**Supplementary Materials**

**Supplementary sample descriptions:**

Recruitment efforts included posting recruitment flyers at the University Hospitals and Faculties and in community centers, domestic violence agencies and shelters, with the goal to oversample for domestic violence-exposed mothers. We refrained from including fathers, due to the number of mothers living under order of protection and in anonymous shelters. Three dyads that were included had interpersonal violence (IPV)-PTSD symptoms, but also had primary PTSD due to reasons other than IPV. The participants that completed Phase 1 but not Phase 2 (24%) did not differ significantly from the ones that completed both Phases (76%) regarding parent or child age, child gender, socioeconomic status or number of traumatic life events.

Data collection and measures

**Phase 1**. In total, 21 measures from Phase 1 were included in the analysis (see table 2A for description of measures). 19 of these measures included data on maternal psychopathology (5 measures), experienced abuse during childhood (3 measures) as well as parental behaviors, skills and tendencies (11 measures). All measures were validated and possess good to very good psychometrics (Table 2). They included the clinician-administered PTSD scale for severity to measure lifetime PTSD (CAPS; 1, the PTSD Checklist Scale (PCL-S) to measure current PTSD symptoms at Phase 1 2,3). Potentially comorbid maternal psychopathologies (depression 4 and psychological dissociation 5) were assessed using standard measures. Further, physical and sexual abuse during childhood and exposure to traumatic violence during adulthood were measured using the Brief Physical and Sexual Abuse Questionnaire BPSAQ; 6 and the Traumatic Life Events Questionnaire TLEQ; 7 to supplement questions on traumatic life events not covered by the BPSAQ 8. Parental skills and behaviors: PRF behaviors were assessed using coding of the Working Model of the Child Interview9,10 and maternal behavior was coded from mother-child interaction videos using the CARE index11, with specific operationalizations further described in previous studies 12,13.

Additionally, socio-economic status and child sex were included, using the Geneva Sociodemographic Questionnaire14. While these were not predictors of interest, this approach allowed us to quantify their impact on results compared to other measures rather than having to regress out their effect and thereby altering the data set. We wanted to avoid the latter, as these factors are known to be related to some of the other input or outcome measures.

**Phase 2**. At Phase 2, 25 measures were included (19 in analysis 1), as shown in Table 1B. We extracted five symptom measures from the Kiddie Schedule for Affective Disorders and Schizophrenia - epidemiologic version (K-SADS 15,16).We counted the number of DSM-V symptoms for the following: depression, anxiety, attention deficit hyperactivity disorder (ADHD), post-traumatic stress, and behavioral disorders. We investigated children’s violent-experiences via the Violent Experiences Scale (VEX-R) 17. We administered the School-Life Survey 18 to assess bullying perpetration and victimization. Further, children took the Test for Emotional Comprehension 19 and we assessed child mental representations via the MacArthur Story Stem Battery MSSB; 20, based on 5 stories-stems (8 subscales/story).

In addition, for analysis 2, mothers reported on child internalizing and externalizing symptoms using the Child Behavior Checklist CBCL; 21,22 and on child temperament via the School Age Temperament Inventory 23.

**Supplementary description of analysis:**

Before we z-standardized sCCA, we replaced missing data with 0-values (i.e. mean values). For sCCA, we used a Matlab script available online 24 that applies an approach with an L1-norm penalty 25.

For either dataset the computed range of candidate values was from 0.5 × √P (high sparsity) to 1 × √P (low sparsity) at increments of 0.1, where P is the number of variables in each dataset. For each analysis, we automatically selected the combination of sparse criteria of the analysis with the maximum sCCA correlation value among all tested sparse criteria combinations. We determined sCCA significance using permutations. Accordingly, the Phase 1 dataset was permuted 10’000 times before undergoing the exact same analysis as the original data in each permutation.

The p-value represents the number of permutations with a higher correlation than the original data divided by 10’000 using all the sparsity combinations available to the main analysis. Consequently, the p-value is compared against the null distribution of maximal correlation values across all estimated sCCAs and in this sense corrected for multiple testing. For significant sCCA, we report the weights of each variable for both Phase 1 and Phase 2 datasets in order to assess their contribution and present those above 0.2 in figures.

Apart from the first mode, we tested an additional 6 modes (component pairs) for significance and reliability with 2000 permutations each. None of these modes proved to be significant.

**Reliability analyses and results:**

To assess whether our results were robust and reliable, we proceeded in five steps of reliability analyses (1) we first performed leave-one out analysis for every participant. (2) We then computed a redundancy-reliability score (Moser’s RR-score, mean and SD) for each significant sCCA 26. Moser’s RR-score is a measure of the stability of the variable-to-variate correlations and indicates whether results can be expected to be reliable independent of sample composition. Meant to be based on a training-test set approach, Moser’s RR-score essentially measures whether test sets have similar associations between variables and variates, whereby results with high RR-scores may be assumed to be truly carried by the entire sample and not to depend on a specific subset of the population that may not be reliably reproduced if one were to replicate the study 26. In the present study, we performed 10’000 splits of training and test sets in order to calculate the mean RR-score (3). We performed a cross validation approach to test whether the weights gained from sCCA in one-half of the sample would reliably lead to a correlation in the other half of the sample. For this, we randomly resampled half the sample 10’000 times (training sets), performed sCCA on each of these sets and then applied the identified weights from each training set to the other half of the sample (the test set). This allowed us to gain information on whether sCCA derived correlations in this study were likely to be dependent on this specific sample or not. (4) We repeated the analysis regressing out maternal age at Phase 1 and child age at Phase 2, to see if results would be impacted. (5) We repeated the analysis by transforming each variable into ranks rather than using original interval scale (prior to standardization), to ascertain again that it was not a combination of multiple outliers together that were responsible for the results.

1) Leave-one out analysis demonstrated that neither analysis was dependent on any single participant, as weights correlated at r>0.97 for either dataset in either analysis if any participant was removed from analysis.

2) The mean Moser’s RR-scores were 0.70 (SD = 0.16) for analysis 1, and 0.77 (SD = 0.13) for analysis 2. Distribution was roughly as could be expected and appeared unimodal and skewed towards the right (higher values, see supplementary Figure 1).

3) Mean correlation of test sets using weights gained from their respective training sets was r = 0.23 for analysis 1 and r = 0.35 for analysis 2. Again, the average distribution appeared unimodal and skewed towards the right (higher values, see supplementary Figure 1).

4) Regressing out age changed very little for either analysis (analysis 1 p = 0.021, correlation with original weights r > 0.97, analysis 2 p = 0.004, correlation with original weights r > 0.99, see supplementary Table 1).

5) Similarly, transforming the data to ranks prior to analysis did not change the result (Analysis 1 p = 0.025, correlation with original weights r > 0.99, Analysis 2 p = 0.004, correlation with original weights r > 0.99, see supplementary Table 1).

Supplementary Table 1A

Weights of individual measures for Analysis 1. Maternal Measures of Phase 1

|  |  |
| --- | --- |
|  | Weights |
|  | Main analysis | Age regressed  | Measures converted to ranks |
| Current PTSD (PCLS) | 0.39 | 0.37 | 0.38 |
| Lifetime PTSD (CAPS) | 0.36 | 0.36 | 0.34 |
| Depression (BDI) | 0.35 | 0.38 | 0.38 |
| Child Witness of Violence | 0.33 | 0.32 | 0.32 |
| Dissociation (Hopkins) | 0.32 | 0.27 | 0.33 |
| Parenting Stress Index: Parental Distress  | 0.31 | 0.37 | 0.32 |
| Socio Economic Status | 0.23 | 0.18 | 0.21 |
| Alexithymia: Difficulty Identifying Feeling | 0.18 | 0.17 | 0.19 |
| Childhood Sexual Abuse (BPSAQ) | 0.17 | 0.18 | 0.16 |
| Adult Partner Violence (BPSAQ) | 0.16 | 0.14 | 0.15 |
| Controlling Maternal Behavior (Crittenden) | 0.16 | 0.16 | 0.17 |
| Parenting Stress Index: Parent Child Dysfunctional Interaction | 0.14 | 0.12 | 0.15 |
| Parent is Drug/Alcohol Abuser | 0.11 | 0.05 | 0.09 |
| Alexithymia: Difficulty Describing Feelings | 0.11 | 0.13 | 0.09 |
| Parenting Stress Index: Difficult Child  | 0.10 | 0.11 | 0.13 |
| Parental Reflective Functioning | 0.08 | 0.10 | 0.09 |
| Alexithymia: Externally-Oriented Thinking | 0.03 | 0.07 | 0.03 |
| Unresponsive Maternal Behavior (Crittenden) | -0.04 | -0.03 | -0.03 |
| Childhood Physical Abuse (BPSAQ) | -0.12 | -0.07 | -0.12 |
| Sensitive Maternal Behavior (Crittenden) | -0.13 | -0.16 | -0.16 |
| Sex Child (higher = more boys) | -0.14 | -0.19 | -0.14 |

Supplementary Table 1B

Weights of individual measures for Analysis 1. Child related Measures of Phase 2

|  |  |
| --- | --- |
|  | Weights |
|  | Main analysis  | Age regressed out  | Measures converted to ranks |
| PTSD symptoms (KSADS) | 0.55 | 0.54 | 0.52 |
| Anxiety Disorders Symptoms (KSADS) | 0.38 | 0.38 | 0.37 |
| Bullying: Perpetration | 0.38 | 0.36 | 0.44 |
| ADHD symptoms (KSADS) | 0.28 | 0.24 | 0.26 |
| Bullying: Victimization  | 0.27 | 0.23 | 0.27 |
| Depression symptoms (KSADS) | 0.25 | 0.22 | 0.22 |
| MacArthur: Negative Parents : Harsh | 0.22 | 0.23 | 0.22 |
| Emotion Comprehension: Mental understanding | 0.11 | 0.14 | 0.12 |
| Behavioral disorders Symptoms (KSADS) | 0.02 | -0.04 | 0.02 |
| MacArthur: Dissociative strategies | 0.01 | -0.03 | -0.01 |
| MacArthur: Aggression | 0.00 | -0.04 | 0.02 |
| MacArthur: Atypical Negative Behaviors | 0.00 | -0.05 | -0.04 |
| Report of physical and nonphysical exposure to violence and crime (VEX) | -0.04 | -0.06 | -0.05 |
| Emotion Comprehension: Reflective Capacities | -0.11 | -0.11 | -0.09 |
| Emotion Comprehension: External Emotions Understanding | -0.12 | -0.09 | -0.10 |
| MacArthur: Avoidance | -0.15 | -0.17 | -0.22 |
| MacArthur: Negative Endings | -0.15 | -0.24 | -0.15 |
| MacArthur: Danger | -0.16 | -0.20 | -0.18 |
| MacArthur: Negative Parents : Ineffectual | -0.19 | -0.22 | -0.17 |

Supplementary Figure 1. Reliability analyses for Moser’s RR-score (left) and cross validation (right) depicting distribution across 10’000 resampled subsamples for both analyses.



**Power Analysis and results:**

In order to assess statistical power, we performed Monte Carlo style power analysis creating two artificial datasets with 62 participants each and 21 and 19 variables, respectively (as in analysis 1) and 25 and 19 variables (as in analysis 2) Through use of an in-house matlab script. For said script we employed the “*sde\_correlate*” function of the SDE master toolbox for matlab 27, to vary correlations between these datasets. We set some correlations between datasets to r=0.4, a bigger number to r=0.3 and yet another one to r=0.2; performed 9 sub analyses, that varied the percentage of said correlations from 0 (sanity check) to 12% for r=0.2 from 0 to 3.2% for correlations at r=0.3 (and from 0 to 0.8 % for correlations at r=0.4 (see also supplementary Table 2). For each of these sub analyses, we created 100 independent datasets with correlations, according to sub analysis randomly distributed, and with 1000 participants. We then drew ten times random subsamples of 62 participants leading to 1000 subsamples overall. We then performed sCCA with the same parameters as in the original analysis for each of the created subsamples. We assessed each subsample for significance, using 1000 permutations. For each sub analysis, we report the percentage of datasets where the sCCA was significant at p<0.05 and the average significance.

Power analysis indicated that 0 Correlations were just above 5% probability of reaching significance with sparse canonical correlation analysis (sCCA), which may be due to overfitting, which in turn was addressed appropriately here with additional reliability measures. Once 9% of associations between variables of the two datasets were at r=0.2, 1.8% at r=0.3 and 1.2% at r=0.4, 82%of results for analysis 1 (88% for analysis 2) would be significant with an average significance level of p= 0.033 for analysis 1 (p = 0.025 for analysis 2, see Supplementary Table 2). Given our data (where 0.7% of all correlations between datasets of analysis 1 were at r>0.4, a further 3.5% were at r>0.3 and another 13.8% at r>0.2), we estimate that power was more than adequate to find an existing significant effect.

Supplementary Table 2. Power analysis following Monte Carlo style simulation.

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Percent r>0.2r>0.3r>0.4 | 000 | 1.50.40.1 | 30.80.2 | 4.51.20.3 | 61.60.4 | 7.52.00.5 | 92.40.6 | 10.52.80.7 | 123.20.8 |
| Analysis 1Percent Significant  | 7.1 | 15.9 | 27.6 | 42.1 | 60.8 | 72.1 | 81.9 | 91.6 | 94.4 |
| Average significance | 0.426 | 0.311 | 0.223 | 0.147 | 0.088 | 0.060 | 0.033 | 0.016 | 0.011 |
| Analysis 2Percent Significant  | 6.3 | 16.5 | 29.6 | 46.5 | 63.9 | 77.4 | 87.7 | 90.5 | 95.1 |
| Average significance | 0.430 | 0.311 | 0.216 | 0.138 | 0.079 | 0.045 | 0.025 | 0.017 | 0.010 |

**Supplementary Discussion:**

Morelen, Shaffer, Suveg 28 found that poor maternal emotion regulation in the context of a history of maltreatment was associated with insufficiently sensitive parenting and impaired development of child emotion regulation. Another study29 showed that the maternal PTSD symptom severity was positively associated with child PTSD symptom severity within-dyads. Both of these studies focused on how maternal PTSD potentially impacted discrete, selected child outcomes. The present study, by contrast, examined the potential influence of a group of several different maternal measures on a group of several different child outcomes simultaneously. This study took into consideration the multivariate complexity of the association(s) between IPV-exposed mothers’ psychopathology and the latter’s consequences on the caregiving environment and thus on their offspring beginning during early sensitive periods of social-emotional development. The results showed important links between maternal and child measures emphasizing and demonstrating the value of applying integrative statistical approaches, such as canonical correlation analyses that focus both on parents and children simultaneously.

We found that harsh parental responses to challenges represented in children’s story-stems was also on the dimension of child outcomes in analysis 1, together with clinician-rated child bullying and psychopathology. This finding corroborates disturbances in parent-child interaction that we observed in Phase I 30. It is possible that such representations in children’s stories are an indicator of children seeing harsh behaviors as part of an over-generalized response by self and others to particularly stressful moments. In the present sample, parental pathology that was associated with interpersonal violence appears to predispose parents to behaving harshly, to bullying and victimizing both within the parental couple as well as behaving insensitively toward their child. Such challenging moments provide frequent opportunities for the child to develop social emotional representations of addressing conflict tinged with these behaviors.

We believe that one strength of our study is that we did not regress out potential confounders, such as SES, child sex and exposure to violence; but rather, we introduced them into the general model. The latter allowed us to quantify their importance in the model and contribution to the overall dimension. Depending on the analysis, potential confounders such as SES, child sex and the child’s exposure to violence indeed make an important contribution to the model; yet this contribution is relatively less than that of maternal overall psychopathology, stress, and of maternal exposure to violence. Adding variables of maternal report of child symptoms and temperament did not fundamentally change the relationships found between mother and child dimensions yet did raise additional issues to consider. Firstly, on the predictors’ side, this second model focused less on classic symptoms of psychopathology and socioeconomic status, and instead more on perceived maternal stress, as well as sex (more importance on girls). Secondly, on the children’s side, this higher focus on maternally perceived problems in Phase 1 still related to a broad spectrum of child symptoms related to violence, but deemphasized bullying and PTSD symptoms. This higher focus on maternally perceived problems in Phase 1 also deemphasized the child’s mental representations of his/her parents, while adding maternal perceived internalizing and externalizing symptoms, as well as child temperament of negative reactivity and task persistence, as reported by the mother, to the model. In other words, the maternal and child/clinician reported symptoms still showed convergence onto a single child outcome dimension. Nevertheless, maternal report was also - even across time – linked not only to objective child symptoms, but also to maternal stress and perception of both herself and her child’s symptoms and temperament. The longitudinal nature of our study points to the importance and long-term consequences that such maternal perceptions can have upon the child and on the mother-child relationship. It is therefore essential for clinicians and affected families to address these posttraumatic perceptions with affected mothers both as individual adults, and in their roles as parents in terms of their relationship with their child.

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