousands were injured. Thus, putting children and families in daily tangible threat of death or severe injury. Children in this group all live in the same neighborhoods, thereby exposed to similar daily threat characteristics since birth. The control group comprised 84 dyads from comparable towns in the central part of Israel, matched for age, gender, birth order, parental age and education, maternal employment, and marital status. Other forms of trauma, abuse, or neglect were excluded (Feldman & Vengrober, 2011). Children were followed in *middle childhood* (T2) ( $M = 7.68$  years,  $SD = 0.7$ ) and *late childhood* (T3) ( $M = 9.3$  years,  $SD = 1.41$ ) and data from the T2 and T3 visits appear elsewhere (Feldman, Vengrober, et al., 2014; Halevi et al., 2017, 2016).

#### Early adolescence (T4)

Children were revisited at preadolescence (11–13 years). The current study utilized data from 111 children who participated in both T1 and T4. Of these, 58 were from the war-exposed group and 53 were controls. Ten children did not have valid RSA data due to excessive movement artifacts or device failure. Ten children did not complete the response inhibition task, six did not complete the ER task, and eight participants did not have valid T1 CT data. Attrition was mainly related to inability to locate families and no demographic differences were found between families who did or did not participate in the current stage in any demographic variable. The study was approved by the local Institutional Review

Board. Mothers signed an informed consent and children received a gift certificate for their participation.

### Procedure

#### Early childhood (T1)

Mother-child dyads were visited at home in the afternoon hours to control for diurnal variability in CT. Following acquaintance, baseline saliva samples were collected by the mother by placing a Salivette (Sarstedt, Rommelsdorf, Germany) in the child's mouth for one minute. Mother and child then engaged in a 10-min free interaction with preselected toys. Next, child PTSD symptoms in the different clusters were diagnosed through a detailed maternal interview, consistent with the guidelines of the *Diagnostic classification of mental health and developmental disorders of infancy and early childhood*: (Revised edition) (Zero To Three, 2005)

#### Early adolescence (T4)

Following arrival at the lab, children were connected by a trained research assistant to a Mindware device (Mindware Technologies Ltd, Gahanna, OH) which collected electrocardiogram (ECG) and respiration (RSP) data for a three-minute baseline. Following, children completed the ER and EF tasks.

### Measures

#### Early childhood (T1)

*Child PTSD avoidance symptoms.* Trained clinicians with a background in early childhood development and psychopathology visited families at home and diagnosed child's PTSD using the Diagnostic Classification: Zero-to-Three (Zero To Three, 2005). Mothers were interviewed extensively, and clinicians elicited detailed information concerning PTSD symptoms by focusing on the nature of the trauma, proximity to the traumatic event, and child's emotional reaction to specific traumatic events. Clinicians were supervised by a senior clinical child psychologist and a child psychiatrist, and cases were conferred every few weeks. A similar interview was conducted with the control group mothers. Control mothers were asked to describe events in the last few months experienced by the child as "traumatic", stressful, or emotionally challenging. Posttraumatic symptoms were assessed in the four categories of early childhood PTSD: re-experiencing, avoidance, hyper-arousal, and fears and aggressions, and the avoidance symptom cluster was used here.

*Dyadic reciprocity.* To assess dyadic reciprocity, mother-child free play interactions were videotaped during the home visits, and interactions were coded offline using the Coding Interactive Behavior (CIB) manual (Feldman, 1998). The CIB is a well-validated global system comprising 45 codes aggregated into several constructs that have been widely used in the research of typical and high-risk contexts across multiple cultures and developmental stages (for review, Feldman, 2012a). Coding was conducted by trained coders, blind to any other information, and reliability on 20% of the interactions exceeded 93% on all codes ( $k = .84$ , range .78–95). Each scale included in the constructs is coded from 1 – target behavior has minimal expression during the interaction to 5 – target behavior has maximal expression during the interaction. Scales are averaged to create the final construct consistent with wide-ranging earlier research (Feldman, 2012b). Here we used the dyadic reciprocity construct that indexes

the degree in which the dyad participates in a joint activity, interact in a “give-and-take” manner and the interaction is fluent, rhythmic, and mutually adaptive.

**Cortisol.** Salivettes were kept cooled until thawed before being centrifuged at 48C at 1000g for 15 min. The samples were then stored at -208C until assayed. CT levels were assayed using a commercial ELISA kit (Assay Design, MI). Measurements were performed in duplicate according to the kit’s instructions. Levels were calculated by using MATLAB version 7 (MathWorks, Natick, MA) according to the relevant standard curves.

#### Early adolescence (T4)

**Respiratory sinus arrhythmia (RSA).** Child’s ECG and RSP signals were collected using MindWare Bionex device (Gahanna, OH). ECG was recorded using a modified lead II configuration and RSP was assessed with a respiratory belt placed around the chest. ECG and RSP data were sampled at 1000 Hz. Data were transmitted to a computer in an adjacent room monitored by a research assistant and captured using BioLab Software version 3.0 (MindWare Technologies Ltd, Gahanna, OH). Data were processed offline using HRV 3.0.21 software (Mindware Technologies Ltd, Gahanna, OH). ECG data were visually inspected for artifacts – missing or errored heartbeats were manually deleted or inserted as appropriate. Data of participants with more than 10% problematic R-waves were excluded. Following, the data were tapered with a Hamming window and submitted to a fast Fourier transform to derive the spectral distribution. RSA was quantified as the integral power within the respiratory frequency band of 0.2–0.42 Hz appropriate for child age (Fleming et al., 2011).

**Executive function (EF).** Child’s EF was indexed by the stop signal task from the computerized Cambridge Neuropsychological Testing Automated Battery system (CANTAB, Cambridge Cognition, Cambridge, UK). The stop signal task measures the ability to inhibit a prepotent response. The task consists of an arrow stimulus presented on a screen and randomly pointing to either right or left direction. The child was instructed to press the corresponding button on a two-button press pad depending on the arrow’s direction as quickly and accurately as possible. Following participant’s introduction to the test and completion of one block of 16 practice trials, the participant was told to withhold response and not select the button if he/she hears an auditory signal that is paired with the presented arrow. The CANTAB system utilizes a staircase design for the delay between the go stimulus and the inhibitory stimuli. This allows the task to adapt to the performance of the participant, narrowing in on the 50% rate of success rate for inhibition. Stop signal reaction time (SSRT) is an estimate of the length of time between the go stimulus and the inhibitory stimulus at which the subject was able to successfully inhibit their response on 50% of the trials. SSRT is considered a reliable parameter of response inhibition capacity and reflects the individual’s capability to internally suppress a response. SSRT has been widely used in both human and animal studies, with SSRT scores representing EF capacity in a reversed manner (Aron, 2007, 2008; Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Boehler, Appelbaum, Krebs, Hopf, & Woldorff, 2010). Thus, higher SSRT was used here to index lower EF and lower SSRT was used to index higher EF.

**Emotion recognition (ER).** We used the Emotion Recognition Toolbox, a computerized ER paradigm developed in our lab

and validated in prior research (Priel et al., 2020). The toolbox tests the recognition and knowledge of single and complex emotions that are presented at different levels of intensity. The software includes 15 short 30-s videos that demonstrate happy, sad, angry, and neutral emotions of low or high intensity. After each movie, the child is asked by the program whether each emotion appeared in the movie and once the child pressed yes or no, another set appears and asks if these emotions also appeared. The proportion scores for each emotion were calculated and the final score was the overall percentage of correct recognition of emotions from the child’s overall response, consistent with our prior work in children of a similar age (Priel et al., 2020).

#### Data analytic plan

Data were analyzed by SPSS 23 (IBM Corp., Armonk, NY). Differences between exposed and controls were examined using the *t* test for the continuous variables and chi-square test for the categorical variables. For testing of our first hypothesis regarding group differences in EF and ER, we utilized two *t* tests and applied a Bonferroni correction to reduce errors due to multiple testing ( $\alpha = 0.05/2 = 0.025$ ). Pearson correlations measured the relationships between study variables. Next, we used conditional process analysis to assess a comprehensive model which includes the direct and moderated mediated paths leading from early trauma exposure to youth regulatory outcomes. Exposure was entered as the independent variable and was dummy coded (*control* = “0”, *war-exposed* = “1”). Early childhood avoidance symptoms and dyadic reciprocity were entered as parallel mediators. CT and RSA were entered as moderators and EF and ER as the dependent variables.

We used PROCESS for SPSS (v. 3.3). PROCESS employs bootstrapping calculations, a nonparametric resampling procedure, which provides the most powerful and reasonable method of obtaining confidence limits for conditional indirect effects at different levels of the moderators (Hayes, 2018a). This approach accounts for violation of normality assumptions that can occur in more traditional approaches to mediation and moderation (Hayes, 2018a). For this analysis, bias-corrected standard errors and confidence intervals (CIs) were generated using 10,000 bootstrapped resamples drawn to derive the 95% CI. Conditional mediation is considered present when the CI for the estimation of the indirect effect does not contain zero. Specifically, we used PROCESS Model 16, as it provides indices of partial moderated mediation that quantify the linear relationship between a moderator and an indirect effect when a second moderator is held constant (Hayes, 2018b). This model allowed us to test moderated mediation with the two independent moderators, CT and RSA, rather than performing separate moderated mediation analysis for each moderator. Consistent with Hayes’ guidelines for analyzing a model with multiple dependent variables, we performed two similar analyses with the dependent variable, ER and EF, entered alternatively, while generating a random bootstrapping seed used in both analyses (Hayes, 2018a, pp. 145–146).

## Results

### Preliminary analysis

Sociodemographic information of the exposed and control groups appears in Table 1. No differences in demographic variables were found between the groups. For a fuller model, we tested group

**Table 1.** Comparison on child and mother sociodemographic variables between the groups

Characteristic	Control ( <i>n</i> = 53)	Exposed ( <i>n</i> = 58)	<i>t</i> / $\chi^2$	<i>p</i>
<b>Child</b>				
Age, mean ( <i>SD</i> )	11.55 (1.14)	11.76 (1.31)	<i>t</i> <sub>(107)</sub> = 0.90	n.s.
Girls, <i>N</i>	36 67.9%	31 53.4%	$\chi^2_{(1)} = 2.42$	n.s.
<b>Mother</b>				
Age, mean ( <i>SD</i> )	41.26 (4.40)	40.06 (5.81)	<i>t</i> <sub>(109)</sub> = -1.22	n.s.
Education, <i>N</i> High-school/Above	9 17%	18 31%	$\chi^2_{(1)} = 2.97$	n.s.
Married, <i>N</i>	49 92.5	56 96.6	$\chi^2_{(1)} = 0.91$	n.s.
SES 1-9 ( <i>SD</i> )	4.52 (1.42)	4.13 (1.02)	<i>t</i> <sub>(107)</sub> = -1.66	n.s.

SES = socioeconomic status

differences of study variables that were not hypothesized to have group differences (Figure 1a–d). As can be seen, in early childhood, exposed children exhibited more avoidance symptoms and lower dyadic reciprocity with their mothers compared with controls.

### Group comparisons and correlations

Consistent with our first hypothesis, exposed preadolescents ( $M = 217.44$ ,  $SD = 47.97$ ) showed significantly higher SSRT (and thereby lower EF) than controls ( $M = 193.30$ ,  $SD = 43.98$ ),  $t_{(99)} = 2.63$ ,  $p = .01$ . In addition, exposed preadolescents ( $M = 76.65$ ,  $SD = 10.53$ ) showed significantly lower ER than controls ( $M = 81.45$ ,  $SD = 10.53$ ),  $t_{(103)} = 2.59$ ,  $p = .01$  (Figure 1e–f).

Pearson's correlations among study variables appear in Table 2. As seen, early childhood avoidance symptoms were significantly correlated with diminished ER in preadolescence as well as with lower dyadic reciprocity in early childhood. Dyadic reciprocity longitudinally correlated with better ER and EF skills; dyadic reciprocity also correlated with lower basal CT levels. The two regulatory outcomes, ER and EF were interrelated.

### Conditional process analysis

#### Pathways to executive function

Results of the moderated mediation model with EF (indexed by SSRT in a reversed manner) as the outcome is presented in Figure 2 and unstandardized path coefficients are presented.

Since EF and ER were interrelated, ER scores were entered as covariates in this analysis (of note, analysis without covariates showed similar pattern of results; see Supplementary Materials Table S.1). As seen, trauma exposures linked with lower dyadic reciprocity and higher avoidance symptoms in early childhood, but only dyadic reciprocity was then significantly related to EF in preadolescence. In addition, the direct association of exposure and later EF was not significant in the presence of the other intervals, indicating an indirect path. The partial moderated mediation index allows to quantify the linear relationship between a moderator and an indirect effect when a second moderator is held constant (Hayes, 2018b). RSA had a significant moderation effect on the indirect pathway leading from trauma exposure to SSRT via dyadic reciprocity (index = -6.29,  $SE = 3.57$ , 95% CI [-14.25, -0.51]).

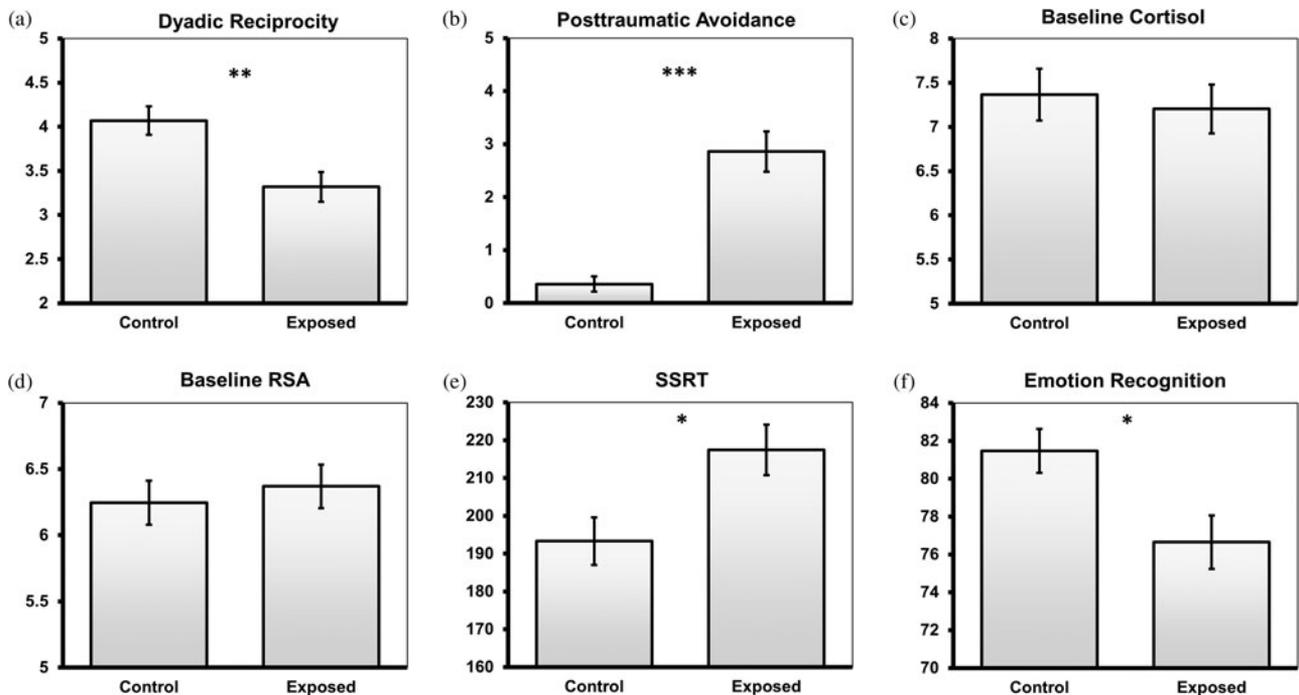
To further understand this indirect effect, we evaluated this path at high (1 *SD* above mean) and low (1 *SD* below mean) levels

of RSA while conditioning the other moderator (CT) at mean level, consistent with Hayes' recommendations (2018b, p. 16). A significant simple slope of dyadic reciprocity was found when RSA was low ( $B = -19.85$ ,  $SE = 8.01$ , 95% CI [-35.83, -3.86]). In contrast, when RSA was high the simple slope of dyadic reciprocity was nonsignificant ( $B = 2.85$ ,  $SE = 7.85$ , 95% CI [-12.82, 18.52]). Hence, at low levels of RSA, there was a negative link between early dyadic reciprocity and preadolescence SSRT, whereas at high levels of RSA such association was not evident. Furthermore, the overall path leading from early exposure to SSRT via dyadic reciprocity was significant only when RSA levels were low ( $B = 12.84$ ,  $SE = 6.94$ , 95% CI [1.57, 28.34]). Overall, these results suggest that while chronic trauma exposure impacts preadolescents' EF via reducing parenting quality in early life, better parasympathetic functioning can buffer this effect.

#### Pathways to emotion recognition

Results of the moderated mediation model with ER as the outcome is presented in Figure 3, showing unstandardized coefficients. Here, EF (SSRT score) was used as covariate (analysis without covariates showed similar pattern of results; see Supplementary Materials Table S.2). As seen in Figure 3, trauma exposure linked with lower dyadic reciprocity and higher avoidance symptoms, but, unlike the previous model, none was significantly associated with ER. However, an interaction of avoidance and CT was significant [ $B = -0.67$ ,  $SE = 0.2$ , 95% CL [-1.06, -0.27]]. Furthermore, significant unconditional mediation is not a prerequisite for a significant moderated mediation (Hayes, 2018a, p. 426). The conditional process analysis revealed that the partial moderated mediation index was significant, indicating CT having a significant moderation effect on the indirect pathway leading from early exposure to ER via avoidance symptoms (index = -1.77,  $SE = 0.7$ , 95% CI [-3.39, -0.67]) and this conditional pathway being the only significant path leading from exposure to ER.

Evaluation of this path at high (1 *SD* above mean) and low levels (1 *SD* below mean) of CT revealed a significant negative simple slope of avoidance when CT was high ( $B = -1.61$ ,  $SE = 0.55$ , 95% CI [-2.71, -0.5]), but a nonsignificant simple slope of avoidance when CT was low ( $B = 0.81$ ,  $SE = 0.59$ , 95% CI [-0.37, 1.99]). Hence, at high levels of CT, there was an association between avoidance and preadolescence ER, whereas at low levels of CT such association was not significant. In accordance, the overall path leading from early exposure to ER through dyadic reciprocity was significant only when CT levels were high ( $B = -1.61$ ,  $SE =$



**Figure 1.** Group differences in study variables. SSRT, stop signal reaction time. RSA, respiratory sinus arrhythmia. Error bars represent the standard error of the mean. \* $p < .05$ ; \*\* $p < .01$ ; \*\*\* $p < .001$ .

**Table 2.** Pearson correlations among study variables

Variable	1	2	3	4	5
1. Dyadic reciprocity – early childhood					
2. Avoidance symptoms – early childhood	–.339**				
3. Basal cortisol – early childhood	–.342**	–.130			
4. Basal RSA	.142	.007	.064		
5. Emotion recognition	.379**	–.279*	.092	.131	
6. SSRT	–.288*	.143	.016	.026	–.293*

\*Bonferroni adjusted two-sided significance level of  $\alpha < 0.05/15 = p < .0033$ .

\*\*Bonferroni adjusted two-sided significance level of  $\alpha < 0.01/15 = p < .00066$ .

RSA = respiratory sinus arrhythmia; SSRT = stop signal reaction time

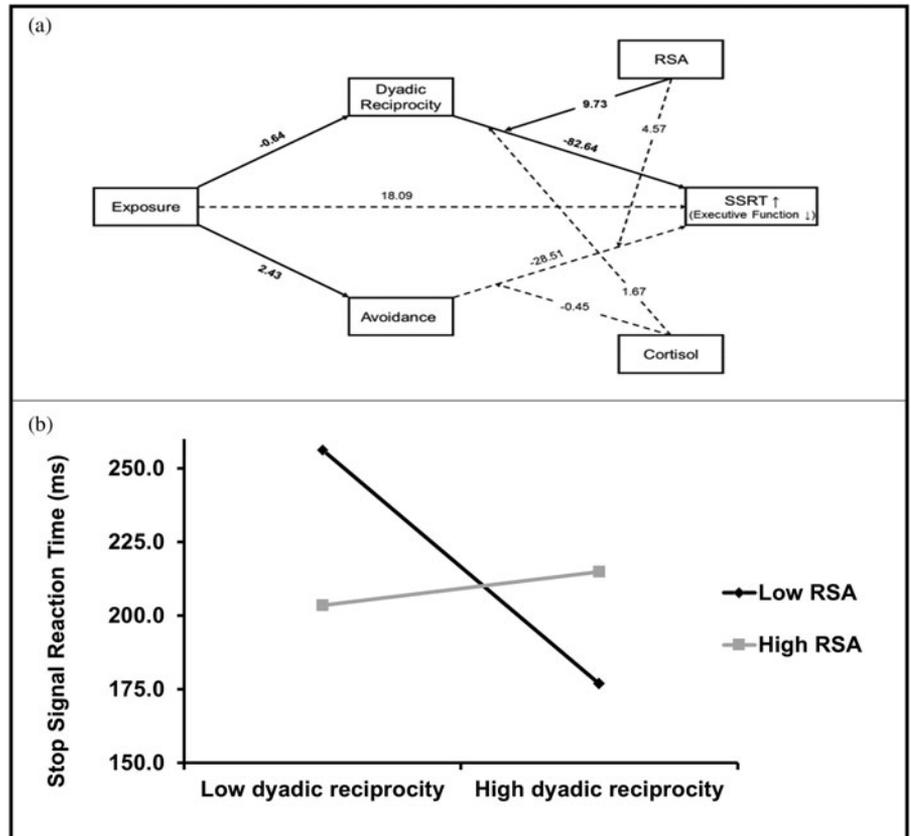
0.55, 95% CI [–2.71, –0.5]). In combination, these results suggest that the effect of early exposure on later ER deficits is mediated by avoidance symptoms in early childhood, but this indirect pathway depends on CT levels. Specifically, this pathway was evident in children with high levels of CT in early childhood, but not in those with low CT.

## Discussion

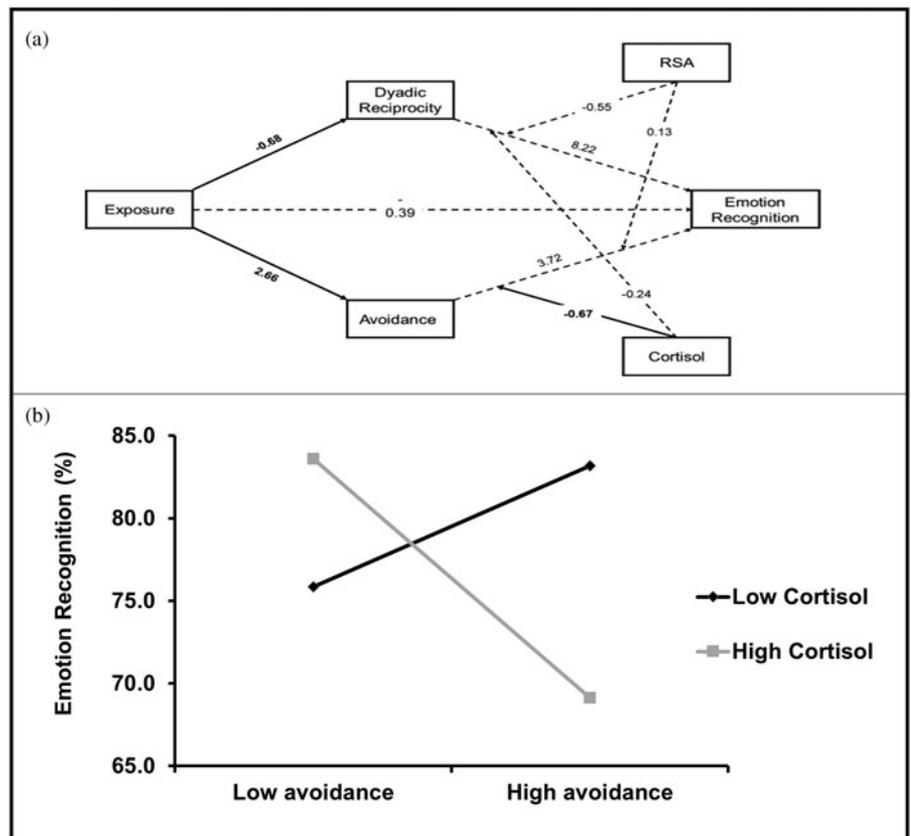
The current study examined the effects of chronic early trauma on the maturation of two regulatory functions: ER and EF, abilities that play a key role in the individual's well-being and social adaptation. To test the interplay of risk and resilience factors on these regulatory functions, we capitalized on a “natural experiment” and followed a unique cohort of preadolescents who were exposed to the same war-related trauma from early childhood to early adolescence. This design allowed us to trace longitudinal biobehavioral pathways by which early trauma exposure impairs the development of core psychological functions at the transition to

adolescence. Specifically, we examined mother–child reciprocity and avoidance symptoms as differential mediators linking trauma exposure with EF and ER. Furthermore, we tested two biomarkers consistently shown to index stress reactivity; RSA and CT production as factors that may condition these pathways. The components of our model and the dynamic interplay among them received substantial empirical attention in the context of early adversity; yet, this is the first study, to our knowledge, that examines a comprehensive model that specifies the longitudinal interplay of these factors in shaping ER and EF as modulated by stress-related biomarkers.

Research on the effects of chronic early trauma on later development has mainly addressed its impact on the prevalence of psychopathology, externalizing and internalizing behaviors, and posttraumatic symptoms. Here we wished to expand the developmental psychopathology perspective by assessing cognitive and emotional regulatory support systems. Both ER and EF consolidate during childhood and by the transition to adolescence enable children to function adaptively in their environment. Still, these



**Figure 2.** (a) Moderated mediation model of the pathway from early trauma exposure to preadolescence SSRT. Reported are unstandardized coefficients. Emotion recognition score was included as covariate (not depicted). Solid lines represent significant effects, and dashed lines represent nonsignificant effects. (b) Simple slopes of early-childhood dyadic reciprocity predicting preadolescence SSRT for 1 SD below and above the mean of RSA.



**Figure 3.** (a) Moderated mediation model of the pathway from early trauma exposure to preadolescence emotion recognition. Reported are unstandardized coefficients. SSRT was included as covariate (not depicted). Solid lines represent significant effects, and dashed lines represent nonsignificant effects. (b) Simple slopes of early-childhood posttraumatic avoidance predicting preadolescence emotion recognition for 1 SD below and above the mean of early-childhood basal cortisol. SSRT, stop signal reaction time. RSA, respiratory sinus arrhythmia.

two abilities have not, to our knowledge, been tested in combination as dual outcomes of early adversity. Overall, our results demonstrate that chronic early trauma compromises children's EF and ER capacities at this important developmental transition. The two potential pathways measured in early childhood as leading to such disruptions involved factors impacted by trauma; the mother-child reciprocal exchange and the increase in avoidance symptoms in early childhood. Our findings demonstrate two differential indirect and conditional pathways leading from trauma exposure to compromised EF and ER, which, in their presence, the direct associations between early trauma and later regulatory outcomes are altered. These pathways included psychological and social factors that interact with the child's biological components via two key biomarkers: RSA and CT. As such, our findings are consistent with both the biobehavioral synchrony model (Feldman, 2015b, 2017) and models on resilience that emphasize the need for condition-specific perspective that considers the interchange of psychological, cognitive, social, and biological factors (Feldman, 2020; Masten, 2007; Rutter, 2013; Southwick et al., 2014).

Deficiencies in EF following trauma exposure were mediated by the trauma's negative effect on the mother-child relationship. We have previously shown that disruptions to the reciprocal component of the mother-child relationship across the first decade of life mediated the effects of chronic maternal depression on child EF at ten years (Priel et al., 2020), suggesting that executive abilities may be particularly sensitive to the experience of reciprocal caregiving beginning in early childhood. Further cascade of this path related to the adolescent's parasympathetic activity. Further analyses showed that the decrease in EF following trauma, which was mediated by reduced early reciprocity, is only evident under a specific condition: when the child's parasympathetic functioning is less adaptive and baseline RSA is low, highlighting the resilience-promoting impact of autonomic-system functionality. Results also confirmed our third hypothesis, that the links between trauma and compromised ER are mediated by the increase in avoidance symptoms as moderated by early baseline CT levels. Further analysis revealed that CT interacted with avoidance symptoms and thus, the path leading from trauma exposure to ER via elevated avoidance symptoms was evident in children with higher basal CT levels in early childhood but was absent in children who exhibited lower stress response.

Careful examination of our results outlines a complex pattern of interactions between automatic nervous system activity and dyadic factors in the context of early trauma. Higher dyadic reciprocity was linked with better child EF a decade later but only among children with lower RSA. These results are consistent with a study indicating that RSA moderated the association between sensitive parenting and later EF such that a positive association was found only among children with low basal RSA (Gueron-Sela et al., 2017). Our study expands previous knowledge to the context of trauma and the important topic of resilience within a longitudinal context. Because RSA was only measured in our study in adolescence, it was not possible to see its longitudinal effects. However, in a longitudinal study that followed premature infants from birth to adolescence, we found that RSA at age 10 impacted concurrent EF (Feldman, Rosenthal, & Eidelman, 2014), but ten-year EF were unrelated to RSA measured in infancy, suggesting that the maturation of parasympathetic control across childhood may be an important buffer for the development of cognitive control, and further longitudinal studies are needed to specify the earliest time point when RSA

becomes a resilient biomarker of later cognitive and regulatory abilities.

Studies have shown that parents exposed to trauma tend to form impaired relationships with their children (Feldman & Vengrober, 2011; Halevi et al., 2016) and these altered patterns of Parent×Child interaction carry negative effects on the child's future mental health and symptomatology (Scheeringa & Zeanah, 2001). Since low RSA is considered a marker of deficient self-regulatory capacities (Porges, 2007), our findings raise the possibility that children exhibiting lower RSA rely more on their mother's reciprocal and attuned parenting for self-regulation. The parent-child relationship plays a vital role in the emergence of self-regulation, which sustains EF, and the reliance on the parents is especially crucial during early childhood and under conditions of adversity (Cuevas et al., 2014; Feldman, 2015a). In the context of mass trauma, mothers are exposed to the same external danger and have diminished resources to develop carefully attuned reciprocity with their young children, leading to a cascade of regulatory difficulties that may impede cognitive flexibility and EF.

Our results on the modulating role of RSA in the context of trauma may echo the differential susceptibility model (Belsky, Bakermans-Kranenburg, & Van Ijzendoorn, 2007), which suggests that environmental hazards impact outcome as a function of the child's innate regulatory abilities. Extant research has indicated that high baseline RSA is considered to be adaptive (Beauchaine, 2001; Porges, 2007), and buffers in the association between unfavorable environmental contexts and child outcomes (Mezulis, Crystal, Ahles, & Crowell, 2015), while low RSA is typically framed as a risk factor in this association (Benjamin Hinnant, Erath, & El-Sheikh, 2015; Dyer, Blocker, Day, & Bean, 2016). However, other studies showed that vulnerable children, particularly those with low basal RSA who are more reactive (Beauchaine, 2001; Rottenberg, 2007), are more susceptible to variations in environmental provisions and are able to gain more from positive parenting (Belsky & Pluess, 2009), consistent with the differential susceptibility model which proposes that negatively reactive infants require especially responsive parenting for positive outcome (Belsky & Pluess, 2013).

Results of our model can be addressed from two different perspectives that incorporate the differential susceptibility frame. Implementing a narrow perspective on the association between dyadic reciprocity and EF, our results can indicate that higher dyadic reciprocity is beneficial for later EF only for children with low basal RSA. Viewing the results from this narrow perspective may also suggest that low RSA children who developed in the context of high dyadic reciprocity showed the highest EF capabilities. However, when observing the overall longitudinal path, it becomes apparent that trauma exposure significantly decreased dyadic reciprocity, hence placing exposed children with low RSA in a vulnerable position, while not impacting the high RSA children who seemed less dependent on the mother's attuned caregiving for the development of EF skills. Dyadic reciprocity is individually stable from infancy to adolescence (Feldman, 2010; Feldman, Bamberger, & Kanat-Maymon, 2013), and thus, the early decrease in reciprocity probably continues across childhood and thus, the impairments in EF in preadolescence among children with low RSA emerge in the context of chronic disruptions to caregiving. These seemingly conflicting results underscore the importance of a longitudinal perspective and illustrate how the same biomarker can serve as both a risk factor on one level, but a resilience factor if viewed from a wider perspective. In addition, these results demonstrate the complex manner by

which autonomic activity shapes the relationship between early trauma exposure and later EF and add a longitudinal angle to the differential susceptibility model.

Findings for the emotional arm of our study showed that the maturation of ER is sensitive to different biological and behavioral features, particularly avoidant symptoms and HPA axis functionality. The avoidance cluster of PTSD has been noted as the most risky pathway in PTSD chronicity following early trauma exposure (Feldman et al., 2014; Feldman & Vengrober, 2011; Perkonig et al., 2005) and is a salient precursor of social dysfunction and professional maladjustment in adults exposed to childhood-onset trauma (Van Voorhees et al., 2012). Our results expand knowledge on the role of early avoidance symptoms, particularly their sensitivity to HPA axis functioning in early childhood, in shaping future disruptions to emotional development. Studies have demonstrated that the avoidance symptoms cluster is the one most strongly linked with dysregulation of CT secretion and various aspects of HPA axis functioning (Yehuda et al., 2009, 1995) and dysregulation of CT metabolism has been suggested as a causal factor in the expression of avoidance symptoms and the degree of recovery following trauma (Whitaker, Farooq, Edwards, & Gilpin, 2016; Yehuda et al., 2009). As suggested by Rutter (2013), resilience involves a complex interplay of biological and behavioral factors as they impact each other over time, and our findings may pinpoint condition-specific, age-specific, and biomarker-specific pathways to the maturation of distinct regulatory outcomes.

The early-childhood PTSD avoidance cluster mediated the path from early trauma to later ER while also interacting with early childhood basal CT levels, such that the overall path was only evident in children who had high CT levels and absent in children exhibiting low CT. ER matures through the practice afforded by social interactions, which allow the child to read, understand, and respond to others' emotions and place emotions within specific social context; hence, avoidance seriously limits the child's opportunities for practice (Schultz et al., 2001). Yet social interactions are insufficient for the proper development of ER, as this skill also depends on cognitive factors, such as memory consolidation (Schultz et al., 2001). Basal CT levels are associated with impaired memory consolidation of emotional faces (Van Honk et al., 2003) and thus, it is possible that while avoidance limits the child's ability to practice ER, a significant and long-lasting deficit in ER is evident only when HPA axis dysfunction may interrupt the consolidation of these experiences into meaningful emotional knowledge.

Several study limitations should be mentioned in the interpretation of our findings. Although this study utilized a prospective longitudinal design, it is important to emphasize that no causal inferences can be derived from the results. Moreover, important biological, neural, or psychological measures that have not been tested here possibly interact with the pathways presented, and further research is needed to assess such multidimensional links. Furthermore, we did not collect EF and ER across childhood and such data could have shed valuable light on the developmental points in which these functions are most sensitive to adversity in order to specify intervention effort. EF was evaluated by one task of response inhibition, which, although central to EF, should be enhanced by attention to other aspects of EF. Moreover, there are findings that suggest a low reliability of individual differences in chief cognitive paradigms, among them the stop-signal task (Hedge, Powell, & Sumner, 2018). These findings underline the need to evaluate inhibition using other tasks as well as other

aspects of EF. Our study did not include fathers, and the father-child relationship could have added valuable insights. Finally, this study is based on a unique cohort of dyads exposed to repeated wartime attacks and further research replicating our findings in other situations of political violence or family-related trauma is essential to generalize our results.

The prevalence of armed conflicts has increased substantially during the past decade and approximately one in five children worldwide is growing up in the context of ethnic, national, or tribal war and violence (Fox, 2019; Østby, Rusted, & Tollefsen, 2018). Much further research is needed to fully understand how such chronic conditions impact children and families. Our study has important clinical implications by demonstrating the lasting effects of war exposure on key functioning of regulatory support systems at a key developmental transition, underscoring the importance of early PTSD diagnosis and highlighting the centrality of early avoidance symptoms. Our results on the interaction between RSA and early dyadic reciprocity emphasize the importance of building dyadic interventions to young children and their family that incorporate biofeedback elements and suggest that children with reduced physiological regulation may benefit from such dyadic interventions. The findings, therefore, may help the construction of more targeted interventions that focus on social engagement and reciprocity, begin early in life, include both mother and child, and consider biological systems that provide the support for the development of long-term adaptation.

**Supplementary Material.** The supplementary material for this article can be found at <https://doi.org/10.1017/S0954579421000067>

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**Conflicts of Interest.** None.

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