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The Social Transmission of Risk: Maternal Stress Physiology, Synchronous Parenting, and Well-Being Mediate the Effects of War Exposure on Child Psychopathology

Galit Halevi and Amir Djalovski
Bar-Ilan University

Yaniv Kanat-Maymon
Interdisciplinary Center Herzliya, Herzlia, Israel

Karen Yirmiya, Orna Zagoory-Sharon, and Lee Koren
Bar-Ilan University

Ruth Feldman
Bar-Ilan University and Yale University

While chronic early stress increases child susceptibility to psychopathology, risk and resilience trajectories are shaped by maternal social influences whose role requires much further research in longitudinal studies. We examined the social transmission of risk by assessing paths leading from war-exposure to child symptoms as mediated by 3 sources of maternal social influence; stress physiology, synchronous parenting, and psychiatric disorder. Mothers and children living in a zone of continuous war were assessed in early childhood (1.5–5 years) and the current study revisited families in late (9–11years) childhood ($N = 177$; $N = 101$ war-exposed; $N = 76$ controls). At both time-points, maternal and child's salivary cortisol (SC), social behavior, and externalizing and internalizing symptoms were assessed. In late childhood, hair cortisol concentrations (HCC) were also measured and mother and child underwent psychiatric diagnosis. The social transmission model was tested against 2 alternative models; 1 proposing direct impact of war on children without maternal mediation, the other predicting late-childhood symptoms from early childhood variables, not change trajectories. Path analysis controlling for early childhood variables supported our conceptual model. Whereas maternal psychopathology was directly linked with child symptoms, defining direct mediation, the impact of maternal stress hormones was indirect and passed through stress contagion mechanisms involving coupling between maternal and child's HCC and SC. Similarly, maternal synchrony linked with child social engagement as the pathway to reduced symptomatology. Findings underscore the critical role of maternal stress physiology, attuned behavior, and well-being in shaping child psychopathology amid adversity and specify direct and indirect paths by which mothers stand between war and the child.

General Scientific Summary

We tested the social transmission of risk and resilience amid adversity in a cohort of war-exposed children and mothers observed across the first decade of life. Maternal chronic and phasic stress biomarkers, indexed by salivary and hair cortisol, attuned parenting, and psychiatric disorder mediated the effects of war on child psychopathology, charting both direct paths to child symptoms and indirect paths via child stress hormones and diminished social engagement. Our findings underscore the critical role of maternal well-being, stress physiology, and relational behavior in mediating the effects of chronic early trauma on child symptom formation.

Keywords: stress response, resilience, war exposure, salivary cortisol, hair cortisol concentrations

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Galit Halevi and Amir Djalovski, Department of Psychology, Bar-Ilan University; Yaniv Kanat-Maymon, Baruch Ivcher School of Psychology, Interdisciplinary Center Herzliya, Herzlia, Israel; Karen Yirmiya, Department of Psychology, Bar-Ilan University; Orna Zagoory-Sharon, The Gonda Brain Sciences Center, Bar-Ilan University; Lee Koren, Department of Biology, Bar-Ilan University; Ruth Feldman, Department of Psychology, The Gonda Brain Sciences Center, Bar-Ilan University; and Child Study Center, Yale University.

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Correspondence concerning this article should be addressed to Ruth Feldman, who is now at the Baruch Ivcher School of Psychology, Interdisciplinary Center, Herzlia, Israel, 4601010. E-mail: feldman.ruth@gmail.com

Although it is well-documented that children growing up in the context of early adversity—whether poverty, abuse, domestic violence, or political strife—are more susceptible to psychopathology, there are components in the social environment that chart pathways of risk and resilience amid hardship (Copeland, Keeler, Angold, & Costello, 2007; MacMillan et al., 2001; Masten, 2001). The most important source of social influence is the mother (Feldman, 2015a, 2015b; Silverman & La Greca, 2002). Biobehavioral models of human development underscore the role of maternal stress physiology, attuned parenting, and well-being in mediating the effects of chronic early stress on child psychopathology (Gewirtz, Forgatch, & Wieling, 2008; Laucht, Esser, & Schmidt, 2001; Scheeringa & Zeanah, 2001; Van Den Bergh, Mulder, Mennes, & Glover, 2005). These human studies parallel research in animal models indicating that maternal glococorticoids and parenting behavior exert a lifelong impact on offspring stress regulation, brain maturation, and adaptation to the social ecology both independently and jointly (Gunnar, Hostinar, Sanchez, Tottenham, & Sullivan, 2015; Seckl & Meaney, 2004). Yet, while numerous perspectives suggest that maternal stress physiology and sensitive parenting adjust the dial of the child's stress response and social repertoire, shaping trajectories of risk and resilience (Albers, Riksen-Walraven, Sweep, & Weerth, 2008; Gunnar & Hostinar, 2015; Hennessy, Kaiser, & Sachser, 2009), very few studies followed children reared within a defined early life stress and measured maternal and child's stress biomarkers and social behavior over lengthy periods. As such, the specific pathways leading from early adversity to child symptomatology as mediated by maternal social influences require much further research.

In the current longitudinal study, we sampled a unique cohort of children growing up in a zone of continuous war who have been followed with their mothers across the first decade of life. Children were previously assessed in early (1.5–5 years) and middle (5–8 years) childhood and links between maternal anxiety and depressive symptoms and sensitive parenting, children's cortisol, and allelic variability on the oxytocin receptor gene with child symptoms at these ages have been reported (Feldman & Vengrober, 2011; Feldman, Vengrober, & Ebstein, 2014; Feldman, Vengrober, Eidelman-Rothman, & Zagoory-Sharon, 2013). In the current study we revisited families in late childhood (9–11 years) to assess maternal social influences within a biobehavioral frame and to address change trajectories in social behavior and child symptoms across the first decade. Specifically, we examined the role of maternal stress physiology, synchronous parenting, and psychiatric disorder in mediating the effects of war exposure on the development of externalizing and internalizing symptoms in 10-year old children. We focused on the hypothalamic-pituitary-adrenal (HPA) axis as the body's main stress response system (Tsigos & Chrousos, 2002), and measured its chronic and phasic aspects as indexed by hair cortisol concentrations (HCC) and salivary cortisol levels (SC) in mother and child. To model change trajectories, we took into account maternal synchrony, child social engagement, and child symptoms measured in early childhood. The central hypothesis guiding our study was that in the context of chronic early adversity, the social transmission of risk occurs via three maternal pathways; maternal stress physiology, parenting behavior, and psychological well-being.

Chronic Early Stress and Alterations in HPA Reactivity

Chronic early stress bears long-term negative consequences for physical and mental health throughout life (Baldwin et al., 2016; Feurer, Hammen, & Gibb, 2016; Herringa et al., 2013; Kempke et al., 2013), and alterations in the body's stress response is a key pathway leading from early stress to later psychopathology (McEwen & Gianaros, 2011). The stress response involves complex interactions of the sympathetic nervous system and the HPA axis allowing to both prepare for danger and return to baseline once threat is removed (Laurent, Ablow, & Measelle, 2012; Lupien, McEwen, Gunnar, & Heim, 2009). These alterations enable organisms to adapt to environmental threats through feedback between the anterior pituitary, hypothalamus, and cortex of the adrenal glands (McEwen & Gianaros, 2010). The HPA axis comprises the hormones CRH, ACTH, and cortisol that interact with contextual factors to shape both momentary stress reactivity and long-term stress physiology (Ellis & Essex, 2007; Romeo, 2010). Cortisol plays a key role in the stress response by preventing overactivation of the immune system to threats and by acting on the hypothalamus and pituitary gland via negative feedback loops to foster homeostasis once safety is achieved (Kudielka, Hellhammer, & Wüst, 2009; Miller, Chen, & Zhou, 2007; Smyth, Hucklebridge, Thorn, Evans, & Clow, 2013). Basal cortisol regulates multiple physiological and metabolic processes that shape immune-system functionality (Munck & Náray-Fejes-Tóth, 1994). Following stress, glucocorticoids are crucially important for stress resistance and when not properly regulated stress-induced cortisol can in of itself carry detrimental effects.

Chronic stress in early childhood—when events repeatedly exceed the child's coping abilities—leads to alterations in cortisol secretion because of frequent HPA-axis activation (Alink, Cicchetti, Kim, & Rogosch, 2012; Bick et al., 2015; Cicchetti, Rogosch, Gunnar, & Toth, 2010; Doom, Cicchetti, & Rogosch, 2014; Kalmakis, Meyer, Chiodo, & Leung, 2015; Trickett, Noll, Susman, Shenk, & Putnam, 2010; Zilioli et al., 2016). HPA-axis dysregulation and allostatic load mediate the long-term effects of chronic stress on child psychiatric disorders, including depression and *posttraumatic stress disorder* (PTSD; Nanni, Uher, & Danese, 2012), and alters functioning of the nervous, endocrine, and immune systems that lead to physical illness (Danese et al., 2009). These effects have been reported in children exposed to prenatal risk (Hunter, Minnis, & Wilson, 2011), poverty (Blair, Raver, & Granger, 2011), institutional rearing (Koss, Mliner, Donzella, & Gunnar, 2016), or violence (Evans, Kim, Ting, Tesher, & Shannis, 2007).

Research on the role of the HPA system in mediating the effects of chronic stress on mental health has yielded inconsistent findings regarding the nature of the dysregulation (Klaassens, Giltay, Cuijpers, van Veen, & Zitman, 2012; Meewisse, Reitsma, de Vries, Gersons, & Olff, 2007). Several studies measuring cortisol in individuals with PTSD in blood, saliva, or urine showed lower levels of cortisol compared with controls (Morris, Compas, & Garber, 2012), while others found the opposite trend, including higher baseline SC (Inslicht et al., 2006; Lindley, Carlson, & Benoit, 2004; Young & Breslau, 2004) and augmented SC reactivity to stress (Blair et al., 2008; Bosch et al., 2012). Notably, in studies of trauma-exposed children, including those with and

without PTSD, higher cortisol production has been reported (Feldman, Vengrober, Eidelman-Rothman, & Zagoory-Sharon, 2013; Steudte-Schmiedgen, Kirschbaum, Alexander, & Stalder, 2016). Overall, these studies suggest that alterations in HPA-axis activity mediate the effects of chronic early stress on the development of psychopathology in children.

Hair Cortisol Concentrations as a Biomarker of Chronic Stress

Most studies on HPA-axis functioning, trauma, and adversity determined cortisol levels from saliva; however, SC captures only short-term stress reactivity (Levine et al., 2007). An important advance in recent years has been the use of hair cortisol analysis in humans. Measuring steroids in integrated matrices, such as hair, is a developing field that may provide new insights into the chronic aspects of the stress response (Burnard, Ralph, Hynd, Edwards, & Tilbrook, 2016; Russell, Koren, Rieder, & Van Uum, 2012; Stalder & Kirschbaum, 2012; Staufenbiel, Penninx, Spijker, Elzinga, & van Rossum, 2013). While circulating steroid levels represent momentary total state, hair provides a longer time-frame where each centimeter of hair approximates 1 month of cortisol secretion, presumably integrating free steroids over the time of growth (Russell et al., 2012; Stalder, Steudte-Schmiedgen, et al., 2016). HCC may provide a retrospective month-by-month measure of cumulative cortisol secretion and serve as a reliable biomarker of chronic stress (Hinkelmann et al., 2013; Ouellette et al., 2015; Simmons et al., 2016; Steudte et al., 2013; Vanaelst et al., 2012).

In adults, HCC has been linked with chronic psychosocial stress (Dettenborn, Tietze, Bruckner, & Kirschbaum, 2010; Manenschijn, van Kruysbergen, de Jong, Koper, & van Rossum, 2011; Stalder et al., 2014), stress-related somatic conditions (Kuehl et al., 2015; Manenschijn et al., 2013; Pereg et al., 2011; Stalder et al., 2013), and psychiatric disorders (Dettenborn et al., 2012; Manenschijn et al., 2012; Steudte, Stalder, et al., 2011; Wei et al., 2015). HCC has also been studied in the context of trauma (Gao et al., 2014; Hinkelmann et al., 2013; Kalmakis et al., 2015; Luo et al., 2012; Schalinski, Elbert, Steudte-Schmiedgen, & Kirschbaum, 2015; Steudte et al., 2013; Steudte, Kolassa et al., 2011; Steudte-Schmiedgen et al., 2015). Elevated HCC were found among earthquake survivors (Gao et al., 2014) and HCC correlated with the number of stressful life events (Grassi-Oliveira et al., 2012; Karlén, Ludvigsson, Frostell, Theodorsson, & Faresjö, 2011; Karlén et al., 2015; Staufenbiel et al., 2014).

Very few studies measured HCC in children and none assessed HCC within a prospective longitudinal design in parent and child. Overall, HCC has been linked with children's lifetime trauma (Simmons et al., 2016) and fearfulness upon school entry (Groeneveld et al., 2013). Higher parenting stress and socioemotional difficulties have been associated with elevated HCC in children (Palmer et al., 2013). Yet, some inconsistencies have been reported; While higher HCC has been linked with major childhood life events (Vanaelst et al., 2012) and socioeconomic status (Rippe et al., 2016; Vliegenthart et al., 2016), HCC in children exposed to intimate partner violence did not differ from controls (Boeckel, Viola, Daruy-Filho, Martinez, & Grassi-Oliveira, 2017). Some of these inconsistencies may stem from differences in trauma severity, temporal distance from the event, chronic versus acute trauma, and child age (Dettenborn, Tietze, Kirschbaum, & Stalder, 2012; Steudte-Schmiedgen et al., 2016). Similar to SC, HCC has been shown to mediate the effects of maltreatment on child symptoms (Stalder, Ising, et al., 2016), suggesting that HCC may mediate the effects of war on child psychopathology. Furthermore, the few studies that measured HCC and SCC concurrently showed no significant correlations between matrices (Short et al., 2016; Flom, St. John, Meyer, & Tarullo, 2017); thus, the two biomarkers of the stress response may tap different aspects of HPA-axis functionality and may chart distinct pathways leading from war exposure to child symptomatology.

Maternal Influences on Children's Stress Physiology and Psychopathology; Cortisol Linkage, Synchronous Parenting, and Maternal Psychopathology

Extant evidence in humans and animal models indicates that patterns of maternal care shape HPA-reactivity in offspring (Gunnar & Hostinar, 2015; Jessop & Turner-Cobb, 2008; Moriceau & Sullivan, 2006; Shionoya, Moriceau, Bradstock, & Sullivan, 2007). Beginning in infancy, when the child's HPA system is labile, and across childhood and adolescence, sensitive and synchronous parenting attenuates children's HPA reactivity, as expressed in smaller cortisol increases or quicker return to baseline after stress (Albers et al., 2008; Blair et al., 2008; Berry et al., 2016; Feldman, Singer, & Zagoory, 2010). In contrast, intrusive, overcontrolling, or unavailable mothering has been associated with augmented cortisol response (Bosquet Enlow et al., 2014; Feldman et al., 2009), and increased risk for psychopathology in trauma-exposed children (Kelley et al., 2010; Scheeringa & Zeanah, 2001).

An important mechanism by which maternal stress physiology mediates the effects of stress exposure on children's stress reactivity is cortisol linkage, the matching of maternal and child's cortisol production (Atkinson et al., 2013; Papp, Pendry, & Adam, 2009; Sethre-Hofstad, Stansbury, & Rice, 2002). Maternal regulation of the infant's stress reactivity begins in utero and is thought to signal to the developing HPA system the amount of stress the future environment contains (Gitau, Fisk, Teixeira, Cameron, & Glover, 2001). Beginning in infancy, linkage between maternal and infant's cortisol baseline and reactivity have been described, associated with the degree of synchronous parenting that augments or attenuates the infant's cortisol levels (Feldman et al., 2010; Ostfeld-Etzion, Golan, Hirschler-Guttenberg, Zagoory-Sharon, & Feldman, 2015; Saxbe et al., 2017; Thompson & Trevathan, 2008). Cortisol linkage in childhood and adolescence has been similarly associated with sensitive parenting (Feldman et al., 2013; Pratt et al., 2017), and maternal cortisol is among the strongest predictors of child cortisol production (Bright, Granger, & Frick, 2012). Notably, cortisol linkage has been found across matrices; in SC after stress-inducing paradigms, diurnal cortisol, or HCC (Bright, Granger, & Frick, 2012; Ouellette et al., 2015; Pratt et al., 2017). In addition, cortisol linkage has been shown to mediate the effects of chronic stress on the development of children's externalizing and internalizing symptoms, for instance, in the current cohort of war-exposed children in early childhood (SC: Feldman et al., 2013) or among children growing up in disadvantaged neighborhoods (HCC: Turner et al., 2016).

In addition to stress physiology, sensitive and synchronous mothering, the mother's online adaptation to the child's state and

signals (Feldman, 2007, 2016), has been shown to decrease in contexts of early adversity (Creech, Hadley, & Borsari, 2014; Kelley et al., 2010; Lewig, Arney, & Salveron, 2010). Reduced maternal synchrony, in turn, has been linked with increased child externalizing and internalizing symptoms both concurrently and longitudinally (Feldman, 2010; Feldman & Eidelman, 2004; Kok et al., 2013; Mäntymaa et al., 2009). One important pathway by which the decrease in maternal synchrony mediates the effects of adversity on child symptoms is via the reduction in children's social engagement and increased withdrawal associated with insensitive parenting (Feldman, 2015a, 2015b). Synchronous parenting prepares children to function competently within the social world, acquire a social repertoire, master appropriate social skills, and develop a socially engaged rather than withdrawn style (Feldman, 2010; Feldman & Masalha, 2010; Marshal & Fox, 2006), and thus, social engagement may function as an indirect mediator of war exposure on child psychopathology. Because alterations in children's HPA-axis functioning have been associated with variability in social engagement (Apter-Levi et al., 2016; Feldman et al., 2013; Saxbe et al., 2017), it is also possible that social engagement may mediate the link between child cortisol and symptom formation.

Finally, extant research has indicated that maternal psychopathology, particularly the presence of maternal psychiatric disorders, is related to increased child externalizing and internalizing symptoms (Goodman et al., 2011; Leen-Feldner et al., 2013). Numerous studies demonstrated such links in the context of war exposure, and war-exposed children of more symptomatic mothers were found to be at greater risk to develop psychopathology, including findings from the current cohort in early childhood (Feldman & Vengrober, 2011; Kaitz, Levy, Ebstein, Faraone, & Mankuta, 2009; Laor, Wolmer, & Cohen, 2001; van Ee, Kleber, & Mooren, 2012). These findings indicate that maternal mental health may mediate the effects of war exposure on child symptoms and suggest that the impact of maternal psychiatric status on child symptom formation is direct, stemming, in part, from shared genetic risk (Rutter, Moffitt, & Caspi, 2006).

The Current Study

The current study addressed the social transmission of risk in the context of a well-defined early adversity by using a unique cohort of children and mothers living in Sderot, a small Israeli town located 10 km from the Gaza border and exposed to repeated and unpredictable missile attacks since birth. Such cohort provides a unique "natural experiment" to study the effects of chronic early trauma on child psychopathology, as all exposed children experienced the same wartime stressors while biological and social factors in mother and child differentiated among families. Children were recruited in early childhood and several prior publications reported findings from earlier stages (Feldman & Vengrober, 2011; Feldman et al., 2013, 2014). Here, we assessed mothers and children in late childhood (9–11 years) to study maternal social influences and focused on change trajectories in social behavior and child symptoms from early to late childhood.

We focused on three pathways by which mothers may mediate the effects of war on the development of child symptoms; Maternal HPA-axis functioning, including its chronic (HCC) and phasic (SC) aspects, maternal synchronous parenting, and maternal psychiatric disorder, charting independent biological, behavioral, and mental health paths for the social transmission of risk. However, while maternal psychiatric disorder was expected to be directly linked with increased child externalizing and internalizing symptoms (direct mediation), maternal stress hormones and synchronous parenting was expected to correlate with the parallel variable in the child (child HCC, SC, and social engagement) in charting the biological and social pathways leading from war exposure to child symptoms (indirect mediation).

Three global hypotheses were formulated. The first considered mean-level differences between war-exposed and control families. Much research has shown that trauma increases psychopathology in mothers and children (Feldman & Vengrober, 2011; Scheeringa, Myers, Putnam, & Zeanah, 2015), and we expected more Axis-I psychopathology in war-exposed mothers and children and more externalizing and internalizing symptoms in the children. Consistent with prior research, we also expected lower synchorny and social engagement and higher SC and HCC in the exposed group (Fayyad et al., 2016; Feldman, Vengrober, et al., 2013; Simmons et al., 2016).

The second hypothesis considered the mother's role in mediating the links between war exposure and child symptoms. Three mediated paths were proposed, via maternal HPA-axis functioning (HCC and SC), synchronous parenting, and psychiatric condition. Because of the key role of children's HPA-axis activity and social engagement in shaping risk and resilience trajectories, we expected that the effects of maternal cortisol and synchronous parenting on child symptoms would pass through their impact on children's cortisol and social engagement.

The third hypothesis considered model testing and addressed the best-fitted path model on the basis of theory and research in comparison with two alternatives. (1) To probe the dynamics of maternal influences across the first decade, we measured change in maternal and child social behavior and child externalizing and internalizing symptoms from early to late childhood. We expected that such model of change would provide a better fit to the data as compared with a model predicting child symptoms in late childhood from variables measured in early childhood. Theoretically, we contended that a model focusing on change trajectories would provide better fit than a model built on pure "sensitive period" perspective. (2) Our focus on the social transmission of risk considered the key role of maternal influences in shaping psychopathology in children. We expected that a model charting the effects of war on child symptoms as mediated by maternal influences—maternal stress hormones, behavior, and psychopathology—would provide a better fit to the data than a model assuming direct effects of war on child hormones, social behavior, and symptoms without including the maternal influences.

Method

Participants

Children were recruited in early childhood (T1: 1.5–5 years) and followed in middle childhood (T2: 5–8 years) and late childhood (T3: 9–11) years. The T2 assessment included only psychiatric evaluation of children and thus, no data from this visit is included here.

In early childhood, participants included 232 children (age: M = 2.76 years, SD = 0.91), of whom 47.6% were males and 47.1% firstborns, and their mothers. The war-exposed group comprised 148 families living in the same frontline neighborhoods in Sderot, Israel, located 10 km from the Gaza border. Individuals in Sderot have been exposed to unpredictable and continuous rocket attacks for more than a decade, leaving only 15 s to enter protected spaces after hearing alert sirens, and exposing citizens to frequent mortar shelling without prior signals. The control group included 84 nonexposed families from comparable towns. Controls were recruited from towns within the greater Tel-Aviv area that were equivalent to the town of Sderot in terms of population size, socioeconomic composition, and housing and employment opportunities. Controls were matched to the exposed group in age, gender, birth order, parental age and education, maternal employment, and marital status and were screened for other trauma. Detailed description of the T1 visit is reported elsewhere (Feldman & Vengrober, 2011; Feldman et al., 2013).

There were 210 children of the original sample were reassessed in middle childhood (5–8 years), including 130 war exposed (87.8% of the original sample) and 80 controls (95.2% of the original sample).

In the current study, we revisited 177 families of the initial sample in late childhood when children were between 9 and 11 years (M = 9.3 years, SD = 1.41), including 48% males and 45.1% firstborns. The war-exposed group included 101 mothers and children (77.6% of the sample at T2) and the control group comprised 76 dyads (95% of the sample at T2). Attrition was mainly related to inability to locate the families and no differences were found in any demographic or early childhood variables between families who did or did not participate in T3. The study was approved by the Institutional Review Board of Bar Ilan University, and all parents signed informed consent.

Procedure

Early childhood. Families were visited at home for about 3.5 hr during the afternoon (after 3:00 p.m.), to control for diurnal variability in cortisol (Engert, Efanov, Dedovic, Dagher, & Pruessner, 2011). Visits included hormone collection, free play, maternal trauma interview, child fear paradigm, and self-reports (see Feldman et al., 2013). Following acquaintance, baseline saliva samples were collected from mother and child by mother placing a Salivette (Sarstedt, Rommelsdorft, Germany) in her own and the child's mouth for 1 min. The experimenter then placed a box of preselected age-appropriate toys and ask mother to play with her child as she normally does.

After play, mothers were interviewed in detail about the traumatic events, the child and family's war-related trauma history, and the child's PTSD symptoms in the re-experiencing, avoidance, hyper-arousal, and fears and aggression categories according to the DC 0–5 guidelines (Zero to Three, 2005). Children were present in the room and were videotaped during the evocation of trauma.

After, the experimenter administered the fear paradigm (Goldsmith & Rothbart, 1996), and finally, mothers completed self-report measures. Second salivary samples were collected 20 min after the fear episode and the final salivary sample 15 min thereafter.

Fear paradigm. In this well-validated procedure to elicit fear in young children, adapted from the Lab-Tab (Goldsmith & Rothbart, 1996), the experimenter puts on four masks of increasing fearfulness while child is looking: clown, pet animal, scary animal, and ghost. The experimenter puts on the mask, calls the child's name, and leaves it on for 10 s.

Late childhood. Families were visited at home for approximately 2 hr (similarly scheduled after 3:00 p.m.). Following acquaintance, baseline saliva samples were collected from mother and child. Next, mothers were interviewed with the SCID (First, Spitzer, Gibbon, & Williams, 1997) and DAWBA (Goodman, Ford, Richards, Gatward, & Meltzer, 2000) to determine psychiatric diagnosis for mother and child and reported on child internalizing and externalizing symptoms. Following, a second salivary sample collection (60 min from baseline), mothers and children were videotaped in two interaction paradigms, well-validated for children at this age, each lasting for 7 min (Feldman, 2015a; Feldman, Rosenthal, & Eidelman, 2014; Lebowitz et al., 2017). The first paradigm involved a conflict discussion, in which dyads were asked to discuss a typical conflict in their relationship. The second was a positive interaction where mother and child were asked to plan together their "best day ever." Ten minutes after the end of the interactions, the third salivary sample was collected. At the end of the visit, hair samples were collected from mother and child.

Measures

Maternal psychiatric diagnosis. At T3, mothers were diagnosed using the Structured Clinical Interview for *Diagnostic Manual of Mental Disorder and Disease-Fourth Edition* (*DSM–IV*) Axis-I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997), a semistructured, clinician-administered diagnostic interview that includes modules corresponding to major *DSM* psychiatric classifications with high reliability and validity (Zanarini et al., 2000).

Child psychiatric diagnosis. The Developmental and Well-Being Assessment (DAWBA) was used to diagnose child Axis-I disorders at T3 (Goodman, Ford, Richards, Gatward, & Meltzer, 2000). The DAWBA is a well-validated diagnostic tool generating ICD-10 and *DSM–IV* psychiatric diagnoses in 5–17-year-old children, including a large epidemiological study in Israel (Mansbach-Kleinfeld, Apter, Farbstein, Levine, & Ponizovsky, 2010). The DAWBA was administered by clinicians supervised by a child psychiatrist, who were blind to any other information, with reliability >85% and cases conferred every few weeks.

Child behavior problems. In early childhood, mothers completed the Child Behavior Checklist 1.5–5 (Achenbach & Rescorla, 2001), a well-validated maternal report of child's symptoms yielding externalizing, internalizing, and total behavior symptoms scores. In late childhood, mothers completed the Child Behavior Checklist 4–16 years (CBCL; Achenbach & Edelbrock, 1983). The CBCL is a parent self-report measure of child behavior problems that includes 113 items each rated on a 3-point scale ranging from 0 (*never applies*) to 2 (*almost always applies*) and clustered into internalizing, externalizing, and total scores. The CBCL is the most widely used instrument for assessing behavior problems in children with established reliability and validity (Dutra, Campbell, & Westen, 2004).

Saliva sample collection. Saliva was collected from mother and child at both T1 and T3 between 3:00 and 6:00 p.m. Participants were instructed not to eat, drink (besides water), exercise, or smoke before the visit, and medication use was recorded.

In late childhood, mother and child were asked to chew on a roll of cotton for 1 min until it became saturated and then was placed in a Salivette (Sarstedt, Rommelsdorft, Germany). Saliva samples were collected on arrival (baseline), before the dyadic interaction, and 10 min thereafter. Salivates were kept cooled and then stored at −20°C until centrifuged at 4°C at 1,500 g for 20 min. Cortisol levels were then assayed using a commercial ELISA kit (Assay Design, MI). Measurements were performed in duplicate according to the kit's instructions. Cortisol levels were calculated by using MatLab-7 according to relevant standard curves. Intraassay and interassay coefficients were less than 10.5 and 13.4%, respectively. Although it is considered preferable to have an intra and inter assays CVs lower than 10%, this minor difference may have resulted from low concentration in some of the samples (Schultheiss & Stanton, 2009). However, our intra and inter assays CV were within the advised range by the kit's insert (Assay Design, MI).

Consistent with prior research, the three SC measures in both T1 and T3 were computed as area under the curve with respect to the ground (AUCg; Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). This measure is used to estimate total cortisol production over a time-period (Pruessner et al., 2003).

Hair-testing. During the visit at late childhood, hair strands were cut as close as possible to the scalp from a posterior vertex position. Hair samples were stored in an envelope in the dark at room temperature until assayed. We extracted steroids from hair using our published protocol for hair-testing (Koren et al., 2002). Briefly, hair was weighed and placed in a glass vial. Methanol was added and the vials were sonicated for 30 min and then incubated overnight at 50 °C with gentle shaking. The methanol was collected and evaporated under a stream of nitrogen. Samples were reconstituted in 10% methanol and 90% assay diluent that was provided with the commercial enzyme-linked immunosorbent assays (ELISA) according to manufacturer's recommendations.

Quantitation of steroids. Cortisol was quantified in hair extracts using commercial ELISA according to the manufacturer's recommendations (Salimetrics; item no. 1–3002-5; Ann Arbor, MI). Serial dilutions of separate pools for women and children showed parallelism with the provided kit standards (univariate analysis of variance in SPSS; $p = .2$). Linearity was demonstrated for all the weight range examined, between 10 and 140 mg of hair extract, corresponding to1,200–9,000 pg/mL of cortisol standard. The lowest concentration we detected by the assay was 0.4 pg/ml, corresponding to 15.3 mg hair. According to the manufacturer, antibody cross-reactivity was reported as 36.4% with dihydrotestosterone, 21.02% with 19-nortestosterone, 1.9% with 11-hydroxytestosterone, 1.157% with androstenedione, and less than 0.489% for all other steroids. Intraassay repeatability was determined using six duplicates of the pool on the same ELISA plate. The calculated coefficient of variation was 14.56%. Interassay precision was determined by running duplicates of the pool on four different days. The coefficient of variation was 14.65%. Efficiency of 80% was retrieved using exogenous cortisol. HCC values were log-transformed, consistent with prior research (Liu, Snidman, Leonard, Meyer, & Tronick, 2016).

Coding. In early childhood, 10 min of dyadic free play was coded using the Coding Interactive Behavior Manual infancy/preschool version (CIB; Feldman, 1998). In late childhood, mother-child interactions (conflict and positive interaction paradigms) were coded using the CIB adolescent version. The CIB is a well-validated rating system for social interactions in infants, children, adolescents, and adults that includes multiple scales for parent, child, and dyad, aggregated into theoretically derived composites. The system has been used in multiple studies of normative and high-risk populations across cultures and has good psychometric properties, including studies of children at these ages (for review; Feldman, 2012) and studies linking coded behavior with cortisol production (Apter-Levi et al., 2016; Feldman et al., 2013). Coding was conducted by trained coders, blind to any other information, and reliability on 20% of the interactions exceeded 93 and 90% on all codes ($k = .84$, range .78–95 and $k = .82$, range = .78–.96), in early and late childhood, respectively. Two constructs were used: mother–child synchrony and child social engagement.

Mother-child synchrony. Addressed the goodness-of-fit between maternal and child's behavior and their mutual adaptation to each other's verbal and nonverbal signals. It includes the codes of affect synchrony, mutual adaptation, and fluency. In late childhood it also includes recognition of partner's signals, containment, behavioral empathy, and giving space ($\alpha = .92$, .91 at T1 and T3).

Child social engagement. Represented the child's active involvement and initiation of social behavior and included codes related to child's initiation, positive affect, vocal output, trust, and creativity, and social focus ($\alpha = .90$, .91 in T1 and T3).

Statistical Analysis

Chi-square and t tests were conducted to examine differences between the exposed and control groups on study variables. Pearson correlations were computed for a full matrix of correlations among study variables at T1 and T3. To provide a comprehensive model on the direct and mediated paths from exposure to child symptomatology as mediated by maternal stress physiology (HCC, SC), synchronous behavior, and psychopathology, we conducted structural equation modeling (SEM) with Amos 21.0 (Arbuckle, 2012; Byrne, 2010). This method allows for model-testing by exploring the degree to which the theoretical model can explain the pattern of intercorrelations in a set of variables. It also enabled us to test direct and indirect effects between variables.

To assess change from early to late childhood, we controlled for T1 variables when predicting T3 mediators and dependent variables. Specifically, for T3 maternal synchrony, child social engagement, externalizing and internalizing symptoms we controlled for the respective T1 variables. This control was achieved by modeling a direct path from the T1 variable to T3 variable; thus, these T3 variables can be interpreted as representing change from T1 to T3. We also modeled all the covariances between T1 variables and war exposure to fully account for T1 variables. Furthermore, we modeled the correlation between mother and child's cortisol (HCC, SC) and child symptoms to account for their potential covariation. Assuming a power of .80 to be sufficient, observed statistical power of close fit (root mean square error of approximation [RMSEA] $\leq .05$) was adequate (power = .78) for the models tested in the current study.

To assess model fit the following indices were used: χ^2, Comparative Fit Index (CFI), Tucker–Lewis Index (TLI), and RMSEA. Model fit with CFI, and TLI equal to or greater than .90, RMSEA equal to or less than .06 are indicative of adequate fit to the data (Hu & Bentler, 1999). Ideally, the χ^2 statistic is expected to be nonsignificant in the case of adequate fit; generally, however this index is no longer used to evaluate fit because of its hypersensitivity to sample size (Hu & Bentler, 1999). We used the full information maximum likelihood method to account for missing data and used the maximum likelihood method as the estimator in all analyses. To assess the significance of the mediation effects, we used a procedure recommended by Hayes (2013) and calculated the 95% confidence intervals (CIs) of 5,000 bias-corrected and accelerated bootstrapping analyses. When the value zero is not included in the CI, this indicates a significant statistical effect at $\alpha < .05$.

Results

No differences in demographic variables, including parent's age, education, child age and gender, and marital status, were found between the exposed and control groups (all $ps > 0.05$).

Means, *SD*, and tests of group differences for study variables at T1 and T3 appear in Table 1. Results presented in Table 1 show that at T3 war-exposed children and mothers had higher SC and mothers displayed lower synchrony (see Figure 1). War-exposed children had more externalizing and internalizing symptoms and mothers and children had more Axis-I disorders. Children psychopathologies included, in a descending prevalence: attention-deficit-hyperactivity disorder (ADHD), anxiety disorder, PTSD, Oppositional Defiant Disorder (ODD), conduct disorder, depression, and very few cases of enuresis, encopresis, tic disorder, obstructive sleep disorder, and adjustment disorder. Among mothers, 38.1% had anxiety disorder, 11.9% depression, 7.1% PTSD, 7.1% other disorders, and 35.7% of mothers had two or more of the above disorders. No group differences emerged in maternal or child's HCC.

Pearson correlations among study variables within and between time-points are presented in Table 2. Results indicate that at T3 mothers' HCC correlated with their children's HCC and SC, with lower child social engagement, and with higher internalizing symptoms. Mothers' and children's SC were also interrelated. Synchrony correlated with child social engagement and negatively with externalizing symptoms and child HCC. Child engagement was related to lower child SC and internalizing problems. Maternal psychiatric disorder was related to children's internalizing and externalizing problems. Cross-time correlations showed individual stability in mother–child synchrony, social engagement, externalizing symptoms, and internalizing symptoms from early to late childhood but not in maternal or child SC.

Finally, we used SEM to test our model on the mediating role of maternal hormones, behavior, and psychopathology in the association between war exposure and child externalizing and internalizing symptomatology. The overall model provided good fit to the data; $\chi^2_{(62)} = 78.18$, $p = .080$, RMSEA = .04, CFI = .97, TLI = .95. The final path model is presented in Figure 2 (paths not shown were not significant).

As seen, trauma exposure was positively linked with child externalizing symptoms via three paths. The first involved maternal synchrony and child social engagement as mediators. Specifically, war exposure predicted a decrease in T3 synchrony while controlling for T1 synchrony. Synchrony, in turn, predicted T3 child engagement while accounting for T1 child engagement. The latter was linked with change from T1 to T3 in externalizing symptoms. Test of mediation showed that this indirect path was significant (95% CI = .001, .030). The second pathway involved an indirect effect via SC and child engagement (95% CI = .001, .007). War exposure was linked with higher maternal SC that correlated with higher child SC, which, in turn, was negatively

Table 1
Differences Between War-Exposed and Control Children and Mothers in Early (T1) and Late (T3) Childhood

		Control		Exposure			
	Study variables	*M*	*SD*	*M*	*SD*	t/χ^2	Cohen's d/Φ
	Gender (male)	40.80%		53.50%		$\chi^2_{(1)} = 2.79$	.13
Time 3							
	Mother hair CT log	.39	.70	.59	.57	$t_{(88)} = -1.48$, $p > .05$	.32
	Child hair CT log	.46	.72	.47	.78	$t_{(88)} = -.1$, $p > .05$	.02
	Mother salivary CT	29,630.84	20,329.01	41,090.47	37,324.13	$t_{(115)} = -1.96$, $p < .05$	.38
	Child salivary CT	17,808.57	13,948.81	26,385.52	31,444.68	$t_{(115)} = -1.78$, $p = .08$	.35
	Maternal synchrony	4.05	.61	3.84	.54	$t_{(162)} = 2.43$, $p < .05$	.38
	Child engagement	4.24	.74	4.12	.67	$t_{(171)} = 1.12$, $p > .05$	.17
	CBCL externalization	4.11	4.73	6.02	7.22	$t_{(172)} = -1.98$, $p < .05$	.31
	CBCL internalizing	3.85	3.74	5.98	6.81	$t_{(172)} = -2.43$, $p < .05$	.39
	Mother Axis I Disorder (disorder)	13.20%		36.60%		$\chi^2_{(1)} = 12.23$, $p < .01$	.26
	Child Axis-I Disorder (disorder)	27.60%		55.40%		$\chi^2_{(1)} = 13.65$, $p < .01$	.28
Time 1							
	Mother salivary CT	57,714.02	17,346.45	57,428.30	9,926.74	$t_{(89)} = .1$, $p > .05$	.02
	Child salivary CT	51,605.11	10,770.73	54,396.47	15,685.21	$t_{(160)} = -1.24$, $p > .05$	.21
	Maternal synchrony	4.12	.76	3.93	.68	$t_{(149)} = 1.56$, $p > .05$	.26
	Child engagement	3.76	.75	3.61	.82	$t_{(151)} = 1.12$, $p > .05$	.19
	CBCL externalization	7.74	5.73	8.48	6.92	$t_{(170)} = -.75$, $p > .05$	.12
	CBCL internalizing	4.64	4.72	7.52	8.47	$t_{(170)} = -2.65$, $p < .01$	.42

Note. CBCL = Child Behavior Checklist.

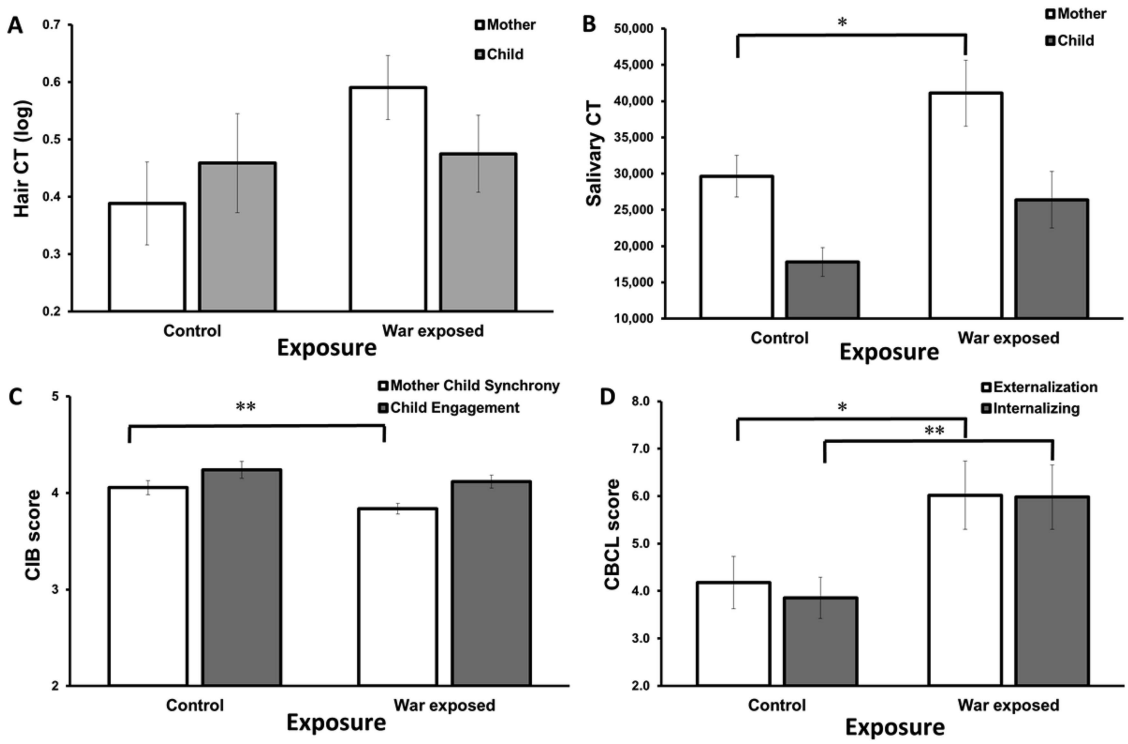

Figure 1. Differences between war exposed and control mothers and children in chronic and phasic cortisol and social behavior and in children's externalizing and internalizing symptoms. * $p < .05$. ** $p < .01$.

related to child engagement and finally to lower externalizing problems. The third indirect pathway related to maternal psychopathology: war exposure linked with higher maternal psychopathology that in turn predicted greater externalizing symptoms (95% CI = .004, .085).

Two additional indirect pathways linked war exposure with T3 internalizing symptoms above and beyond T1 internalizing symptoms. The first indirect path involved HCC (95% CI = .001, .015). War exposure was positively linked with maternal HCC, which predicted higher child HCC associated with more internalizing

Table 2
Pearson Correlations Among Study Variables in Early (T1) and Late (T3) Childhood

Study variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Time 3															
1. Mother hair CT log	1														
2. Child hair CT log	.23*	1													
3. Mother salivary CT	.07	−.01	1												
4. Child salivary CT	.32*	.08	.42**	1											
5. Maternal synchrony	−.31**	−.34**	−.01	−.12	1										
6. Child engagement	−.23*	−.13	.07	−.22*	.67**	1									
7. Mother disorder	−.04	.10	−.01	−.05	.01	.08	1								
8. CBCL externalization	−.04	.04	.01	.04	−.24**	−.17*	.24**	1							
9. CBCL internalizing	−.22*	−.14	.05	.06	−.23**	−.02	.35**	.61**	1						
Time 1															
10. Mother salivary CT	.15	−.16	.00	−.03	−.11	.05	−.02	−.08	−.08	1					
11. Child salivary CT	.03	−.06	.20*	.15	.06	.04	−.02	−.12	−.10	.22*	1				
12. Maternal synchrony	−.20	−.21	.07	−.23*	.48**	.37**	−.03	.02	.02	.00	−.04	1			
13. Child engagement	−.07	−.16	.07	−.13	.23**	.25**	.08	.09	.04	.05	.00	.56**	1		
14. CBCL externalizing	.17	.14	−.03	.00	−.27**	−.19*	.20*	.52**	.36**	−.06	−.01	−.02	.08	1	
15. CBCL internalizing	.15	.09	−.01	.08	−.28**	−.18*	.29**	.50**	.56**	.00	−.04	−.09	.04	.77**	1
16. War exposure	.15*	.14	.20**	.18*	−.18*	−.10	.26**	.14	.17*	−.01	.08	−.10	−.12	.06	.19*

Note. CBCL = Child Behavior Checklist.
* $p < .05$. ** $p < .01$.

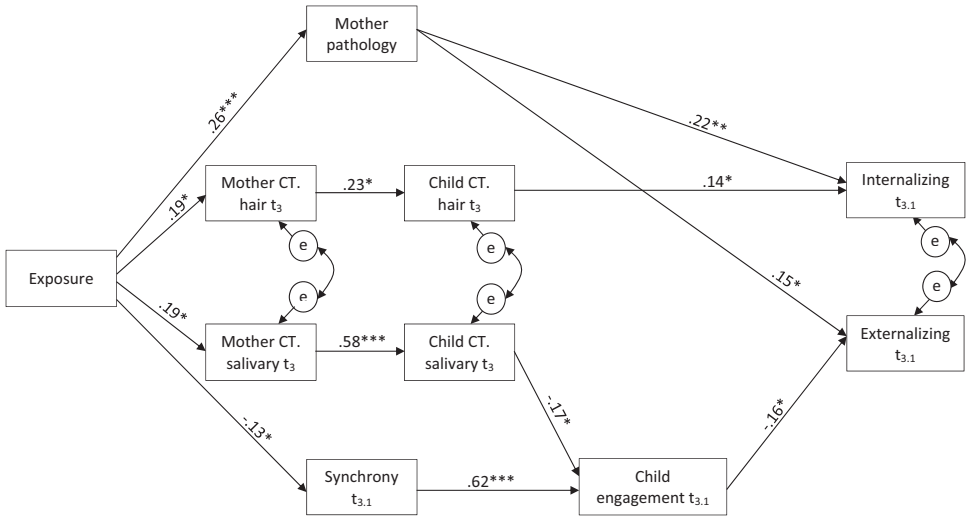

Figure 2. Path model leading from war exposure to child symptoms via three pathways of maternal mediation: Maternal stress hormones, synchronous parenting, and psychiatric disorder. * $p < .05$. ** $p < .01$. *** $p < .001$.

problems. The second indirect path involved maternal psychopathology that was predicted by war and was linked with higher internalizing symptoms (95% CI = .019, .108).

Lastly, to further validate the presented path model we compared it to two alternative models and these are presented in supplementary material. In the first alternative model (Figure 1 in the supplementary material), which proposed the same three maternal mediation pathways, we utilized available T1 variables (maternal and child SC and social behavior) as predictors of T3 externalizing and internalizing symptoms. The only variable charting a significant path in this model was maternal psychopathology at T3 (maternal psychiatric diagnosis was not conducted at T1). The T1 cortisol path and the T1 behavioral path did not transmit the effect of trauma on symptoms. The second alternative model (Figure 2 in the supplementary material) included only child variables to test our proposition on the key role of maternal social influences. In this model, exposure was not directly linked with child chronic and phasic cortisol. Similar to the current model, child HCC was linked with internalizing symptoms and child SC with externalizing symptoms via child engagement. Both models did not provide adequate fit to the data, lending support to the current model.

Discussion

Approximately one-fifth of the world's children are growing up in the context of continuous war, ethnic strife, religious or tribal battles, and regional conflicts; yet, no study to our knowledge has followed war-exposed children over lengthy periods. Our cohort provides a unique "natural experiment" to study the consequences of early adversity by following children living in the same frontline neighborhoods and exposed to the same wartime trauma. Having a distinct, measurable, and similar adversity across participants is a rare condition in research on early life stress and our findings may thus provide novel insights on the specific factors that shape development in risky contexts. Guided by biobehavioral models that highlight the critical role of the mother in humans and animals (Feldman, 2016; Gunnar & Hostinar, 2015), we examined the social transmission of risk and focused on three pathways of maternal social influence. These include maternal HPA-axis, the key stress response system indexed by HCC and SC, synchronous parenting, a maternal style that adapts online to child communications, and maternal psychiatric disorder. Before discussing the findings, it is important to emphasize that the terms "impact" or "effects" used here describe statistical, not causal effects.

Our cohort has been followed for over a decade and several prior publications described findings from this cohort at earlier stages, providing the basis for the current hypotheses. For instance, we found associations between children's posttraumatic symptoms in early childhood with maternal anxiety, depression, and PTSD symptoms and maternal sensitive containment during trauma evocation (Feldman & Vengrober, 2011); links between child salivary cortisol and symptoms in early childhood (Feldman et al., 2013); and correlations between risk alleles on oxytocin- pathway genes and child psychopathology (Feldman et al., 2014). Building on these findings, the current study revisited families in late childhood to address maternal social influences in relation to change trajectories from early to late childhood and symptom formation. We also included here for the first time a full maternal psychiatric diagnosis and a comprehensive assessment of maternal and child's stress response, taping the chronic and phasic aspects of HPA-axis functioning by measuring both SC and HCC.

Several overall insights emerge from our decade-long study. First, while war exposure exerted a direct impact on all maternal pathways and was associated with increased maternal psychopathology, reduced synchrony, and augmented cortisol production, its effects on child hormones and behavior were mediated by the mother, lending support to our social transmission model. The findings that no child biological (HCC, SC) or social (social engagement) factor showed main effect for exposure is of interest and supports the notion that the effects of adversity on children is often indirect; yet such indirect effects are those that over time shape symptom formation through multiple physiological and re-

lational pathways, for instance, by increasing allostatic load of decreasing involvement with the social world. It is therefore important to conduct studies that involve both mother and child and include factors that are directly shaped by adversity as well as those indirectly impacted by it.

A second overall insight relates to change trajectories, a topic receiving nearly no empirical attention in longitudinal studies. As seen, a model charting change over time in maternal and child factors provided a better fit to the data than a model predicting child psychopathology at 10 years from early childhood variables, indicating that the system's end-point is better predicted from change trajectories than from initial conditions, even when considering variables that show stability over time. This is consistent with the dynamic systems' approach to social development (Feldman & Eidelman, 2004) and with conceptual models underscoring the need to focus on risk and resilience trajectories in contexts of early adversity utilizing longitudinal studies (Masten, 2001). More important, while maternal and child's social behavior and child internalizing and externalizing symptoms were individually stable and change trajectories of these factors were modeled from early to late childhood, salivary cortisol showed no individual stability. Furthermore, a model predicting symptomatology in late childhood from early childhood cortisol showed no significant effects (supplementary material Table 2). These findings suggest the need for much further research on the development of the multifaceted HPA system across developmental stages and in relation to multiple risk conditions in parents and children.

A third overall insight addresses the direct and indirect mediation described by our model. Whereas maternal psychopathology was directly linked to child symptoms, the impact of maternal hormones and behavior were indirect and described a field of mutual influences between mother and child, biology and behavior unfolding over time as the critical matrix to consider when testing long-term effects of adversity on children (Feldman, 2015a). Maternal psychiatric disorder, comprising in our sample mainly anxiety disorders and PTSD, charted a path from war exposure to child symptoms. This is consistent with a large body of literature indicating that in adverse contexts in general, and war zones in particular, maternal psychopathology directly impacts child symptoms (Scheeringa & Zeanah, 2001; Smith, Perrin, Yule, & Rabe-Hesketh, 2001; Thabet, Abu Tawahina, El Sarraj, & Vostanis, 2008). However, we found that this well-documented association initiated no further paths related to child hormones or social behavior. The pathway linking maternal psychopathology and child symptoms is thought to be mediated, at least partly, by shared genetic risk and the context of ongoing war, to which both mother and child are exposed, may exacerbate this shared disposition via genetic and epigenetic mechanisms (Rutter et al., 2006). It is possible that war exposure contributes to the formation of child symptoms via two global routes. The first involves an increase in the prevalence of maternal psychiatric disorders, which is directly related to elevation in child psychiatric disorders and externalizing and internalizing symptoms; the second operates by initiating a cascade related to the stress response and its chronic and phasic aspects and the mother's online attunement to the child's signals, which lead to alterations in children's engagement with the social world, culminating in elevated symptoms.

Maternal and child's HPA-axis functioning, as expressed in both SC and HCC, showed cortisol linkage, the biological coupling of maternal and child's CT production. Biobehavioral synchrony, the coupling of maternal and child's physiological and behavior responses, is a key feature of mammalian development; mammalian young are born immature and their brain and neuroendocrine systems develop through the external regulation of maternal care and physiological systems (Hofer, 1995). Extant research in humans and animal models indicates that children's HPA-axis matures in the context of the mother's glucocorticoid system, both in utero and during early life (Seckl & Meaney, 2004; Van Den Bergh et al., 2005) and the stress response is among the least canalized and most open to maternal social influences (Talge, Neal, & Glover, 2007; Weinstock, 2008). Cortisol linkage has been shown in infants, children, and adolescents (Atkinson, Jamieson, Khoury, Ludmer, & Gonzalez, 2016; Hibel et al., 2015; Ruttle et al., 2011) and is considered a survival-related mechanism by which mothers signal the degree of environmental danger to her offspring. Here we show that the effects of war on children's HPA-axis functioning is mediated by the mother's cortisol, suggesting that maternal stress physiology may adapt the child's stress-sensitive systems, augmenting or attenuating children's HPA-reactivity to chronic stress.

An interesting find was that two pathways emerged between war exposure and child symptoms via HPA-axis activity; the chronic pathway and the phasic pathway. As to the chronic pathway indexed by HCC, our model charts a path leading from war exposure to an increase in maternal HCC, which was linked to increased child HCC and culminated in higher internalizing symptoms. The impact of war on child psychopathology via chronic HPA-axis functionality may thus tap a maternal social influence via mechanisms of cortisol linkage: when maternal HCC was not entered as a mediating variable, the model was not significant. However, this pathway included only maternal and child hormones and did not require further mediation by hormone-behavior links. These findings are the first to address maternal and child HCC in the context of war exposure and underscore the potential utility of HCC as a unique stress biomarker in conditions of early life stress. Consistent with prior research (Short et al., 2016; Flom et al., 2017), we found no correlations between HCC and SC in either mother or child, suggesting that the two measures index distinct aspects of HPA-axis functionality and including both in future research may provide a more comprehensive assessment of the stress response.

The phasic pathway, linking maternal and child SC to externalizing symptoms, indicated that both maternal synchronous parenting and the child's SC converged on child social engagement as the pathway leading to lower externalizing symptoms. Social engagement is an important factor in the context of early adversity and a greater tendency to engage enables children to elicit caregiving from kin or nonkin adults when parental resources are limited. The child's socially engaged orientation is biologically based, yet it is longitudinally shaped by sensitive and synchronous parenting and shows individual stability over time and across relationships with mother, father, and close friends (Feldman, 2010; Feldman, Gordon, et al., 2013; Marshal & Fox, 2006). Social engagement in childhood has been shown to predict greater competence in the peer group, better emotion regulation and socialization, and lower psychopathology (Feldman & Masalha, 2010; Marshal & Fox, 2006). Social withdrawal, on the other hand is associated with dysfunctions in children's HPA-axis reactivity,

including flat curves (Apter-Levi et al., 2016) or augmented cortisol reactivity (Feldman et al., 2009). In the context of war, flat and inflexible cortisol curves have been associated in this cohort with limited child ability to seek proximity and with greater risk for PTSD in early childhood (Feldman et al., 2013). The current findings similarly highlight the mediating role of social engagement and may suggest that interventions to increase children's social skills or peer-group involvement may be helpful in contexts of adversity.

From a theoretical standpoint, Bowlby's early formulations on attachment, separation, and loss (Bowlby, 1969)—similarly conceptualized on the basis of his experience with war-exposed children during WWII—emphasized that disengagement, withdrawal, and the shut-down of human contact mark the most pathological response to war-related trauma and define a behavioral constellation not easily reversed. Hofer (1995), testing these ideas in animal models, showed that behavioral withdrawal pinpoints a significant and often chronic risk condition and described its system-specific neuroendocrine markers. Our findings similarly suggest that war exposure may lead, partly because of pathological physiological stress, to social disengagement orientation that fosters symptoms formation. The convergence of maternal physiology and behavior on child engagement as the leading pathway to psychopathology highlights the importance of the child's social competence in adverse contexts. This is particularly critical during late childhood, a period when children shift their investment from the parent–child relationship to the social group and social skills become a key factor in shaping risk and resilience.

Our longitudinal, maternal-focused model was tested against two alternatives, both of which provided less adequate fit; a model assuming direct effects of war on children without considering maternal influences and a model predicting symptoms in late childhood from variables measured in early childhood, not from change trajectories. As to the first comparison, our findings clearly show that the maternal social influences on children symptom formation in the context of war are substantial and multidimensional. To some degree, our data suggest that mothers stand between war and the child and when mothers are more resilient and are able to maintain psychological health, keep attuned parenting, and exhibit lower stress reactivity, they may provide a protective shield for their children. As to the second comparison, our findings corroborate theoretical perspectives that highlight the need to follow children longitudinally and chart trajectories of risk and resilience in contexts of early adversity, particularly among children reared within ongoing political violence (Hobfoll et al., 2009). When the environment is chronically stressed, it is crucially important to define biobehavioral factors in mother and child that enable some children who exhibit greater initial risk to recover or to detect factors that show "sleeper" effects and play a greater role as children grow (Masten, 2001). Thus, while very few studies undertake biobehavioral follow ups of high-risk children over lengthy periods, our findings suggest that it may be useful to focus on the dynamics of change and address factors such as malleability, resilience after initial risk, or developmental regression.

Our findings have several implications for intervention. First, the fact that over 55% of war-exposed children exhibited a full-blown Axis-I disorder at 10 years should raise concern. Such findings indicate that children do not get used to living in the shadow of war and early symptoms do not resolve naturally as children grow. It should also alerts clinicians, researchers, and social policymakers to the fact that millions of the world's future citizens may be developing chronic psychopathologies and those may bear a critical impact on society. Second, our data highlight the pervasive impact of the mother's physiology, behavior, and mental health on children's well-being and suggest that interventions to assist mothers in areas of ongoing violence need to be devised and implemented. Finally, the critical role of child social engagement in mediating the links between stress physiology and parenting with symptom formation suggests that school-age children in harsh contexts may benefit from interventions specifically targeted to enhance social skills, form positive social groups, and maintain an involved social orientation as a pathway to resilience.

Finally, several study limitations should be noted. First, on account of practical constraints, only mothers were examined in the current study. As both parents shape their children's well-being, the inclusion of fathers' stress physiology and behavior could have provided a more comprehensive understanding and should be included in subsequent research. Second, future studies would benefit from exploring the potential stress-buffering effect of other close relationships, such as siblings, peers, or grandparents and the differential influence of these attachments on HPA-functioning. Third, since hair measurement is a new technology, no HCC assessment was available in early childhood, precluding a full matrix of correlations across time. Another limitation of the current study is that the children's internalizing and externalizing behavior were only reported by the parent. In the next stages of this study we intend to include child-report measures. Maternal prenatal stress can shape the development of children's HPA-axis via fetal programming (Seckl & Meaney, 2004; Van Den Bergh et al., 2005). As mothers from the study group were residing in Sderot while pregnant, prenatal stress may have affected fetal development of their children through several mechanisms, and this may have influenced the etiological pathways of our results. Finally, because of the unique characteristics of the current context, generalizability of the findings to other war-exposed regions or chronically stressful contexts should be scrutinized, especially when attempting to extrapolate them on extremely violent war zones, which have suffered severe bloodshed and numerous fatalities, or on remote or economically undeveloped locations.

As the current study opens an important avenue of research on the relationships between war-exposed mothers' and children's behavior, psychopathology, and stable and reactive stress physiology, future research is needed to fully understand the neurobiology of stress, including stress genes and stress-related neural networks. Furthermore, examining additional stress-responses mediated by the autonomic nervous system, such as heart rate, blood pressure, and skin conductance measures, and exploring their influence on the child's immune system, is essential for developing a comprehensive model that integrates biological, behavioral, and social levels of analysis to devise more effective interventions for war-exposed children and their families.

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