

From mothers to children and back: Bidirectional processes in the cross-generational transmission of anxiety from early childhood to early adolescence

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Abstract

Background: Maternal psychopathology and caregiving behavior are linked with child anxiety and these associations may be particularly salient when families face mass trauma together and members influence each other's symptomatology and resilience. Despite the well-known mother-to-child effects, less research addressed the longitudinal bidirectional effects of maternal and child's anxiety symptoms on each other.

Methods: Mothers and children exposed to chronic war-related trauma from Sderot, Israel, and comparison group were followed at three time-points; Early childhood (T1:N = 232, M_{Age} = 2.76 years), late childhood (T3:N = 176, M_{Age} = 9.3 years), and early adolescence (T4:N = 110, M_{Age} = 11.66 years). At each time-point maternal and child's anxiety symptoms were evaluated via questionnaires and maternal sensitivity was coded from videotaped observations of parent-child interactions. Bidirectional associations were examined using traditional cross-lagged panel model (CLPM) and CLPM with random intercepts (RI-CLPM).

Results: Trauma-exposed mothers and children exhibited more anxiety symptoms and lower maternal sensitivity. Cross-lagged panel models revealed cross-time bidirectional associations between maternal anxiety and child anxiety from early to late childhood. Child anxiety at each time-point predicted maternal anxiety and maternal sensitivity at the next stage; however, maternal sensitivity did not show longitudinal associations with child anxiety, highlighting children's role in shaping caregiving.

Conclusions: Findings demonstrate bidirectional cross-generational influences of mother and child on each other's anxiety in contexts of trauma and pinpoint early childhood as a sensitive period for such mutual influences. Children's increased anxiety following trauma appears to be further exacerbated via its impact on increasing maternal anxiety and compromising sensitive caregiving, underscoring the potential benefits of parental and mother-child interventions for trauma-exposed populations.

KEYWORDS

anxiety, bidirectional influences, longitudinal studies, maternal sensitivity, trauma

1 | INTRODUCTION

Exact evidence points to the pervasive effects of the rearing environment on children's propensity for psychopathology following trauma (Masten & Narayan, 2012; Pine & Cohen, 2002). Parental effects, including both parental symptomatology and caregiving behavior, are particularly salient in the context of trauma exposure; parenting provides protection, security, and emotion-regulatory functions for the child's immature neurobiological systems and when parenting suffers child adaptation is compromised (Gewirtz et al., 2008; Hamiel et al., 2017; Leen-Feldner et al., 2013). However, while the parent-to-child effects received extensive research, much less attention has been directed to the role children play in shaping their parents' behavior and influencing parental psychopathology or to the bidirectional transactions by which parent and child mutually influence each other's resilience (Drake et al., 2014; Pardini, 2008). Such research requires a longitudinal approach; hence, studies that span lengthy developmental epochs are particularly important for elucidating the mutual influences of parent and child on each other over time. Moreover, whereas the bidirectional perspective can shed light on the roots of resilience in general, it is particularly informative under conditions where the entire family is exposed to chronic stressors or trauma at the same time and members may play a role in shaping each other's resilience (Feldman, 2020). The current study focuses on the bidirectional transactions between maternal and child's anxiety symptoms as they unfold from early childhood to early adolescence in the context of chronic war-related trauma.

War exposure is an example of mass trauma where large populations are exposed to the same traumatic events at the same time (Masten & Narayan, 2012). Exposure to mass trauma places individuals at a greater risk for adverse psychological outcomes, the most common being posttraumatic stress disorder (PTSD), anxiety, and depression (Halevi et al., 2016; Heim & Nemeroff, 2001; Pine et al., 2005). Mass trauma typically involves exposure of the entire family to the same adverse events, a situation that is thought to trigger a "compound effect" in which family members influence each other's symptoms (Scheeringa & Zeanah, 2001). Mass trauma can therefore increase children's anxiety not only directly but also indirectly, through its impact on other family members. To fully understand the long-term effects of chronic collective trauma and devise effective interventions, it is important to explore the bidirectional influences of parent and child on the development of symptoms in the partner (Scheeringa & Zeanah, 2001). Unlike conditions such as abuse or neglect, which often occur within the family context, the source of suffering in conditions of mass trauma—including war, mass immigration, or natural disasters—is outside of the home (Masten & Narayan, 2012). In such conditions, which affect millions of children worldwide, it is possible to pinpoint the relatively independent role of maternal factors on child outcome, an assessment that is less relevant in relational trauma where the caregiver may be the source of trauma or an accomplice to it. Chronic collective trauma may thus provide a "natural experiment" to explore the mutual influences of maternal symptoms on child symptoms and back from the

child's to the maternal symptoms and how this bidirectional process is shaped by caregiving at different developmental nodes.

Apart from the mutual influences of maternal and child's anxiety symptoms on each other, the role of parenting behavior in the development of child anxiety disorders has been well-documented (Gouze et al., 2017; Negreiros & Miller, 2014; Wood et al., 2003; Yap et al., 2014). Although studies are inconclusive with regard to the specific parenting behaviors that predict anxiety (Negreiros & Miller, 2014; Rapee et al., 2009) and causality is unclear (Schrock & Woodruff-Borden, 2010; Williams et al., 2012), low maternal acceptance and warmth and increased intrusiveness and control are consistently described as risk factors for the development of anxiety disorders in childhood (Wood et al., 2003; Yap et al., 2014). Furthermore, while a meta-analysis indicated that only 4%–6% of the variance in child anxiety is explained by parenting behaviors (McLeod et al., 2007), others argue that the contribution of parenting to children's anxiety is underestimated (Williams et al., 2012).

Several studies assessed child-to-parent influences to describe the ways children, particularly those with anxiety disorders, may shape their parents' behavior. Overall, children with anxiety symptoms tend to elicit less optimal responses from their parents. For instance, while parents of non-anxious children responded to their child's negative emotions with positive or neutral feedback, parents of anxious children exhibited a more negative response (Hummel & Gross, 2001) and directed more negative affect and criticism toward their children (Hudson et al., 2009). However, these studies were cross-sectional, assessed children at the transition to adolescence (10–11 years), and did not consider parental anxiety and its influence over time. One study that measured longitudinal associations between maternal criticism and adolescents' depression and generalized anxiety disorder concluded that adolescents' depressive and anxiety symptoms not only co-occur with maternal criticism but erode the parent-child relationship over time (Nelemans et al., 2014). Similarly, a study on the impact of children's and parents' anxiety on the dyadic relationship found that each partner is influenced by the anxiety status of the other (Schrock & Woodruff-Borden, 2010). Authors have suggested that parent-child mutual influences of anxiety can be attributed, at least in part, to shared genetic predispositions (Hettema et al., 2001). Still, longitudinal studies found that anxiety symptoms in infants and children are more closely linked with their adoptive parents' anxiety levels than with genetic risk (Ahmadzadeh et al., 2019; Brooker et al., 2015). Thus, to further understand the bidirectional transmission of anxiety over time, longitudinal studies that repeatedly assess maternal anxiety, child anxiety, and parenting quality are required.

In the current study, we focused on maternal sensitivity as the key indicator of parenting quality. Maternal sensitivity is the central behavioral construct utilized in observational studies of the parent-child relationship, shows individual stability from infancy to adolescence, and has shown longitudinal associations with secure attachment and adaptive social-emotional development (Feldman, 2007, 2021; van IJzendoorn et al., 1995). Maternal sensitivity may be compromised under various risk conditions related to maternal and

contextual stress (Campbell et al., 2007; Feldman & Vengrober, 2011; Ulmer-Yaniv, Djalovski, Yirmiya, Halevi, Zagoory-Sharon, Feldman 2017), and the decrease in maternal sensitivity has shown to mediate the effects of harsh rearing environments on child outcomes (Bouvette-Turcot et al., 2017; Farrell et al., 2017). To complement the assessment of maternal sensitivity and its long-term influences, we also considered an alternative model that included maternal intrusiveness, a maternal style associated with mother and child's anxiety (Cooper-Vince et al., 2014; Feldman et al., 1997; Wood, 2006). We chose to focus on internalizing symptoms in early childhood and anxiety disorders in late childhood and adolescence as anxiety disorders are the most common psychiatric conditions beginning in early childhood and continuing throughout life (Kessler et al., 2005, 2012). Furthermore, internalizing problems in early childhood have been associated with later anxiety disorders (Zahn-Waxler et al., 2000). In our sample, anxiety disorders were the most common disorders in the mothers and the second most common disorders in the children (after ADHD) in late childhood (Halevi et al., 2017).

We utilized a cohort of mothers and children living in Sderot, Israel, a small town located near the Gaza border and exposed to repeated and unpredictable war-related trauma for nearly two decades. Over the years, thousands of rockets and missiles have been launched on Sderot, leaving citizens with only 15 s to find shelter when alarm sirens are heard. Previous studies on mass trauma typically assessed the co-occurrence of psychopathology in mothers and children, the associations between parenting behavior and child risk and resilience, and the effects of mass trauma on maternal mental health and parenting (Costa et al., 2009; Gewirtz et al., 2008; Halevi et al., 2017; Hamiel et al., 2017; Yirmiya et al., 2018), but no study to our knowledge addressed the longitudinal bidirectional influences of maternal and child's symptoms on each other over time. Very few cross-generational studies on anxiety have been conducted with families exposed to extreme adversity, and, to our knowledge, none utilized longitudinal data and random-intercept cross-lagged panel modeling (RI-CLPM) (Hamaker et al., 2015). The RI-CLPM approach has the potential to overcome some of the methodological problems observed in the traditional CLPM approach, particularly the mixture of within- and between-person effects that may compromise the validity of research findings (Curran & Bauer, 2011; Hamaker et al., 2015).

The goals of the current study were (1) to examine the effects of chronic trauma on mothers' and children's anxiety symptoms and on maternal sensitivity from early childhood to early adolescence and assess their inter-relationships at each time-point; (2) to test the cross-lagged effects of maternal anxiety and sensitive parenting at each time-point on child anxiety at the next developmental stage, and (3) vice versa, to describe the associations between child anxiety at each time-point on maternal anxiety and sensitive behavior at the next stage. Based on prior research (Ulmer-Yaniv, Djalovski, Yirmiya, Halevi, Zagoory-Sharon, Feldman 2017; Yirmiya et al., 2018), we hypothesized that trauma-exposed children and mothers would show more anxiety symptoms at each stage as compared to control mothers and children and that maternal sensitivity would be compromised in the exposed group. We further hypothesized that maternal

anxiety would be associated with child anxiety both concurrently and longitudinally and that both maternal and child anxiety would correlate with reduced maternal sensitivity. Finally, we expected bidirectional associations between maternal anxiety, maternal sensitivity, and child anxiety across the three developmental stages. We assumed that maternal sensitivity and maternal anxiety would impact child anxiety, particularly during early childhood when mothers play a critical role in regulating the child's stress (Scheeringa et al., 2011). As children grow, child effects were expected to increase in magnitude and we hypothesized that at later stages anxious children may impact their mothers' symptoms and behavior, leading to a cycle of reduced sensitivity that further compromises symptoms and caregiving (Rapee et al., 2009).

2 | MATERIALS AND METHODS

2.1 | Participants

T1: Early childhood. Participants of this study were recruited in 2004–2005 and included 232 children and their mothers (47.6% males and 47.1% firstborns). Of these, 148 dyads were living in the same frontline neighborhoods in Sderot, Israel, and comprised the trauma-exposed group. This group was recruited through clinicians living in Sderot, advertisements in day-care centers, and snowball recruitment. A comparison group of 84 dyads was recruited from comparable towns within the greater Tel-Aviv area who matched on sociodemographic variables to the town of Sderot in terms of population size, socioeconomic composition, housing, and employment opportunities. These areas were not exposed to chronic war-related trauma and did not suffer from missile or rockets attacks. Control children were matched to the exposed group in age, gender, birth order (firstborn/late born), maternal and paternal age and education, and maternal employment and marital status (Feldman & Vengrober, 2011). Before recruitment, control families were screened by phone for major traumatic events in the child's life (e.g., motor vehicle accidents), and those reporting such trauma were excluded. Children in both the war-exposed and control groups were screened for records of physical abuse or neglect. Home visits were conducted when children were between the ages of 1.5–5 ($M = 2.76$ years, $SD = 0.91$).

T2: middle childhood. Of the original cohort, 210 children were seen between the ages of 5–8 ($M = 7.68$, $SD = 0.7$). Data from this stage included only phone-based interviews and did not contain mother-child interaction or questionnaires and is therefore not reported here.

T3: late childhood. Of the initial cohort, 177 families were revisited at their home when children were 9–11 years old ($M = 9.3$ years, $SD = 1.41$; 101 exposed and 76 control).

T4: Early Adolescence. At this stage, 111 mother-child dyads visited the Lab when children were between 11 and 13 years old ($M = 11.66$, $SD = 1.23$; 58 exposed and 53 control).

Attrition was mainly related to the inability to locate families or families moving out of Sderot. No differences in sociodemographic,

child anxiety, maternal anxiety, and maternal sensitivity from T1 were found between continuing versus non-continuing families (see Table S1). The study was approved by the University's Institutional Review Board and all parents signed informed consent.

3 | PROCEDURES

T1: Early childhood. Families were visited at home for about 3.5 h. Trained clinical psychologists conducted psychiatric interviews, mothers filled questionnaires and 10 min of mother-child free play interactions was videotaped (see Feldman et al., 2013). The experimenter placed a box of preselected toys in front of mother, and instructions were "Play with your child as you normally do."

T3: Late Childhood: Mothers and children were revisited at home, interviewed, and engaged in interaction paradigms. We used the well-validated positive interaction paradigm in which mother and child are asked to plan the "best day ever" to spend together for 7 min (Halevi et al., 2017; Ulmer-Yaniv et al., 2017). Following, participants completed questionnaires.

T4: Early Adolescence: Mothers and children visited the lab and visits included interviews, hormonal assessment, and relational and individual paradigms. Dyads were asked to play together an "Etch a Sketch" game for 7 min where each partner controlled one knob and the two collaborated in drawing a joint picture (Yirmiya et al., 2018). Following, mothers and children completed questionnaires.

4 | MEASURES

4.1 | Child behavior checklist (CBCL: T1)

At T1, mothers reported on their children's problem behaviors using the Child Behavior Checklist (CBCL/1.5–5, Achenbach & Rescorla, 2000). This version of the CBCL consists of 99 items and responses range from 0 (never applies) to 2 (almost always applies). In the current study, we used the internalizing problems scale, which comprises the sum score of the following subscales; anxious/depressed (8 items), emotionally reactive (9 items), somatic complaints (11 items), and withdrawn (8 items). The choice to use the CBCL internalizing score stemmed from the need to match as much as possible the symptoms reported on the anxiety questionnaires at the next stages. Cronbach's α for the internalizing scales problems in the current sample was .88.

4.2 | Screen for child anxiety related emotional disorders (SCARED: T3, T4)

In late childhood and adolescence mothers and children each completed the Screen for Child Anxiety Related Emotional Disorders (SCARED). The SCARED (Birmaher et al., 1997) is a 41-item child/parent report used to screen for anxiety disorders in children during

the past three months with responses on each item ranging from 0 (not true or hardly ever true) to 2 (true or often true). Total SCARED scores of mother and child were inter-related at both T3 and T4 ($r = .28$ and $.31$, respectively, $ps < .001$). Consistent with previous studies that indicated the benefits of using both children's and mothers' reports, we used the average of maternal and child's reports in the following analyses (Bar-Haim et al., 2007; Wren et al., 2004; Yirmiya et al., 2018). Cronbach's α for the SCARED questionnaire in the current sample were .98, .91 and .91, .91 for children and mothers at T3 and T4, respectively.

4.3 | Maternal trait anxiety (T1, T3, T4)

The Trait Anxiety Inventory (TAI) is a subscale of the Spielberger State-Trait Anxiety Inventory (STAI), a widely used self-report tool to assess general anxiety (Spielberger et al., 1970). The STAI-T comprises 20 descriptive items ranging from 1 (almost never) to 4 (almost always) and the final score is their aggregated sum. The Cronbach's α coefficient reported in the current study was .94, .84, .89 at T1, T3, and T4 respectively.

4.4 | Maternal sensitivity and maternal intrusiveness (T1, T3, T4)

Dyadic interactions at T1, T3, and T4 were each coded using the Coding Interactive Behavior (CIB) Manual (Feldman, 1998). The CIB is a well-validated tool to evaluate social behavior across ages that has shown good psychometric properties across different ages and cultures. Interactions are coded on multiple global scales from 1 (low) to 5 (high) based on frequency, intensity, and duration of each behavior or social orientation. The maternal sensitivity construct was chosen due to its widespread use as one of the most researched maternal constructs and its relevance in the context of anxiety and trauma (Degnan et al., 2015; Feldman & Vengrober, 2011; Scheeringa et al., 2015; Warren & Simmens, 2005). The maternal sensitivity construct included the following codes: maternal positive affect, maternal acknowledgment of child social communication, maternal praising, appropriate range of affect, maternal enthusiasm and energetic focus, and maternal warmth. At late childhood and early adolescence, when interactions are based on verbal exchange, we also added the code of maternal empathy. To complement the assessment of parenting behavior in the intergenerational study of anxiety we tested an alternative model in which mother intrusiveness was entered instead of sensitivity. The maternal intrusiveness construct was similarly coded for interactions at T1, T3, and T4 and includes the following codes; maternal forcing, overriding, maternal negative affect/anger, hostility, mother anxiety, and criticizing. Two coders, blind to any other information, exceeded 90% reliability on 20% interactions for all coded behaviors ($k = .82$, range = $.74-.95$). Cronbach's α values for the three time-points were .83, .91 and .88 for T1, T3, and T4, respectively.

5 | STATISTICAL ANALYSIS

We used SPSS for Windows version 23 (IBM et al., 2016) and *Mplus* 8.0 (Muthén & Muthén, 2017). To test the first hypothesis, ICCs were evaluated and group comparisons in maternal and child variables were computed with *t* tests (Hypothesis 1). Following, the mean score for each variable was computed and standardized to examine group differences across the three time-points. Pearson correlations were computed for a full matrix of correlations among study variables. To characterize bidirectional mother-child effects (Hypotheses 2 and 3), we generated cross-lagged panel analyses (CLPM). The CLPM is a widely endorsed statistical approach for estimating within-person causal influences in longitudinal panel data. In brief, the CLPM uses the participant's standing on a specific variable at a given time-point as a predictor for change in the outcome variable from that time-point to the next. Our hypotheses were first examined using the traditional CLPM and included stability paths between variables at each consecutive wave, cross-lagged paths among variables at each consecutive wave, and correlations between variables within a wave (see Figure 1a). Recently, however, the CLPM approach has been criticized on grounds that the stability paths (i.e., autoregressive relationships) of the CLPM fail to adequately account for the trait-like, time-invariant nature of constructs, and, as a result, the lagged parameters obtained by the CLPM do not represent the actual within-person relationships over time (Hamaker et al., 2015). It has been suggested that the RI-CLPM can better separate within-person effects from between-person stability in symptoms and can thus better detect within-person reciprocal relationships among variables over time (Curran & Bauer, 2011; Hamaker et al., 2015) (Figure 1b).

The RI-CLPM uses a latent variables approach to person mean centering and to distinguish stable between-person differences in constructs across time-points from within-person variations at each time-point on those same constructs. The resulting stability and cross-lagged paths represent how variations in levels of a construct within each family member predict change in the same or different constructs in that individual or the partner over time. In turn, correlations among the latent between-person variables represent whether individuals who show overall high ranking on a construct across time-points (as compared to other participants) also rank higher or lower overall on another construct (Hamaker et al., 2015). Missing data at both the CLPM and RI-CLPM were handled using the full information maximum likelihood method (FIML), which is a state-of-the-art method for obtaining parameter estimates (Schafer & Graham, 2002). The method utilizes all available data to calculate maximum likelihood parameter estimates with standard errors that are robust to non-normality. The following indicators were used to evaluate the model fit for the CLPM and RI-CLPM models: χ^2 values, and their degrees of freedom and *p*-values, with good fit indexed by nonsignificant values; root mean square error of approximation (RMSEA), values that are <0.06 are considered to indicate a good fit; comparative fit index (CFI), and Tucker-Lewis index (TLI), values >0.90 are considered to indicate a good fit (Hu & Bentler, 1999). We estimated our models with and without sex as a covariate to ensure that it had minimal effects as well

as compared our final model to three alternatives RI-CLPM and these are presented in the Supporting Information.

6 | RESULTS

6.1 | Descriptive statistics and correlations

Group comparisons in sociodemographic variables in childhood and adolescence can be found in previous studies (Feldman & Vengrober, 2011; Halevi et al., 2017; Yirmiya et al., 2018). Means, *SD*, and *t* tests of group differences for study variables are presented in Table 1 and across all time-points in Figure 2. Maternal anxiety was significantly higher across the study in war-exposed mothers. War-exposed children showed more internalizing symptoms at T1 and higher anxiety at T3 and T4. Lower levels of maternal sensitivity were found in war-exposed mothers, compared with controls, at T1 and T4; however, this difference did not reach statistical significance at T3 ($p = .06$). Maternal intrusiveness was significantly higher among exposed mothers compared to controls at T4, although no group differences in maternal intrusiveness were found at T1 and T3. As expected, assessments of maternal sensitivity, maternal anxiety, and child anxiety were generally correlated, pointing to individual stability in anxiety symptoms and parenting style (Table 2). Sociodemographic differences between dropout families and families participated at T4 are presented in Table S1, and comparisons between groups in main demographic variables from T1 and T4 are presented in Table S2. Intraclass correlation coefficients (ICCs) suggested that a substantial part of the variance in all study variables stemmed from the within-person level, that is, considerable part of the observed variation resulted from fluctuations over time in the study variables and not from stable between-person differences. ICCs were 0.57 for maternal anxiety, 0.56 for child anxiety, and 0.13 for maternal sensitivity, indicating that there was sufficient within-person variation to run the RI-CLPM (Berry & Willoughby, 2017; Hamaker et al., 2015).

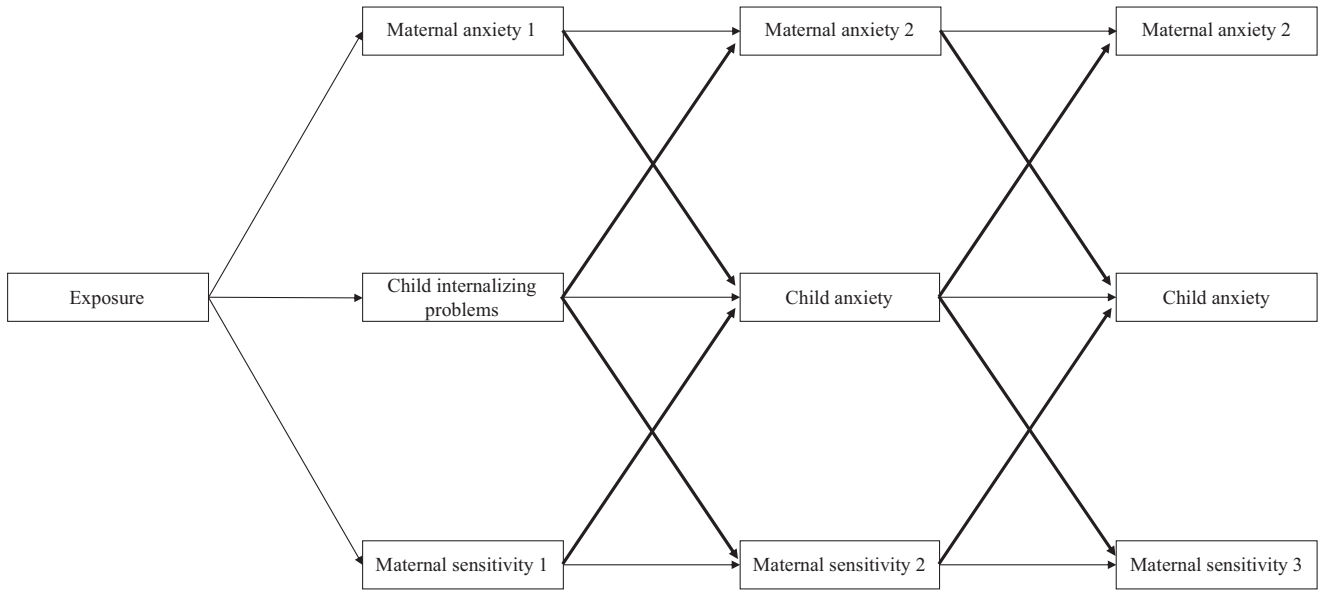
7 | STANDARD CROSS-LAGGED PANEL MODEL

Results from the standard CLPM indicated bidirectional relationships between maternal anxiety, maternal sensitivity, and child anxiety symptoms from early childhood to adolescence, demonstrating an overall good model fit: $\chi^2_{(16)} = 19.66, p = .24$; RMSEA = .03; CFI = 0.99; TLI = .96. All unstandardized and standardized path coefficients and covariances of the study model are presented in Table 3. Exposure was associated with maternal anxiety, maternal sensitivity, and child internalizing problems at T1.

Effects of maternal anxiety on child anxiety: Maternal anxiety at T1 predicted child anxiety at T3, but no association was found between maternal anxiety at T3 and child anxiety at T4.

Effects of maternal sensitivity on child anxiety: Maternal sensitivity did not predict child anxiety from T1 to T3, and, similarly, not from T3 to T4.

(a)



(b)

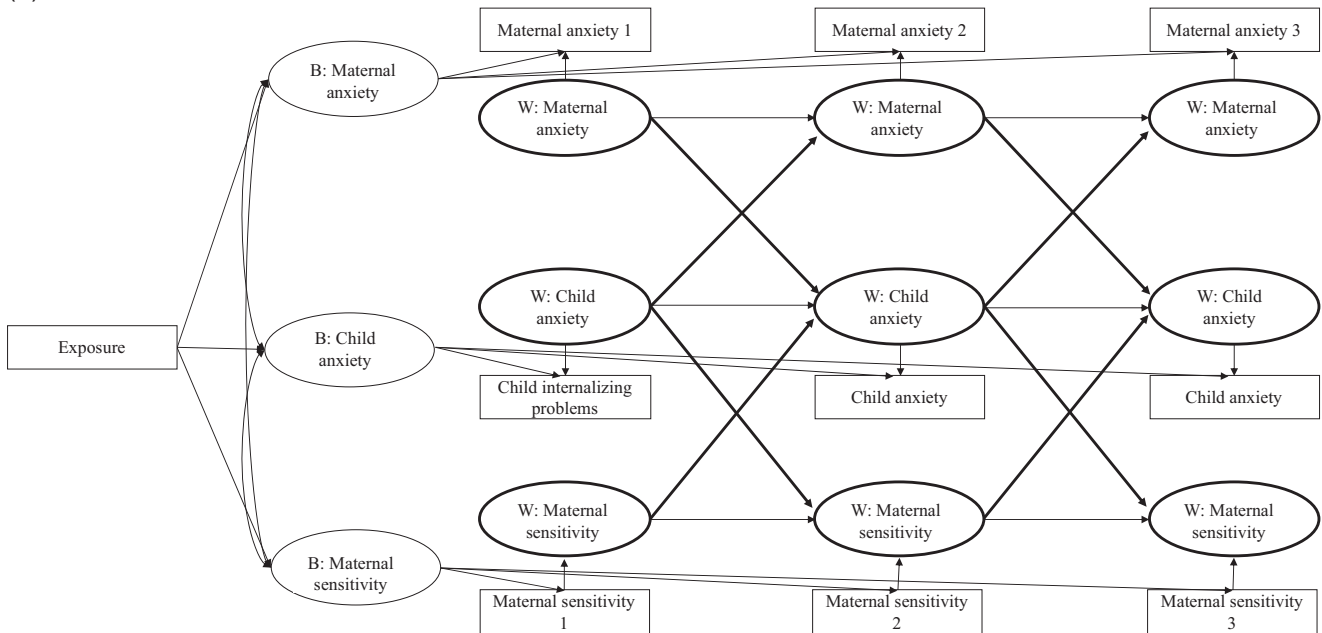


FIGURE 1 Standard Cross Lag Panel Model (a) and Random Intercept Cross-Lagged Panel Model (RI-CLPM) (b). Within-wave correlations among latent variables are modeled in the RI-CLPM but not depicted. B, between-person; W, within-person

Effects of child anxiety on maternal anxiety: Child internalizing symptoms at T1 predicted maternal anxiety at T3. However, child anxiety at T3 did not predict maternal anxiety at T4.

Effects of child anxiety on maternal sensitivity: Child internalizing symptoms at T1 predicted maternal sensitivity at T3. Child anxiety at T3 also predicted maternal sensitivity at T4.

Adding sex as a covariate to the model revealed no change in significance for any path and discrepancies in beta weights were generally small. All stability paths in the model were positive and significant.

8 | RANDOM INTERCEPT CROSS-LAGGED PANEL MODEL

The RI-CLPM fit the data well: $\chi^2_{(16)} = 11.07, p = .81, RMSEA = .00, CFI = 1.0, TLI = 1.05$. All unstandardized and standardized path coefficients and covariances of the study model are presented in Table 4.

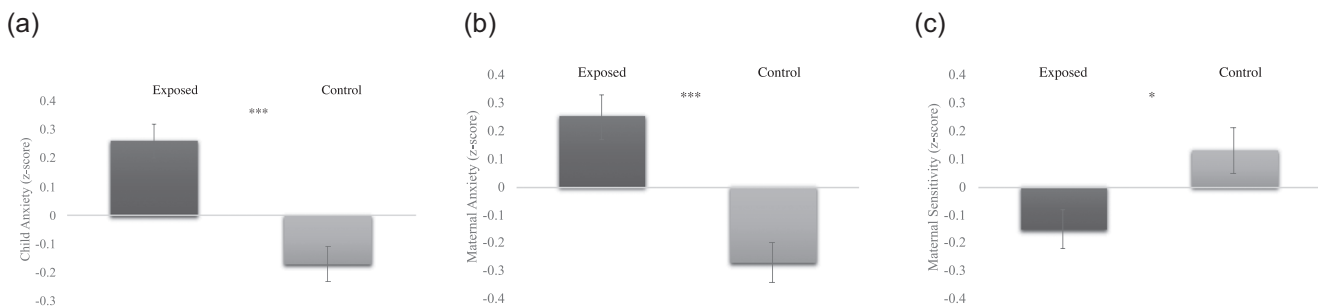
Effects of maternal anxiety on child anxiety: Similar to the results of the CLPM, at the within-person level, maternal anxiety at T1

	Control		Exposed		t test
	M	SD	M	SD	
Time 1	N = 84		N = 148		
Child internalizing problems	5.07	5.15	8.23	9.62	$t_{(193)} = -2.92, p < .01$
Mother STAI	34.20	7.90	39.86	10.40	$t_{(222)} = -4.51, p < .01$
Maternal sensitivity	3.69	.75	3.44	.79	$t_{(201)} = 2.33, p < .05$
Maternal intrusiveness	1.44	.38	1.52	.52	$t_{(201)} = -1.57, p > .05$
Time 3	N = 76		N = 101		
Child SCARED	17.67	8.43	22.76	9.92	$t_{(173)} = -3.66, p < .01$
Mother STAI	31.16	7.88	37.83	10.24	$t_{(169)} = -4.78, p < .01$
Maternal sensitivity	2.85	.82	2.60	.68	$t_{(171)} = 1.87, p = .06$
Maternal intrusiveness	1.15	.36	1.10	.22	$t_{(171)} = 1.18, p > .05$
Time 4	N = 58		N = 53		
Child SCARED	18.91	0.10	22.79	11.07	$t_{(108)} = -2.08, p = .04$
Mother STAI	34.00	7.63	39.37	9.98	$t_{(101)} = -2.98, p < .01$
Maternal sensitivity	2.37	.80	2.09	.77	$t_{(98)} = 2.08, p < .05$
Maternal intrusiveness	1.67	.60	2.02	.75	$t_{(98)} = -2.59, p = .01$

TABLE 1 Means and standard errors for main study variables

Note: Child internalizing problems at T1 were evaluated using CBCL 1.5-5; maternal sensitivity was evaluated using the Coding Interactive Behavior (CIB).

Abbreviations: SCARED, Scale for Child Anxiety Related Emotional Disorders; STAI, Spielberger State-Trait Anxiety Inventory.



Note: numbers represent mean z-score of T1, T3 and T4.

FIGURE 2 Mean score comparisons between war-exposed and control subject in (a) child anxiety (b) maternal anxiety and (c) maternal sensitivity. Note: Numbers represent mean z-score of T1, T3, and T4

predicted higher child anxiety at T3, but no association was found between maternal anxiety at T3 and child anxiety at T4.

Effects of maternal sensitivity on child anxiety: Similar to the results of the CLPM, maternal sensitivity did not predict child anxiety from T1 to T3, nor from T3 to T4.

Effects of child anxiety on maternal anxiety: Similar to the results of the CLPM, at the within-person level, child internalizing symptoms at T1 predicted maternal anxiety at T3. However, in contrast to the CLPM model, child anxiety at T3 predicted maternal anxiety at T4.

Effects of child anxiety on maternal sensitivity: Similar to the results of the CLPM, at the within-person level child anxiety at T1

predicted maternal sensitivity at T3, and child anxiety at T3 predicted maternal sensitivity at T4.

Autoregressive pathways: The within-persons level of the model is also examined by the autoregressive pathways, that is, whether the individual's deviation from the expected symptoms carries over to the next measurement time-point, reflecting stability of this variable. While child anxiety autoregressive pathways were significant throughout the study, maternal anxiety were less consistent; After accounting for trait-like stability in maternal anxiety in the context of the RI-CLPM, maternal symptoms showed only within-persons carry over effects from T1 to T3, and no carry over effects were found for maternal behavior.

TABLE 2 Pearson correlations between child anxiety, mother anxiety, and maternal sensitivity

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Child internalizing problems T1	1										
2. Child SCARED T3	.36**	1									
3. Child SCARED T4	.18	.58**	1								
4. Mother STAI T1	.35**	.34**	.21*	1							
5. Mother STAI T3	.40**	.30**	.17	.52**	1						
6. Mother STAI T4	.19	.30**	.31**	.67**	.71**	1					
7. Maternal sensitivity T1 (1-5)	-.14	-.08	-.07	-.26**	-.05	-.20	1				
8. Maternal sensitivity T3 (1-5)	-.17*	-.07	<.001	-.14	-.13	-.05	.30**	1			
9. Maternal sensitivity T4 (1-5)	-.19	-.28**	-.22*	-.24*	-.20*	-.11	.29**	.41**	1		
10. Maternal intrusiveness T1 (1-5)	.21**	.11	.22	.24*	-.01	.06	-.45**	-.16*	-.24*	1	
11. Maternal intrusiveness T3 (1-5)	.16*	-.03	-.01	.12	.08	.13	-.14	-.37*	-.16	.224**	1
12. Maternal intrusiveness T4 (1-5)	.18	.02	.12	.12	.11	.05	-.07	-.21*	-.50**	.16	.08

Note: Child internalizing problems at T1 were evaluated using CBCL 1.5-5; maternal sensitivity was evaluated using the Coding Interactive Behavior (CIB). Abbreviations: SCARED, Scale for Child Anxiety Related Emotional Disorders; STAI, Spielberger State-Trait Anxiety Inventory.

** $p < .01$, * $p < .05$.

TABLE 3 Parameter estimates for cross-lagged panel model linking maternal anxiety, child anxiety, and maternal sensitivity

Parameters	B	SE	p	β	B	SE	p	β	B	SE	p	β
Exposure → Mother.Anx. T1	5.71	1.24	***	.294								
Exposure → Child.Anx. T1	2.95	1.06	.005	.194								
Exposure → Mother.Sens. T1	-.253	.109	.019	-.161								
Stability path	T1 → T3			T3 → T4			T1 → T4					
Mother.Anx.	.424	.068	***	.431	.492	.068	***	.499	.383	.066	***	.395
Child.Anx.	.350	.094	***	.279	.569	.089	***	.556	-.037	.114	.742	-.029
Mother.Sens.	.294	.076	***	.294	.323	.099	.001	.300	.222	.101	.028	.207
Cross-Lagged Path	T1 → T3			T3 → T4								
Mother.Anx. → Child.Anx.	.241	.074	.001	.246	.064	.089	.471	.063				
Child.Anx. → Mother.Anx.	.272	.088	.002	.217	.046	.065	.480	.046				
Child.Anx. → Mother.Sens.	-.017	.008	.027	-.164	-.019	.008	.014	-.219				
Mother.Sens. → Child.Anx.	-.156	.912	.864	-.013	.597	1.004	.552	.048				
Correlation/Correlated Change	T1			T3			T4					
Mother.Anx. ↔ Child.Anx.	19.85	5.133	***	.287	7.025	5.351	.189	.102	9.48	4.69	.043	.207
Mother.Anx. ↔ Mother.Sens.	-1.65	.518	.001	-.230	-.325	.458	.478	-.055	1.05	.451	.020	.250
Mother.Sens. ↔ Child.Anx.	-.576	.436	.186	-.100	.101	.486	.835	.016	-.543	.600	.365	-.092

Note: $\chi^2_{(16)} = 19.66$, $p = .24$, RMSEA = .03, CFI = .99, TLI = .96.

Abbreviations: Child.Anx., child anxiety; CFI, comparative fit index; Mother.Anx., mother anxiety; Mother.Sens., mother sensitivity; RMSEA, root mean square error of approximation; T, time; TLI, Tucker-Lewis index.

*** $p < .001$; significant ($p < .05$) paths in bold.

TABLE 4 Random Intercept Cross-Lagged Panel Model (RI-CLPM) linking maternal anxiety, child anxiety and maternal sensitivity

Parameters	B	SE	p	β	B	SE	p	β	B	SE	p	β
Exposure→ Mother.Anx. T1	5.666	1.050	***	.382								
Exposure→ Child.Anx. T1	3.339	.897	***	.488								
Exposure→ Mother.Sens. T1	-.250	.084	.003	-.298								
Stability path	T1→T3				T3→T4							
Mother.Anx.	-.505	.209	.016	-.535	.079	.141	.576	.079				
Child.Anx.	.378	.168	.025	.313	.600	.099	***	.582				
Mother.Sens.	.007	.169	.967	.007	.161	.158	.307	.149				
Cross-Lagged Path	T1→T3				T3→T4							
Mother.Anx.→ Child.Anx.	.468	.203	.021	.293	.067	.157	.667	.039				
Child.Anx.→ Mother. Anx.	.449	.116	***	.629	.218	.080	.006	.368				
Child.Anx.→ Mother.Sens.	-.015	.007	.033	-.190	-.017	.008	.025	-.236				
Mother.Sens. → Child.Anx.	.441	1.349	.744	.029	.987	1.314	.453	.065				
Correlation/correlated Change	T1				T3				T4			
Mother.Anx.↔ Child.Anx.	18.718	4.76	***	.371	16.639	7.99	.037	.443	9.834	4.74	.038	.226
Mother.Anx.↔ Mother.Sens.	-1.135	.39	.004	-.279	-.031	.42	.942	-.011	1.097	.45	.015	.296
Mother.Sens.↔ Child.Anx.	-.516	.43	.229	-.096	.262	.52	.61	.047	-.380	.61	.536	-.067
Between person correlation	Across Waves											
Mother.Anx.↔ Child.Anx.	2.830	12.77	.825	.10								
Mother.Anx.↔ Mother.Sens.	-.294	.50	.031	-.33								
Mother.Sens.↔ Child.Anx.	-1.068	.97	.762	-.18								

Note: $\chi^2_{(16)} = 11.07$, $p = .81$, RMSEA = .00, CFI = 1.0, TLI = 1.05. Variance explained by the random intercept (R^2): Mother.Anx. T2 = .04; Child.Anx.T2 = .25; Mother.Sens.T2 = .43; Mother.Anx. T3 = .16; Child.Anx.T3 = .36; Mother.Sens.T3 = .08; Mother.Anx. Total = .31; Child.Anx.Total = .15; Mother.Sens.Total = .09.

Abbreviations: Child.Anx., child anxiety; CFI, comparative fit index; Mother.Anx., mother anxiety; Mother.Sens., mother sensitivity; RMSEA, root mean square error of approximation; T, time; TLI, Tucker-Lewis index.

*** $p < .001$; significant ($p < .05$) paths in bold.

At the between-person level, the latent trait factors of maternal anxiety and maternal sensitivity were significantly and negative correlated, indicating that mothers who were typically higher in maternal sensitivity across the three time-points tended to be anxious over this developmental period. No significant between-variables associations were found between child anxiety and maternal anxiety or between child anxiety and maternal sensitivity.

8.1 | Alternative path analysis models

To further validate the presented path model, we compared it to several alternatives RI-CLPM and these are presented in the Supporting Information. First, we ran the same model with maternal intrusiveness instead of maternal sensitivity (Table S3). This model provided a less adequate fit to the data, and no significant associations were observed between maternal intrusiveness at T1, T3, and T4, as well as between child anxiety and maternal intrusiveness.

Second, to account for the unique information regarding children's anxiety, we ran separate models of mother-reports and child-reports. Child-report was available only at T3 and T4 and we therefore used mother-report at T1 in all models. Both models had good fit (Tables S4 and S5). In the mother-report model, all stability and cross-lagged paths that were significant in the original RI-CLPM remained significant. However, in the child-report model, the cross-lagged paths between child anxiety at T3 and maternal sensitivity and maternal anxiety at T4 did not reach statistical significance. Furthermore, we ran our model without maternal sensitivity (a two variable model) as a sensitivity analysis. This model revealed similar effects to the original model and the model fit was also good (Table S6). Lastly, to further validate our original path model we compared it to three alternative models. The first alternative model utilized the same model with added paths from maternal sensitivity to maternal anxiety and vice versa (Table S7). In the second alternative model all variables were controlled for maternal anxiety in T1, to assure that the effects in late childhood and early adolescence are not merely a

consequence of the levels of anxiety mothers experienced in early childhood (Table S8). In the third alternative model, we controlled all variables with maternal sensitivity from early childhood (Table S9). All three models showed good fit to the data and the original paths found in the original RI-CLPM remained significant. There were no significant differences between the alternative models and the original model.

9 | DISCUSSION

We followed a cohort of war-exposed and control mothers and children from early childhood to early adolescence to describe the mutual influences of maternal behavior, maternal anxiety, and child's anxiety symptoms on the subsequent development of anxiety in the partner. To address these cross-time cross-generational effects, we capitalized on a unique "natural experiment" of war-exposed families living in the same frontline neighborhoods where the source of trauma is outside the family and the traumatic events are shared by mother and child. Results indicate that chronic trauma carries significant effects on the development of anxiety symptoms in mother and child and on the expression of parenting behavior and all three variables measured—maternal anxiety, child anxiety, and mother sensitivity—showed group differences at most time-points. In contrast, when we replaced maternal sensitivity with maternal intrusiveness, group differences were found only at one time-point and the path model showed a less adequate fit to the data as compared to the model including maternal sensitivity. Overall, our study provides the first evidence for the bidirectional influences of maternal and child's anxiety in the context of a chronic stressor and our results describe how mother and child shape each other's anxiety over time through both mother-to-child and child-to-mother cross-generational influences. Furthermore, our cross-generational cross-time models uniquely employed both the traditional CLPM and a relatively new method, the RI-CLPM, which enables the separation of between- and within-person effects. These models revealed mainly consistent findings as well as several distinct results that are discussed below.

9.1 | Bidirectional effects of maternal and child's anxiety

Both the CLPM and the RI-CLPM models showed that during the early stages of development the associations between maternal and child's anxiety symptoms are bidirectional. Specifically, maternal and child's anxiety symptoms in late childhood were uniquely predicted by the individual's own anxiety in early childhood as well as by partner's anxiety level at the same time-point. Our results on the mutual influences at the within-person level between maternal and child's anxiety across the first decade of life are consistent with previous research indicating that young children of anxious mothers are at a greater risk for the development of adjustment problems, anxiety, and attention problems compared to children of non-anxious mothers (Gouze et al., 2017; O'Connor et al., 2002;

Rees et al., 2019). Factors such as modeling and the provision of appropriate information in the face of threats have been associated with the cross-generational transmission of anxiety from mothers to young children (Murray et al., 2009). Another line of explanation suggests that due to their own disorder, anxious parents may limit their children's experiences and restrict the child's opportunities to face challenging situations and acquire a sense of competence (Murray et al., 2009). These within-person level bidirectional effects may be further enhanced by the "compound effect" that characterizes mass trauma where events influence family members both directly and indirectly through the difficulties of other members (Scheeringa & Zeanah, 2001).

Mother-child bidirectional anxiety effects were not significant between late childhood and early adolescence in the CLPM model; however, the RI-CLPM model revealed a within-person significant effect in which child anxiety in late childhood predicted maternal anxiety in early adolescence. The fact that maternal anxiety did not predict child anxiety at the transition to adolescence is surprising, particularly since these assessments were closer in time as compared to the longer gap between early and late childhood. One possibility is that the effects of parental anxiety on child anxiety weaken as children grow and develop close social contacts outside the family setting, including friends, teachers, and mentors. A similar decrease in parental influences has been reported for the effects of parenting behavior on children's anxiety, which was found to be stronger in childhood than in adolescence (Verhoeven et al., 2012), reflecting an overall weakening of the child's dependence on the mother. One study has shown that anxiety in adolescence is predicted by stressors experienced in the first five years of life, even while controlling for current adversities, and the authors concluded that anxiety may be more strongly linked with early stress exposure (Phillips et al., 2005). However, as most research on child anxiety is cross-sectional and taps early stages of development, the effects of maternal anxiety on children as they grow require much further research, particularly on the mutual influences of anxiety between parents and older children and adolescents. Separating the within-person process from the between-person associations revealed that when controlling for individual differences in maternal anxiety, mothers' anxiety levels can be predicted from their children's anxiety symptoms at earlier stages. Although several studies described the mechanisms by which child anxiety shapes parenting (Gar & Hudson, 2008; Moore et al., 2004), much less research addressed the role of children's anxiety in shaping maternal anxiety over time and our findings may offer a unique perspective on the child-to-parent effects in the context of chronic adversity. It is also important to point out that the between-person-level effect of maternal anxiety and child anxiety was nonsignificant.

9.2 | Bidirectional effects of maternal behavior and child anxiety

Similar to maternal anxiety, which was predicted by children's anxiety in late childhood and early adolescence, maternal sensitivity was also longitudinally linked with children's anxiety at the previous stage at

the within-person level, as shown by both the CLPM and RI-CLPM models. These findings are consistent with perspectives on anxiety and parenting in childhood and adolescence suggesting that child effects on parental warmth and control is at least as strong as the effects of parental factors on children's anxiety (McLeod et al., 2007; Waite et al., 2014). Parents of anxious children are less likely to exhibit or discuss positive emotions as compared to parents of non-anxious children (Suveg et al. 2005), and mothers of anxious children aged seven to fifteen were found to be less warm regardless of their own diagnosis (Moore et al., 2004). Longitudinal studies show that child anxiety symptoms emerged earlier than parental overprotection (Rubin et al., 1999) and tended to predict parental hostility (Gouze et al., 2017) and maternal criticism (Nelemans et al., 2014). In comparison, findings for the opposite direction, from negative parenting to child anxiety symptoms, were less consistent across childhood (Gouze et al., 2017; McLeod et al., 2007; Wood et al., 2003). Another point worth noting is that our behavioral factors at the three time-points were based on three age-appropriate tasks and may have tapped different aspects in the maternal behavior. Moreover, most studies on parenting behavior and anxiety in children used questionnaires to evaluate parenting, typically reported by the parent (Wood et al., 2003; Yap et al., 2014). Observational data may differ from parental self-reports and some studies found weak correlations between the two approaches (Unternaehrer et al., 2019); hence, our findings may highlight a distinct aspect in the associations between parenting and anxiety disorders in children. Although most of the aforementioned studies used different observational tools from the one used here (Hudson & Rapee, 2001), studies employing similar observational paradigms reported similar results on the associations between children's anxiety and maternal behavior (Eley et al., 2010) or between maternal sensitivity in infancy and separation anxiety at 6 years (Dallaire & Weinraub, 2005). Taken together, these findings suggest that children's anxiety may play a role in shaping, or at least augmenting, parental practices that are less adaptive and further exacerbate the child's anxiety.

Our model did not show any between-person effects of maternal behavior on child anxiety as well as no within-person effects of maternal sensitivity on child anxiety across the study period. These findings are inconsistent with studies that highlight the mediating role of maternal sensitivity on young children's anxiety (Mount et al., 2010; Penela et al., 2012; Warren & Simmens, 2005). On the other hand, our results accord with research that did not identify a specific role for parenting behavior in the etiology of anxiety disorders in childhood (Rapee et al., 2009) and with the inconsistent associations found between parental warmth and children's anxiety (McLeod et al., 2007; Wood et al., 2003). Although in our sample maternal sensitivity did not predict children's anxiety, high maternal anxiety and low sensitivity in early childhood were inter-related and these two features characterized the war-exposed mothers. These findings may suggest that in the context of chronic stress and across the first decade of life maternal psychiatric conditions may play a greater role than the mother's actual caregiving, which may be insufficient to buffer severe maternal symptomatology.

Anxious mothers often respond to their child's vulnerabilities with negative comments and behaviors, which reinforce the child's difficulties and enhance susceptibility to anxiety (Murray et al., 2009). Our models revealed closer associations between child anxiety and maternal sensitivity compared to maternal intrusiveness, despite previous findings that emphasize the effects of parental intrusiveness on the development of child anxiety (Lawrence et al., 2020; Wood, 2006). Several studies have shown that the anxious mother's style is marked by intrusiveness, criticism, negativity, overprotection, lack of encouragement, and over-involvement, and these patterns, in turn, are linked with increased anxiety symptoms in the child (Hudson & Rapee, 2001; Murray et al., 2009; Yap et al., 2014). An additional mechanism that may play a role in the increased susceptibility to anxiety in children of anxious mothers relates to emotion regulation (de Wilde & Rapee, 2008). Adolescents of intrusive parents often show poor emotion regulatory skills (Yap et al., 2014), which render them more vulnerable to the emergence of anxiety disorders that are underpinned by emotion dysregulation (Bosquet & Egeland, 2006).

9.3 | Stability in study variables: Autoregressive pathways

Results from the two models, the CLPM and the RI-CLPM, revealed different findings with regard to stability of the study variables as reflected by the within-person autoregressive paths over time, that is, associations between previous symptoms or behaviors and the future expressions of these same variables. In the traditional CLPM, autoregressive pathways represent the maintenance of rank-order stability within the sample across the study period. However, in the RI-CLPM, the autoregressive pathways describe whether the individual's deviation from the expected trajectory carries over to the next time-point measurement. In our study, the child's anxiety pathway from T1 to T3 and from T3 and T4 was significant in both the CLPM and the RI-CLPM analysis. In comparison, the homotypic pathways for maternal anxiety and for mother sensitivity were less consistent. Although rank order stability in these variables was evident in the CLPM, after accounting for trait-like stability in the RI-CLPM, maternal anxiety showed within-person carry-over effects only from T1 to T3 whereas maternal sensitivity and intrusiveness, which may reflect a trait-like characteristic, did not show significant stability effects. The stability in children's anxiety symptoms may reflect an underlying biological vulnerability for the development of anxiety disorders, which may be driven by genetic, temperamental, and information-processing factors (Murray et al., 2009). These findings accord with studies suggesting that anxiety symptoms are moderately stable throughout life, particularly across childhood and adolescence (Bosquet & Egeland, 2006; Rapee et al., 2009), and with studies indicating that genetic factors may underpin much of the stability in childhood anxiety (Frani et al., 2010). Shared genetic vulnerability to anxiety symptoms may also account for the reported bidirectional associations between parental and child's anxiety (Smoller, 2016). Conversely, a children-of-twins study found that the

transmission of risk for anxiety in adolescence was mediated solely by environmental factors; still, these findings are yet to be replicated in other samples and age-groups (Eley et al., 2015). Further support for the important role of the environment come from adoption studies that show associations between anxiety in adoptive parents and their infants (Brooker et al., 2015) and children (Ahmadzadeh et al., 2019) despite lack of genetic relatedness. This suggests that exposure to anxious parenting may play an important role in both the emergence and persistence of anxiety disorders across childhood and our findings are among the first to show the pervasiveness of children's anxiety symptoms from early childhood to early adolescence and their repeated associations with maternal anxiety symptoms.

9.4 | Limitations and directions for future research

Several study limitations should be acknowledged. First, it is important to emphasize that all terms related to “effects”, “influences”, or “impact” used in this study imply statistical effects, not causal effects, and our findings in no way suggest causality. Another important limitation is the lack of data on father anxiety symptoms and paternal behavior, which could have either mitigated or exacerbated the development of maternal and child's anxiety. We also did not include trauma measures to identify the subjective and objective trauma exposure of each participant and future studies could benefit from collecting more detailed information. Additional limitation relates to the measurements used to assess anxiety symptoms. Child symptoms were measured at T1 using a different measure (CBCL) from the one used in the other time-points (SCARED), as the SCARED is not suitable for toddlers. However, it is acceptable and common to use these tools at the respective ages due to the fact that the two assessment tools are highly correlated (Monga et al., 2000; Weitkamp et al., 2010). Furthermore, children's anxiety scores in late childhood and early adolescence were computed as the average of the child's self-report and the mother's report and thus, mothers reported on both their own and the child's anxiety. Using an average of the two reports is both common (Bar-Haim et al., 2007) and recommended (Wren et al., 2004); nonetheless, we further tested our model with the child report and this showed a good fit, despite the fact that at T1 mothers had to report on the child's symptoms and therefore the shared-method-variance bias was not completely removed. In the child-report model there was no significant association between child anxiety at T3 and maternal anxiety and behavior at T4; still, the pattern of associations was similar to the original model albeit weaker (Model S4 in Supporting Information). The findings that the cross-lagged paths in the mother-report model were similar to the original model suggest that the stability of measures was not impacted by the different informants (model S5 in Supporting Information). Another limitation is the lower sample size at the final time-point, which might have had an impact on the statistical power. Finally, since we only had available data on children's anxiety, we cannot determine whether our model is specific to anxiety or may show similar results for other child disorders.

Still, our study provides a unique perspective on the development of anxiety symptoms in children exposed to chronic stress and trauma. While previous studies examined the associations between parenting behavior and child anxiety as well as the links between parent and child's symptoms, no study to our knowledge addressed these three components together within a longitudinal design that differentiates within- from between-subject variance. Our findings indicate that in the context of chronic trauma maternal anxiety may be an important factor in the development of children's psychopathology and resilience. However, the other direction, from children to parents, appears to present a more dominant effect and the data show that children's anxiety symptoms may trigger the expression of non-optimal parenting across lengthy epochs of development. Our findings on the stability of anxiety from early childhood to adolescence underscore the need for early detection and intervention for families exposed to chronic stressors. Furthermore, results on the cross-generational associations between maternal anxiety and the development of child anxiety may suggest that interventions targeting mothers are needed. It has been shown that maternal involvement in the therapeutic process is crucial for treatment success in late childhood and adolescence (Rapee et al., 2009) and our findings similarly highlight the importance of involving mothers in the intervention effort in the first years of life. Such mother-focused interventions may help mothers to properly identify and adequately regulate her own as well as the child's stress and improve the dyadic capacity to overcome fears, withstand trauma, and enhance resilience.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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