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# Risk and resilience trajectories in war-exposed children across the first decade of life

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Background: Although the effects of early-onset trauma on susceptibility to psychopathology are well-acknowledged, no study to date has followed risk and resilience trajectories in war-exposed young children over lengthy periods and charted predictors of individual pathways. Method: In this prospective longitudinal study, we followed 232 children, including 148 exposed to repeated wartime trauma and 84 controls, at three time points: early childhood (1.5-5 years), middle childhood (5-8 years), and late childhood (9-11 years). Children were diagnosed at each time point and four trajectories defined: children exhibiting no pathology at any time point, those displaying early pathology that later remitted, those showing initial resilience followed by late pathology, and children presenting chronic pathology across the entire first decade. Maternal behavioral containment during trauma evocation and child social engagement during free play were observed in early childhood and maternal emotional distress self-reported across time. Results: War-exposed children showed significantly higher rates of psychopathology, with 81% exhibiting pathology at some point during childhood. In middle childhood, exposed children displayed more posttraumatic stress disorders (PTSD), anxiety disorders, and attention-deficit/hyperactivity disorders (ADHD), and in late childhood more PTSD, conduct/oppositional defiant disorders, and ADHD. War-exposed children had more comorbid psychopathologies and number of comorbidities increased with age. Notably, war-exposure increased prevalence of chronic pathology by 24-fold. Maternal factors, including mother's uncontained style and emotional distress, increased risk for early and chronic psychopathology, whereas reduced child social engagement augmented risk for late pathology. Conclusions: Early-onset chronic stress does not heal naturally, and its effects appear to exacerbate over time, with trauma-exposed children presenting a more comorbid, chronic, and externalizing profile as they grow older. Our findings demonstrate that responses to trauma are dynamic and variable and pinpoint agespecific effects of maternal and child factors on risk and resilience trajectories. Results highlight the importance of conducting long-term follow-up studies and constructing individually tailored early interventions following trauma exposure. Keywords: Trauma; early life stress; longitudinal studies; social engagement; childhood psychopathology.

#### Introduction

Evidence indicates that chronic exposure to traumatic experiences - defined as events involving actual or threatened death or serious injury to the physical or psychological integrity of the self or close others which cause significant distress, horror, or helplessness (American Psychiatric Association (APA), 2000) - bears long-term negative consequences for children's social, emotional, academic, and physical growth (Fairbank & Fairbank, 2009; Yule, 2001). Yet, whereas the pervasive impact of trauma on children and adolescents has been studied for over 30 years (Terr, 1983), only recently have researchers begun to address the detrimental effects of trauma on infants and young children. Within this emerging body of research, studies have examined young children's response to natural disasters (Scheeringa & Zeanah, 2008), motor vehicle accidents (Meiser-Stedman, Smith, Glucksman, Yule, & Dalgleish, 2008), or domestic violence, abuse, and maltreatment (Hulette et al., 2008; Levendosky, Huth-Bocks, Semel, & Shapiro, 2002); however, war exposure, while affecting millions of young children across the globe, has received very little

empirical attention. Authors have suggested that traumatic events may induce particularly high stress in young children, placing them at a greater risk for adverse psychological outcomes (Chu & Lieberman, 2010; Feldman & Vengrober, 2011). Furthermore, consistent with the notion of 'stress' as first coined by Selye (1956), which describes how prolonged exposure to stress-inducing conditions permanently alters the body's stress response, early and chronic life stress often carries lifetime effects on physical and mental health (Slone & Mann, 2016).

Assessment of posttraumatic stress in young children is especially challenging due to the young child's limited verbal skills and complete dependence on the caregiver for physical and emotional protection, making the differentiation of child 'own' disorder from disordered mother-child relationship difficult and rendering observations of mother-child interactions in both stressful and natural contexts a necessary component of the diagnosis (Chu & Lieberman, 2010; Lieberman, 2004). While several studies have focused on developing assessment tools for posttraumatic stress disorders (PTSD) in young children, very little research has followed traumaexposed young children longitudinally. The few existing longitudinal studies demonstrate that young children who developed PTSD after traumatic

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experiences do not simply 'grow out' of their stress-related symptoms and present natural recovery but show less remission with time compared to older children and adults (Meiser-Stedman et al., 2008; Scheeringa, Zeanah, Myers, & Putnam, 2005). Moreover, rates of psychopathology markedly increase when repeated, lengthy, and potentially lethal events are experienced during the first years of life (Feldman, Vengrober, & Ebstein, 2014; Pat-Horenczyk et al., 2013). These findings highlight the need to detail the developmental course of young children exposed to repeated trauma and define maternal and child factors that may chart individual trajectories of risk and resilience.

# Chronic exposure to trauma in early childhood and trajectories of risk and resilience

Continuous exposure to traumatic events carries a cumulative impact that may worsen the severity of posttraumatic symptoms (Pat-Horenczyk et al., 2013) and lead to functional impairment and diminished health throughout life (Danese & McEwen, 2012). Prolonged exposure to trauma in early childhood, including domestic violence, neglect, or political violence and terrorism, creates a shadow of threat, anxiety, and uncertainty that compromises the child's sense of well-being (Yule, 2001). Furthermore, the effect of chronic trauma in early childhood appears to intensify over time as it depletes the child's resources from investing in age-appropriate tasks (Hobfoll et al., 2009).

The current rise in global terrorism has become a worldwide affliction, exposing millions of young children to continuous trauma and unpredictable violence whose long-term effects on the citizens of tomorrow's world are still unknown. In this study, we recruited a cohort of children living in Sderot, a small Israeli town located 10 km from the Gaza border which has been the target of repeated, unpredictable missile attacks for over 15 years. We followed families for 10 years, observing children at early childhood (1.5-5 years), middle childhood (5-8 years), and late childhood (9-11 years) to chart trajectories of risk and resilience following a continuous and uniform stressor. In contrast to single traumatic events, such as natural disasters or motor vehicle accidents, children of Sderot have been exposed since infancy to rapid and unpredictable fluctuations between periods of relative calm and acute violence. Such conditions resemble, to some extent, the context of domestic violence, which involves constant uncertainty about threat and safety, incessant vigilance, frequent trauma reminders, and inability to maintain a stable routine (Levendosky et al., 2002). Yet, while the quantity, quality, and intensity of traumatic events differ among maltreating families, all children in this study were growing up in the same frontline neighborhoods and exposed to the same wartime

stressors. As such, our prospective longitudinal design affords a unique 'natural experiment' to assess whether the effects of ongoing trauma increase or decline over time and to define predictors of individual pathways. According to Garmezy (1991), longitudinal studies are crucial in the context of continuous stress, providing a unique tool for understanding how the emergence of new skills and vulnerabilities shape individual trajectories. As psychological responses to trauma are highly diverse (Ozer, Best, Lipsey, & Weiss, 2003), we will explore the role of specific maternal and child factors at critical developmental nodes.

Risk and resilience fundamentally reflect developmental processes that unfold over time and depend on both individual and context (Masten, 2011). Developmental theorists argue that adjustment to early adversity is not shaped by discrete factors but reflects a cumulative mix of intraindividual and sociocontextual components (Garmezy, 1991). Hobfoll et al. (2009) describe four trajectories of risk and resilience in the face of chronic trauma that will be tested here. In the first resistance trajectory, individuals never develop symptoms or disorders; in the second resilience trajectory, initial symptoms are followed by recovery; in the third chronic distress trajectory, initially symptomatic individuals remain symptomatic over time, and in the fourth delayed distress trajectory, initial resistance is followed by psychopathology. These four trajectories will guide our study.

# Maternal and child factors and risk and resilience following trauma

Research has consistently emphasized the effects of maternal mental health on children's responses to trauma (Chemtob et al., 2010; Feldman & Vengrober, 2011) and shows that children are affected not only by direct exposure to trauma but also by the mothers' response to it (Chemtob et al., 2010). According to attachment theory, infants seek comfort from their caregivers when alarmed or under threat and their sense of safety and well-being is organized around the availability and responsiveness of the attachment figure to such moments of stress (Bowlby, 1969). Yet, the mother's own emotional distress may prevent her from buffering the child's stress (Van Ee, Kleber, & Mooren, 2012).

Traumatic events may also impact the mother-child relationship, which plays a key role in the interplay of risk and protective factors in the context of early trauma (Scheeringa, Myers, Putnam, & Zeanah, 2015). Scheeringa and Zeanah (2001), proposing a relational trauma framework, described the co-occurrence of posttraumatic distress in mother and child and underscored the importance of the parent-child relationship. Quality of parenting following trauma may be profoundly influenced

by maternal emotional distress, indexed by symptoms of depression, anxiety, and posttrauma. Continuous stress may increase maternal anxiety and fear, thereby diminishing her capacity to contain the child's fears, compromising caregiving, and exacerbating child psychopathology (Feldman et al., 2014). In particular, trauma reminders may subvert the child's sense of security and activate re-experiencing symptoms (Lieberman, 2004). When the same traumatic reminders trigger intense maternal distress, the mother may become preoccupied with her own anxiety and behave in a reenactingfrightening pattern, creating secondary traumatization for the child (Scheeringa & Zeanah, 2001). A fearful-frightening maternal style creates a paradox where the person from whom protection is sought is also the one that increases fear (Main & Hesse, 1990). It is, thus, important to examine both maternal emotional distress and behavioral containment of the child's fear during trauma reminders as predictors of risk trajectories.

Ecological models indicate that child temperamental factors serve as sources of risk and resilience, particularly when exposed to chronic trauma (Feldman, Vengrober, Eidelman-Rothman, & Zagoory-Sharon, 2013). Of particular interest in the present context is child social engagement, defined as active involvement in social interactions and tendency to initiate social behavior (Marshal & Fox, 2006). Child engagement during social interactions depends on both temperamental dispositions (Porges, 2003) and parental facilitation of child social participation (Feldman, Greenbaum, Mayes, & Erlich, 1997). Moreover, child social engagement plays a role in the child's ability to benefit from social contacts and create a social support network, thereby shaping trajectories of risk and resilience.

#### The current study

This study utilized a unique cohort followed over 10 years to test maternal and child predictors of risk and resilience following chronic exposure to warrelated trauma. In early childhood, children's PTSD was assessed, and in middle and later childhood, children's psychopathology was diagnosed in order to chart the four trajectories detailed by Hobfoll et al. (2009). Two maternal factors were examined: maternal symptomatology, including mothers' depression, anxiety, and posttraumatic symptoms, and the mother's ability to contain the child's fear during the evocation of trauma reminders. In addition, child social engagement during mother–child interaction was observed.

Three main hypotheses were formulated. First, repeated trauma exposure would markedly increase the prevalence of child psychopathology, including chronic, remitted, and late child psychopathology. We also expected greater comorbidity of psychiatric

disorders in war-exposed children compared with controls. Second, mothers in the exposed group would report greater emotional distress and would display less containment of the child's distress during trauma evocation. Finally, war-exposed children were expected to show lower social engagement during mother-child interactions. We expected maternal factors to be more crucial in early child-hood and to predict early remitted or chronic pathology, whereas child social engagement was expected to play a greater role as children mature and to predict children's later-emerging psychopathology.

#### Methods

# **Participants**

Participants were recruited in two groups and were followed across the first decade.

T1: early childhood. In order to observe children in the toddler and preschool years, participants at T1 included 232 children aged 1.5-5 years (M = 2.76 years, SD = 0.91), including 47.6% males and 47.1% firstborns, and their parents. The war-exposed group included 148 families living in the same neighborhoods in Sderot, Israel, located 10 km from the Gaza border. Individuals in Sderot have been exposed to continuous rocket attacks, 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 non-exposed families from comparable towns in the greater Tel-Aviv area. 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 forms of trauma. The study was approved by the University's Institutional Review Board, and all parents signed informed consent.

 $T2: middle\ childhood.$  We re-examined 210 of the original sample at middle childhood (5–8 years old; M=7.68 years, SD=0.7) to observe children during the first period of school entry, a period of brain re-organization accompanied by the emergence of complex cognitive skills and greater social engagement.

*T3:* late childhood. Of the initial samples, 177 families were re-visited when children were 9–11 years old (M=9.3 years, SD=1.41) to observe children in late childhood prior to the transition to adolescence.

Attrition was mainly related to inability to locate families or families moving out of Sderot. No differences were found between continuing or non-continuing families.

# Procedure

T1: early childhood. Families were visited at home for about 3.5 hr. Mothers were interviewed by trained clinicians to diagnose child PTSD. Clinicians elicited detailed description of the traumatic events, the child's specific posttraumatic symptoms, and developmental regression or arrest. The child was present in the room and situated near the mother when trauma-related information was elicited. This enabled the observation of child behavior during trauma evocation as part of the diagnosis, as recommended for PTSD diagnosis in young children (Chu & Lieberman, 2010). Control mothers were similarly interviewed about recent trauma/difficulty in the child's life (e.g. death of grandparent). Following, mother-child

interactions were videotaped for 10 min followed by several interactive and emotion-regulation paradigms. Finally, mothers completed self-report measures (additional details appear in Supporting information).

*T2:* middle childhood. Child psychopathology was reevaluated using the Developmental and Well-Being Assessment (DAWBA).

T3: late childhood. Visits included diagnosis, mother-child interactions, and maternal self-report measures.

#### Measures

The following measures were used here:

- Early Childhood PTSD: Child PTSD was evaluated at time 1 through maternal interview and direct observations using the Diagnostic Classification: Zero-to-Three (DC 0-3R; Zero to Three, 2005), a validated system using DSM-IV criteria for diagnosing psychiatric disorders in children under 5.
- 2. Child Psychiatric Diagnosis: The developmental and Well-Being Assessment was used to diagnose child Axis-I disorders at T2 and T3. The DAWBA is a structured interview generating ICD-10 and DSM-IV psychiatric diagnoses in 5- to 17-year-old children (Goodman, Ford, Richards, Gatward, & Meltzer, 2000). The DAWBA, administered to mothers, is well-validated, including a large epidemiological study in Israel (Mansbach-Kleinfeld, Apter, Farbstein, Levine, & Ponizovsky, 2010). The DAWBA was administered by clinicians and supervised by child psychiatrist, blind to any other information, with reliability > 85% and cases conferred every few weeks.
- 3. Maternal Emotional Distress: The mother's emotional distress was calculated as the mean z-score at T1 and T3 for each disorder (depression, anxiety, PTSD) separately, which were then averaged together. Depression was assessed with the Beck Depression Inventory (Beck, 1978), a 21-item self-report inventory of depressive symptoms. Anxiety was evaluated with the well-validated State-Trait Anxiety (STAIT/STAIS; Spielberger, Gorsuch, & Lushene, 1970), assessing transitory and stable anxiety. Maternal PTSD symptoms were assessed with the Post-Traumatic Diagnostic Scale (Foa, 1995), a well-validated tool assessing posttraumatic symptoms.

#### Coding

Mother–child interactions and maternal behavior during trauma evocation were coded using the Coding Interactive Behavior Manual (CIB; Feldman, 1998), a well-validated rating system for social interactions. The CIB includes multiple codes for mother, child, and dyad, aggregated into theoretically derived composites. The system has been used in multiple longitudinal studies of normative and high-risk populations across various cultures and has good psychometric properties (for review; Feldman, 2012). Coding was conducted by trained coders, blind to any other information, and reliability on 20% interactions exceeded 90% on all codes (k = .82, range = .74–.95). Two constructs were used here: maternal uncontained style during trauma evocation and child social engagement during free play.

Maternal uncontained style during trauma evocation ( $\alpha=.82$ ) describes the mother's anxiety-provoking, fearful, or frightening behaviors during trauma evocation and included codes assessing maternal overwhelming/frightening behavior, uncontained fear expressed in verbal or non-verbal behaviors,

maternal intrusion on child activity, maternal interruption of child's communications, mother's emotional overload, and inconsistent/irrational behavior.

Child social engagement during free play ( $\alpha$  = .80) included the following codes: child gaze/joint attention, positive affect, alertness, social initiation, creative/symbolic play, and competent use of environment. Child engagement was found to be individually stable across childhood (Feldman, 2010).

#### Statistical analyses

We first examined differences between exposed and non-exposed groups in demographic condition, maternal and child behavior, and child psychopathology using *t*-tests, chi-square and McNemar's test, Mann–Whitney *U*, and Pearson correlations. Next, we compared children's pathological trajectories using chi-square and ANOVAs. Finally, hierarchical multinomial logistic regression was applied to identify predictors of each trajectory, and Cox & Snell, Nagelkerke, and McFadden statistics were used to estimate effect sizes (Field, 2013; Tabachnick & Fidell, 2007).

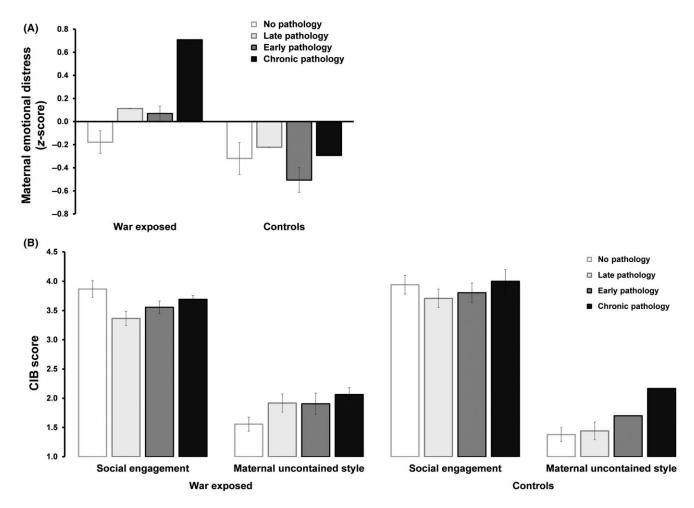
### Results

Mother and child's interactive behavior, maternal emotional distress, and child psychopathology in war-exposed and control families

Before conducting our analyses, we examined demographic differences between the exposed and control groups. No differences were found in parent's age or education, child gender, and marital status (p > .05). As to maternal and child's interactive behavior, exposed children were significantly less engaged during interactions t(175) = -2.31, p > .05. Mothers in the exposed group showed less containment during trauma evocation, t(175) = 4.12, p > .01, and were more emotionally distressed t(175) = 5.65, p > .01 (Figure 1).

Following, we examined child psychopathology across the three time points. During early childhood, 37.8% of war-exposed children developed a full-blown PTSD (compared to none among controls, who were screened for PTSD; Feldman & Vengrober, 2011).

We next tested group differences in the type of pathology children exhibited (Figure 2). In middle childhood, war-exposed children exhibited significantly more PTSD,  $\chi^2 = 17.39$ , p < .01, anxiety disorders,  $\chi^2 = 9.59$ , p < .01, and less attention-deficit/ hyperactivity disorders (ADHD),  $\chi^2 = 4.25$ , p < .05. In late childhood, war-exposed children showed more PTSD,  $\chi^2 = 14.15$ , p < .01, oppositional defiant disorders (ODD) or conduct disorder,  $\chi^2 = 4.35$ , p < .05, and ADHD,  $\chi^2 = 7.32$ , p < .01. Using McNemar's test for paired nominal data, we measured changes in prevalence of disorders from middle to late childhood in each group. Among war-exposed children, a significant increase was found in prevalence of ODD or conduct disorder (from 0.99% to 10.89%, p < .01) and ADHD (from 2.97 to 28.71, p < .01). However, no change over time in prevalence of these disorders was found among controls.



**Figure 1** Maternal and child factor in war-exposed and control children of the four risk and resilience trajectories. Maternal emotional distress was calculated by the mean z-score of maternal depression (BDI), anxiety (STAIT/STAIS), and PTSD symptoms (PDS) at T1 and T3. Maternal uncontained style and social engagement were coded using Coding Interactive Behavior (CIB) and range from 1 to 5

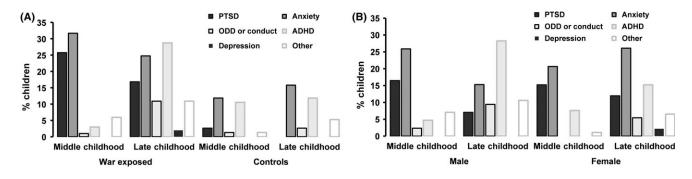


Figure 2 Distributions of psychopathologies by exposed group and gender for children in middle and late childhood. 'Other' category includes enuresis, encopresis, tic disorder, obstructive sleep disorder, and adjustment disorder

In the next step, we used Mann–Whitney U test to examine differences in the number of comorbid psychiatric disorders children exhibited during middle and late childhood. War-exposed children had significantly more comorbid disorders in both middle childhood, z = -4.1, p < .01, and late childhood, z = -4.03, p < .01. Further investigation using Wilcoxon signed-rank test showed significant increase in number of comorbidities only for exposed children, z = -2.24, p < .01, but not for controls, z = -0.86, p > .05 (Figure 3).

Finally, using Pearson's correlations, we tested homotypic versus heterotypic continuity (i.e. continuity in same vs. different disorders) in children's psychopathology. PTSD in middle childhood was associated with PTSD ( $r=.23,\ p<.01$ ) and ADHD ( $r=.15,\ p<.05$ ) in late childhood. Anxiety in middle childhood was related to ODD or conduct disorder ( $r=.15,\ p<.05$ ), ADHD ( $r=.17,\ p<.05$ ), depression ( $r=.2,\ p<.01$ ), and other disorders ( $r=.17,\ p<.05$ ) in late childhood. Additionally, ODD or conduct disorder in middle childhood was associated

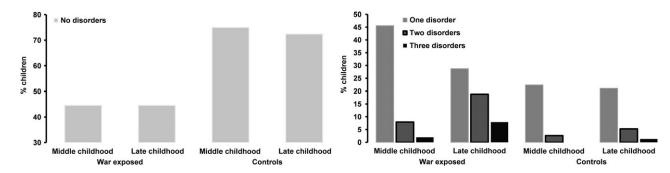


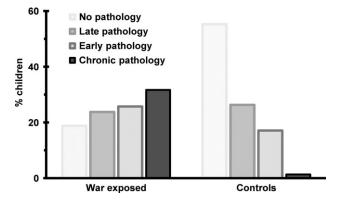
Figure 3 Percentages of comorbidities in war-exposed and control children. Comorbidity was calculated as the sum of being diagnosed with either PTSD, ODD, or conduct, depression, anxiety, ADHD or other according to DC 0–3R and Developmental and Well-Being Assessment (DAWBA)

with ODD or conduct disorder (r = .38, p < .01) and ADHD (r = .2, p < .01) in late childhood. Lastly, ADHD in middle childhood was correlated with ODD or conduct disorder (r = .2, p < .01) in late childhood.

Assessing correlations for each gender separately, we find that in males the continuity was mainly homotypic (ODD/conduct disorder and ADHD in middle childhood correlated with ODD/conduct and ADHD in late childhood, respectively). However, in females continuity was mainly heterotypic (PTSD and anxiety in middle childhood correlated with ODD/conduct disorder, ADHD, and depression in late childhood; see Supporting Information for gender differences and tables).

# Trajectories of risk and resilience in war-exposed and control children

Children were divided into four trajectories: no psychopathology – children without psychopathology at any assessment; early psychopathology – children diagnosed at T1 or T1 + T2 who remitted at T3; late psychopathology – children without diagnosis at T1 or T1 + T2 but diagnosed at T3; and chronic



**Figure 4** Distribution of psychopathology trajectories in warexposed and control children. No psychopathology – children without psychopathology at any assessment; early psychopathology – children diagnosed at T1 or T1 + T2 who remitted at T3; late psychopathology – children without diagnosis at T1 or T1 + T2 but diagnosed at T3; chronic psychopathology – children diagnosed at all three time points

psychopathology – children diagnosed at all three time points. Significant differences in the distributions of these trajectories emerged between exposed and control children,  $\chi^2 = 39.75$ , p < .01 (Figure 4). Importantly, while 55.26% of controls showed no pathology at any time point, only 18.81% of warexposed children were in the no pathology trajectory.

Subsequently, we used one-way ANOVA with Dunnett's post hoc test to compare each pathology trajectory to the 'no pathology' trajectory in the exposed control groups and test differences in maternal and child behavior and mother emotional distress. Among war-exposed children, main effects emerged for mother emotional distress, F(3, 97) = 7.44, p < .01,  $\eta_p^2 = .19$ , and child social engagement F(3, 97) = 2.17, p = .09,  $\eta_p^2 = .06$ . Dunnett's post hoc test revealed that, as compared to 'no pathology', children in the 'late pathology' trajectory were less socially engaged (p < .05). Additionally, mothers in the 'chronic pathology' trajectory were more distressed (p < .01) and showed less containment during trauma evocation at a marginally significant level (p = .06).

# Predicting trajectories of risk and resilience

Hierarchical multinomial logistic regression examined predictors of the three pathology trajectories (chronic, early, and late pathology) as compared with the 'no pathology' trajectory for both groups (Table 1). Five variables were entered in four steps to assess the likelihood of belonging to each pathology trajectory. The first step included exposure group and gender to control for these variables. As seen, war exposure significantly increased the likelihood of having pathology at any stage. The second step included children's social engagement; this factor significantly increased chances of developing 'late pathology', OR = 1.5, p < .01. The third step included maternal uncontained style and this factor significantly increased the likelihood of developing 'early pathology', OR = 1.74, p < .05. As for chronic pathology, maternal uncontained style during trauma evocation predicted chronic pathology in the third step, OR = 2.29, p < .01, and when maternal emotional distress was entered in the fourth step and was found

 Table 1
 Multinomial logistic regression comparing psychopathology trajectories with relative to 'no pathology

Step         Variable         OR         P-Value         Lower bound         Upper         Lower         Upper				Lati	Late pathology			Early p	Early pathology			Chronic pathology	athology				
Exposure (exposed)         Co.5         -Value         Lower bound         Lower bound         Lower bound         bound bound <t< th=""><th></th><th></th><th></th><th></th><th>95% Confid</th><th>lence</th><th></th><th></th><th>95% Cor</th><th>ıfidence</th><th></th><th></th><th>95% Co<sub>1</sub></th><th>nfidence</th><th></th><th></th><th></th></t<>					95% Confid	lence			95% Cor	ıfidence			95% Co <sub>1</sub>	nfidence			
Exposure (exposed)         2.65         .02         1.18         5.93         4.29         .00         1.81         10.15         67.67         .00         8.58         533.55         0.23         0.26           Gender (male)         1.02         .96         0.46         2.28         1.51         .34         0.65         3.53         1.82         .23         0.69         4.82         0.29         6.78         0.06         8.43         528.75         0.29         0.30           Gender (male)         0.26         .07         0.38         1.36         .49         0.57         3.20         1.72         .28         0.64         4.59         0.30           Gender (male)         0.86         .73         0.39         0.67         1.18         0.97         .93         0.50         1.88         0.30         0.30         0.30         0.31         0.89         1.36         0.77         3.23         1.39         0.67         3.23         1.39         0.73         1.39         0.61         3.24         4.43         0.65         2.23         0.73         0.34           Involvement         0.54         .03         0.77         .39         0.77         .39         1.74	Step			p-Value	Lower bound	Upper	OR	p-Value	Lower	Upper	OR	p-Value	Lower	Upper	Cox & Snell	Nagelkerke	McFadden
Gender (male)         1.02         .96         0.46         2.28         1.51         .34         0.65         3.53         1.82         .23         0.69         4.82           Exposed (exposed)         2.26         .05         0.99         5.19         3.92         .00         1.64         9.38         66.78         .00         8.43         528.75         0.28         0.30           Gender (male)         0.86         .73         0.38         1.36         .49         0.57         3.20         1.72         .28         0.64         4.59         0.30         0.30           Involvement         0.50         0.1         0.29         0.87         3.73         0.01         1.32         7.91         52.59         0.0         6.52         424.36         0.32         0.34           Gender (male)         0.88         .76         0.38         1.47         .39         0.61         3.54         4.3         0.55         0.32         0.34           Involvement         0.54         .03         0.31         0.77         .39         0.61         3.58         1.36         .259         0.0         1.34         4.33         0.65         2.77           Maternal uncontain		Exposure (exposed)	2.65	.02	1.18	5.93	4.29	00.	1.81	10.15	67.67	00.	8.58	533.55	0.23	0.26	0.12
Exposed (exposed)         2.26         .05         0.99         5.19         3.92         .00         1.64         9.38         66.78         .00         8.43         528.75         0.28         0.30           Gender (male)         0.86         .73         0.38         1.36         .49         0.57         3.20         1.72         .28         0.64         4.59         0.30         0.31         0.30         0.38         0.50         1.88         0.39         0.30         0.39         0.39         0.50         1.88         0.30         0.34         0.39         0.50         1.88         0.30         0.34         0.39         0.30         0.34         0.39         0.50         1.88         0.32         0.34         0.39         0.61         3.24         2.23         1.3         1.34         4.3         0.50         0.34 <td></td> <td>Gender (male)</td> <td>1.02</td> <td>96.</td> <td>0.46</td> <td>2.28</td> <td>1.51</td> <td>.34</td> <td>0.65</td> <td>3.53</td> <td>1.82</td> <td>.23</td> <td>69.0</td> <td>4.82</td> <td></td> <td></td> <td></td>		Gender (male)	1.02	96.	0.46	2.28	1.51	.34	0.65	3.53	1.82	.23	69.0	4.82			
Gender (male)         0.86         .73         0.38         1.98         1.36         .49         0.57         3.20         1.72         .28         0.64         4.59           Involvement         0.50         .01         0.29         0.87         1.77         3.23         .01         1.32         7.91         52.59         .00         6.52         424.36         0.32         0.34           Exposure (exposed)         2.04         .10         0.87         4.77         3.23         .01         1.32         7.91         52.59         .00         6.52         424.36         0.32         0.34           Gender (male)         0.88         .76         0.38         2.03         1.47         .39         0.61         3.54         2.23         .13         0.79         6.31         0.34           Involvement         0.54         .03         0.77         .39         0.61         3.58         3.43         .04         1.07         10.95           Exposure (exposed)         1.76         .21         0.72         4.27         3.20         .01         1.26         8.09         24.66         .00         2.93         207.84         0.39         0.42           Gender (m	7	Exposed (exposed)	2.26	.05	66.0	5.19	3.92	00.	1.64	9.38	82.99	00.	8.43	528.75	0.28	0.30	0.12
Involvement         0.50         .01         0.29         0.85         0.67         .16         0.38         1.18         0.97         .93         0.50         1.88           Exposure (exposed)         2.04         .10         0.87         4.77         3.23         .01         1.32         7.91         52.59         .00         6.52         424.36         0.32         0.34           Gender (male)         0.88         .76         0.38         2.03         1.47         .39         0.61         3.54         2.23         .13         0.79         6.31         0.34         0.34           Involvement         0.54         .03         0.77         .39         0.43         1.34         .43         0.65         2.77           Maternal uncontained         1.76         .21         0.77         .39         0.61         3.58         3.43         .04         1.07         1.095         0.42           Involvement         0.54         .03         0.40         1.74         .04         1.35         3.43         .04         1.07         10.95           Involvement         0.54         .03         0.77         .39         0.77         .39         0.42         1.35		Gender (male)	0.86	.73	0.38	1.98	1.36	.49	0.57	3.20	1.72	.28	0.64	4.59			
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Gender (male)         0.88         .76         0.38         2.03         1.47         .39         0.61         3.54         2.23         .13         0.79         6.31           Involvement         0.54         .03         0.77         .39         0.43         1.34         .43         0.65         2.77           Maternal uncontained         1.36         .25         0.81         2.29         1.74         .04         1.03         2.92         2.09         .00         1.30         4.04           Exposure (exposed)         1.76         .21         0.72         4.27         3.20         .01         1.26         8.09         24.66         .00         2.93         207.84         0.39         0.42           Gender (male)         0.94         .89         0.40         2.20         1.04         1.07         10.95         10.95           Involvement         0.54         .03         0.77         .39         0.42         1.35         1.45         .35         0.67         3.13           Maternal uncontained         1.27         .37         0.75         2.16         1.70         .05         0.77         3.9         0.45         1.77         .06         0.97 <t< td=""><td>က</td><td>Exposure (exposed)</td><td>2.04</td><td>.10</td><td>0.87</td><td>4.77</td><td>3.23</td><td>.01</td><td>1.32</td><td>7.91</td><td>52.59</td><td>00.</td><td>6.52</td><td>424.36</td><td>0.32</td><td>0.34</td><td>0.14</td></t<>	က	Exposure (exposed)	2.04	.10	0.87	4.77	3.23	.01	1.32	7.91	52.59	00.	6.52	424.36	0.32	0.34	0.14
Involvement         0.54         .03         0.31         0.93         0.77         .39         0.43         1.34         .43         0.65         2.77           Maternal uncontained         1.36         .25         0.81         2.29         1.74         .04         1.03         2.92         2.09         .00         1.30         4.04           Exposure (exposed)         1.76         .21         0.72         4.27         3.20         .01         1.26         8.09         24.66         .00         2.93         207.84         0.39         0.42           Gender (male)         0.94         .89         0.40         2.20         1.48         .39         0.61         3.58         3.43         .04         1.07         10.95           Involvement         0.54         .03         0.31         0.95         0.77         .39         0.42         1.39         1.45         .35         0.67         3.13           Maternal uncontained         1.27         .37         0.75         2.16         1.70         .05         1.01         2.86         1.77         .06         0.97         3.25           Emotional Distress         1.69         .22         0.73         3.88		Gender (male)	0.88	92.	0.38	2.03	1.47	.39	0.61	3.54	2.23	.13	0.79	6.31			
Maternal uncontained         1.36         .25         0.81         2.29         1.74         .04         1.03         2.92         2.29         .00         1.30         4.04           Exposure (exposed)         1.76         .21         0.72         4.27         3.20         .01         1.26         8.09         24.66         .00         2.93         207.84         0.39         0.42           Gender (male)         0.94         .89         0.40         2.20         1.48         .39         0.61         3.58         3.43         .04         1.07         10.95           Involvement         0.54         .03         0.31         0.95         0.77         .39         0.42         1.39         1.45         .35         0.67         3.13           Maternal uncontained         1.27         .37         0.75         2.16         1.70         .05         1.01         2.86         1.77         .06         0.97         3.25           Emotional Distress         1.69         .22         0.73         3.88         1.11         .82         0.45         2.73         5.29         .00         2.02         13.87		Involvement	0.54	.03	0.31	0.93	0.77	.39	0.43	1.39	1.34	.43	0.65	2.77			
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0.54 .03 0.31 0.95 0.77 .39 0.42 1.39 1.45 .35 0.67 1.27 .37 0.75 2.16 1.70 .05 1.01 2.86 1.77 .06 0.97 1.69 .22 0.73 3.88 1.11 .82 0.45 2.73 5.29 .00 2.02		Gender (male)	0.94	68.	0.40	2.20	1.48	.39	0.61	3.58	3.43	.04	1.07	10.95			
1.27     .37     0.75     2.16     1.70     .05     1.01     2.86     1.77     .06     0.97       1.69     .22     0.73     3.88     1.11     .82     0.45     2.73     5.29     .00     2.02		Involvement	0.54	.03	0.31	0.95	0.77	.39	0.42	1.39	1.45	.35	0.67	3.13			
1.69 .22 0.73 3.88 1.11 .82 0.45 2.73 5.29 .00 2.02		Maternal uncontained	1.27	.37	0.75		1.70	.05	1.01	2.86	1.77	90.	0.97	3.25			
		<b>Emotional Distress</b>	1.69	.22	0.73	3.88	1.11	.82	0.45	2.73	5.29	00.	2.02	13.87			

Not exposed' and 'females' were used as reference groups

to be significant, OR = 5.29, p < .01, the contribution of the maternal uncontained style decreased but remained marginally significant, OR = 1.77, p = .06. Notably, exposure increased the likelihood of developing chronic psychopathology by 24-fold, OR = 24.66, p < .01, and boys were three times more likely to develop chronic psychopathology than girls, OR = 3.43, p < .05.

#### Discussion

As millions of the world's children are exposed to continuous war, terrorism, and armed conflict, particularly with the shift of battlefield into the heart of civilian locations, better understanding of their longterm impact on child well-being is essential. This study is the first, to our knowledge, to detail the implications of early exposure to wartime trauma across the first decade of life using both formal diagnosis and observational measures. Our sample provides a rare opportunity by affording a unique 'natural experiment' where all children are exposed to the same chronic stressors, while relational, social, and maternal mental health factors are used to define individual trajectories of psychopathology and resilience. Overall, our findings demonstrate that time does not heal the long-lasting scars of prolonged stress and children growing up amidst armed conflict suffer significant psychopathology and rarely exhibit natural recovery. Yet, whereas some children are more susceptible to develop stress-related symptoms, of a remitted or chronic course, others appear to be more protected. Our findings are unique in pinpointing the interplay of maternal and child factors as they unfold over time and in shaping trajectories of risk and resilience in the context of chronic early stress. Specifically, while maternal factors increased the risk of early-onset psychopathology, child social engagement, which indexes a dispositional as well as relational factor, increased the propensity for late-onset disorders.

Substantial differences emerged between warexposed and control children in the prevalence of psychopathology of any type. Our findings show that over 80% of children exposed to early and chronic trauma are not just symptomatic but display a full-blown Axis-I disorder at some point in their childhood. Moreover, while only 1.3% of controls showed an unremitted psychopathology at all time points, about one-third (31.7%) of warexposed children remained chronically symptomatic for nearly a decade. Such sizable chronicity corresponds to the high incidence of chronic distress reported for war-exposed adults, and the low proportion of children without pathology at any time (18.8%) mirrors the small numbers of resilient warexposed adults (Hobfoll et al., 2009). Possibly, the high rates of psychopathology relate to both the chronic and unpredictable nature of the stress, which may lead to the formation of a 'learned

helplessness' response (Foa, Zinbarg, & Rothbaum, 1992). The findings that children exposed to chronic early stressors were four times more likely to develop a disorder at some point in the first decade than to exhibit resilience should raise concern among policy makers and call for the allocation of more resources to develop early-detection tools and individually targeted interventions.

Two additional aspects of child psychopathology should be considered: type of disorder and number of comorbid pathologies, each of which showed significant differences between exposed and control children. In addition to the high prevalence of PTSD, exposed children exhibited a wide range of psychopathologies, including anxiety disorders, disruptive behaviors, and ADHD. Our data alarmingly indicate that early-onset chronic stress profoundly increases the number of comorbid disorders in middle and late childhood. This additive effect emphasizes the dose-response relationship between the amount and intensity of traumatic exposure and the severity of mental health outcomes (Pine, Costello, & Masten, 2005), underscoring the importance of early interventions to prevent subsequent deterioration. As to gender differences, we found that boys were more susceptible to develop chronic psychopathology and their specific psychopathologies tended to persist over time, as opposed to females for whom continuity of symptoms was more heterotypic, underscoring the less flexible profile of risk for boys. Furthermore, the number of comorbidities increased from middle to late childhood and this rise was found only among war-exposed children, confirming that continuous exposure accumulates over time and depletes the child's inner resources.

Research has shown that symptomatology increases with age and school-aged children are the most vulnerable (Shaw, 2003). Although older children have better cognitive skills to process traumatic experiences, their struggle with autonomy, identity formation, and social rank position places them at greater risk (Kuterovac-Jagodic, 2003). The findings also mirror prior research indicating that externalizing symptoms are more prevalent in school-aged children exposed to war and terrorism, including attention difficulties and behavior problems, which may interfere with their academic achievement and social adjustment (Alkhatib, Regan, & Barrett, 2007). Younger children, in our study as well as prior research, displayed more anxiety disorders, possibly due to their more limited cognitive, social, and emotional abilities that enhance vulnerability to fearfulness and apprehension (Alkhatib et al., 2007; Joshi & O'Donnell, 2003).

Not all children were equally influenced by continuous exposure to stress. In the context of armed conflict, an ecological perspective is particularly important to assess the dynamic integration of personal and contextual parameters across development. The fluid nature of development adds another

dimension of variability, and different sets of predictors were found to correlate with various pathologyonset trajectories. Pathology in early childhood was predicted by maternal response to trauma evocation and the chronic course was shaped by both maternal lack of containment during trauma reminders and mothers' psychopathological symptoms, consistent with perspectives suggesting that maladaptive response to trauma in early childhood is associated with maternal symptoms and dysfunctional behavior (Slone & Mann, 2016). Young children possess less cognitive and emotional resources to cope with danger and rely on the social buffering of caregivers, and their response to trauma resonates with the parent's reaction; thus, traumatized parents' anxious behaviors may exacerbate their children's symptomatology (Scheeringa & Zeanah, 2001). Caught in this vicious cycle of needing parental containment who themselves are traumatized by the same events, young children develop early and often chronic psychopathology that may hamper their lifetime mental health and personal achievement.

The ongoing effects of trauma may become overwhelming as children grow older and some who were more resilient in early childhood may develop pathology at later stages. As age-related developmental tasks become more complex and children begin the transition from the family to the peer group, the range of social competencies required for navigating the social environment increases dramatically and social relationships become more critical to the child's emerging sense of self. Child maladaptive social functioning, including introversion and social problems, defines poorer prognosis for adjustment following trauma (Joshi & O'Donnell, 2003), possibly due to its effect on limiting the ability to use social relationships to manage stress. Children's social engagement likely indexes a temperamental disposition, but also reflects the parent's inability to draw the child into mutual and rewarding social exchanges beginning in infancy. This may lead to the child's diminished trust in social relationships and to an inability to seek social support, thus increasing the propensity for psychopathology in later childhood. These findings echo Garmazy's (1991) model, which underscores the necessity for longitudinal studies on children's response to chronic stress and suggests that as children grow, new skills and vulnerabilities dynamically shape the trajectories of risk and resilience that are expressed at different stages of the child's life.

The different developmental trajectories of our war-exposed children stress the need for applying different treatment approaches to match the child's unique symptom trajectory and empathizes the necessity for early detection and intervention. The specific influence of maternal and child factors on symptomatology at different time points highlights the importance of constructing individual interventions that are sensitive to the child's developmental

stage, pathology onset, and family constellation. The strong effects of maternal symptoms and uncontained behavior on young children's symptom chronicity underscore the need for mother-child dyadic therapy during early childhood when families face stress of a chronic nature and highlight the importance of focusing on the parent-child relationship in the treatment of traumatized preschoolers. Such dyadic attachment-based intervention should concentrate on enhancing the mother's ability to contain the child's fears and draw her attention to the types of behavior which erode young children's security and elevate distress in the face of trauma reminders. Interventions focusing on the practice of parent-child transactions may provide the facilitative scaffolding necessary for the parent and child to work together through the traumatic events (Lieberman, Van Horn, & Ippen, 2005). In addition, it is important to address the mother's symptoms and encourage mothers to seek support when needed due to the significant impact of maternal emotional distress on young children's mental health. During late childhood, our findings suggest that interventions should focus on social training to enhance child social engagement and teach skills required for social initiation and participation in social groups, enabling children to benefit more from social contacts and support.

In addition to the focus on mother and child, the child's general ecology must be addressed in the context of chronic war exposure. War, by definition, disrupts the child's contextual, cultural, and social fabric. Thus, interventions must be guided by an ecological perspective and respect all layers of the child's ecology, including cultural customs, meaning systems, and religious rituals. As the family is the most proximal social environment and all members of the family suffer from the effects of war, the development of community-based interventions for the entire family is essential. Additionally, educational institutions should extend their role to provide accessible services to children affected by war, reduce trauma-related psychopathology, and emphasize normalization and interventions for school-age children. Moreover, the complementary function of schools in communities and their potential role in partnership with parents, extended family, and social networks should be exploited.

Several study limitations should be considered. First, the lack of father data is an important

drawback that precludes assessment of the child's entire rearing ecology. Second, including other familial attachments, such as grandparents, teachers, and close friends, could have given a more comprehensive assessment. Furthermore, a range of biological, brain, and endocrine factors could play a significant role in defining individual trajectories and the inclusion of such measures could enhance our understanding of risk and resilience across development. Finally, generalizability of the findings to other regions of political violence is required. Much further research is essential to assess the long-term impact of chronic war exposure on children's developmental trajectories. Additional followups are required at further critical nodes, such as the pubertal transition, leaving home, and forming intimate relationships. The effects of prolonged exposure to war-related trauma on allostatic load and subsequent effects on stress-response systems and physical health should also be examined, including stress genes, stress hormones, and stress-related neural networks. Finally, early interventions to improve well-being in war-exposed children, many of whom have witnessed horrendous atrocities, are critical to reduce the lasting negative effects of the current increase in political violence on citizens of tomorrow's world.

#### **Supporting information**

Additional Supporting Information may be found in the online version of this article:

**Table S1.** Pearson's correlations between middle and late childhood disorders.

**Table S2.** Pearson's correlations between middle and late childhood disorders by gender.

**Appendix S1.** Participants, methods, and correlation tables.

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# **Key points**

• Effects of chronic early trauma on children's mental health are well-recognized; yet, this study is the first to follow risk and resilience trajectories in war-exposed children across the first decade and chart predictors of individual pathways.

- Compared with controls, war-exposed children displayed significantly more Axis-I psychopathology and more comorbid disorders. Nearly a third of war-exposed children remained chronically symptomatic.
- Maternal factors, including mother's uncontained style during trauma evocation and symptomatology, increased risk for early-onset and chronic psychopathology.
- Low child social engagement increased propensity for late-onset psychopathology.
- Effects of early stress are not healed naturally and tend to exacerbate over time, with trauma-exposed children presenting more comorbid, chronic, and externalizing profiles as they grow. Results highlight the need for individually tailored interventions.

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