Journal of Psychiatric Research 177 (2024) 305–313

Contents lists available at ScienceDirect

Journal of Psychiatric Research

journal homepage: www.elsevier.com/locate/jpsychires

A biomarker of brain arousal mediates the intergenerational link between maternal and child post-traumatic stress disorder

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ARTICLE INFO

Keywords:
Resting EEG
Aperiodic slope
Spectral power
Maternal PTSD
Neurodevelopment
Intergenerational

ABSTRACT

This study examined whether there is a biological basis in the child's resting brain activity for the intergenerational link between maternal interpersonal violence-related posttraumatic stress disorder (IPV-PTSD) and child subclinical symptoms. We used high-density EEG recordings to investigate the resting brain activity in a sample of 57 children, 34 from mothers with IPV-PTSD, and 23 from mothers without PTSD. These children were part of a prospective, longitudinal study focusing on the offspring of mothers with and without IPV-PTSD, reporting how the severity of a mother's IPV-PTSD can impact her child's emotional regulation and risk for developing mental illness. However, we had not yet looked into potential EEG biomarkers during resting state that might mediate and/or moderate effects of maternal IPV-PTSD severity on child mental health, and in particular the risk for PTSD. The alpha band spectral power as well as the aperiodic exponent of the power spectrum (PLE; power-law exponent) were examined as mediators of maternal IPV-PTSD and child PTSD.

While there was no difference in alpha spectral power between the two groups, PLE was significantly reduced in children of mothers with IPV-PTSD compared to control children, indicating cortical hyper-arousal. Interestingly, child PLE was negatively correlated with the severity of maternal IPV-PTSD, suggesting an intergenerational interaction. This interpretation was reinforced by a negative correlation between child PLE and child PTSD symptoms. Finally, causal analyses using structural equation modelling indicated that child PLE mediated the relationship between maternal PTSD severity and child PTSD. Our observations suggest that maternal IPV-PTSD has an intergenerational impact on the child neurobehavioral development through a correlated abnormal marker of brain arousal (i.e. child PLE). These findings are potentially relevant to psychotherapy research and to the development of more effective psycho-neurobehavioral therapies (i.e. neurofeedback) among affected individuals.

1. Introduction

Interpersonal violence (IPV) affects large numbers of women and children with increasing numbers since the Covid-19 pandemic (van Koppen et al., 2023). We define IPV as physical and sexual abuse and assault, experienced or witnessed from childhood onwards (WHO, 2020,

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https://doi.org/10.1016/j.jpsychires.2024.07.034
Received 29 April 2024; Received in revised form 20 June 2024; Accepted 22 July 2024
Available online 23 July 2024

2021).

Chronic exposure to IPV beginning early in life is associated with increased psychopathology, notably post-traumatic stress disorder (PTSD), depression, suicidality, borderline personality disorder, dissociation and substance abuse (Afifi et al., 2009; Dunn et al., 2020; McLaughlin and Lambert, 2017; Nelson et al., 2020; Russotti et al., 2021). Research suggests that IPV-related PTSD often affects the early mother-child relationship during formative development of emotion regulation, and is associated with subsequent psychopathology in school-age children and adolescents (Esteves et al., 2020; Glaus et al., 2022; Greene et al., 2020; Schechter et al., 2011). The psychobiological mechanisms by which intergenerational transmission of violence is made more likely have only been partially elucidated (Lunnemann et al., 2019; Martin et al., 2002). Several studies have pointed to maternal psychological dysfunction due to difficulty in maintaining mutual emotion-regulation or an "affiliative" mode due to the child and/or other environmental or internal factors triggering the mother to feel threatened, and thus dysregulated psychophysiologically. She thus is often seen by the infant in a fearful, hypervigilant state, which compromises her emotional availability. And as the infant learns that he/she cannot predict when his/her mother will be emotionally available, fearful or frightening, the infant also becomes hypervigilant to the mother's emotional communication. And so, one can speak of a transmission of hypervigilance related to maternal IPV-PTSD (Aktar et al., 2019; Lang and Gartstein, 2018).

Among the possible methods to explore psychobiological mechanisms underlying intergenerational transmission of hypervigilance and other violence-related effects, electroencephalography (EEG) has the advantage of a high (i.e. sub-second) temporal resolution for examining fast large-scale brain dynamics (Butt et al., 2019; Palmwood et al., 2022; Weon et al., 2021). Several EEG biomarkers have been reported in patients with PTSD, including abnormal cortical oscillations, event-related potentials, and microstates (Nicholson et al., 2023; Perizzolo Pointet et al., 2020; Perizzolo et al., 2019; Shim et al., 2017; Terpou et al., 2022). Of these, one of the more replicated findings across studies is a signature of reduced alpha power at rest (Clancy et al., 2017; Nicholson et al., 2023; Ros et al., 2017). Given that a number of previous studies have linked states of low alpha power with increased cortical metabolism and/or excitability (Mathewson et al., 2011; Romei et al., 2008), this hints at the possibility that patients with PTSD might exhibit a state of cortical hyperarousal even during "rest". Pertinent EEG studies examining child physical and/or sexual abuse reported significantly attenuated alpha power at rest (Howells et al., 2012; Nicholson et al., 2023) as well as significant correlations with hyperarousal severity (Ros et al., 2017). Interestingly, alpha power has also been shown to be inversely linked with the 1/f slope of the EEG power spectrum, which is another promising marker of cortical activation and disinhibition (Lendner et al., 2020; Ostlund et al., 2021). The resting-state EEG power spectrum is composed of both oscillatory (i.e., periodic) and non-oscillatory (i.e., aperiodic) activity (Donoghue et al., 2020). The aperiodic activity, whose slope is represented by the power-law exponent (PLE), may be considered as the "baseline" on top of which cortical rhythms with well-defined peaks (e.g. alpha at 10 Hz) are superimposed (see Supplementary Material Fig. S1). Physiologically, both alpha oscillations and the PLE have been reported to reflect the excitatory/inhibitory (E/I) balance in cortical circuits, being lower when $E > I$ (i.e. during neural activation) and larger when $E < I$ (i.e. during neural deactivation) (Gao et al., 2017; Muthukumaraswamy and Liley, 2018; Podvalny et al., 2015).

Despite the extensive research on adult patients with PTSD, there is little agreement in the literature regarding clear EEG markers of the clinical diagnosis. This is likely due to the diverse clinical presentations, the variability of the samples studied, and the lack of replication given the divergent methods used (Butt et al., 2019). In line with the concept of Research Domain Criteria or "RDoC" (Insel et al., 2010), focusing on biological markers of a specific dysfunction linked to characteristic symptoms or symptom clusters is expected to be more clinically useful than markers of current psychiatric diagnoses like PTSD (Stover and Keeshin, 2018). One example of this is the likely connection between reduced alpha power unilaterally in right parietal brain areas and hypervigilance to external stimuli (Benedek et al., 2014). Pertinent to the current study, one study found that frontal alpha asymmetry moderated the relationship between inner city mothers' childhood history of trauma and their young children's externalizing symptoms (van de Ven et al., 2020). Despite this one example, very little has been published characterizing the electrophysiological activity of children with PTSD and/or offspring of mothers suffering from IPV-PTSD. One study found that left, rather than right lateralized alpha asymmetry, was associated with child trauma exposure as a risk factor for later PTSD (Im et al., 2022). In a previous event-related evoked potential study, it was found that abused children with PTSD showed a significantly larger increase in the P2–N2 ERP component compared to abused children without PTSD when exposed to increasingly intense aversive auditory stimuli. This increase in ERP component was related to reexperiencing symptoms and hypervigilance symptoms (McPherson et al., 1997).

Our previous results from the longitudinal study showed that mothers with IPV-PTSD influenced their children's emotional assessment of negative emotions, as measured by EEG during an Emotional Face Matching Task (Perizzolo et al., 2019). The severity of maternal IPV-PTSD was linked to hypervigilance towards angry facial expressions, as previously observed (Shackman and Pollak, 2014). Children of IPV-PTSD mothers showed greater energic resources in processing facial expressions compared to non-PTSD mothers' children. Additionally, children of IPV-PTSD mothers exhibited decreased neural activity in specific brain areas in response to negative emotional expressions. These findings indicate similar emotion processing among individuals exposed to maltreatment (Doretto and Scivoletto, 2018).

While these aforementioned results are related to response to visual stimuli, no previous study has been published on resting-state EEG of children from mothers specifically with IPV-related PTSD, nor on children with histories of maltreatment and/or PTSD. Based on the link between cortical arousal and PTSD symptomatology described above, EEG alpha power and PLE are potentially relevant functional biomarkers of intergenerational transmission of violence, related psychopathology and corresponding brain signatures. In the present study, we aimed to investigate whether a child's resting state EEG could potentially mediate the impact of maternal IPV-PTSD on the child's psychopathology. Our primary hypothesis was to discover a link between the severity of maternal IPV-PTSD, the child's PTSD, and the child baseline alpha spectral power and/or PLE. To our knowledge, there are no existing studies that have examined the effects of maternal IPV-PTSD on child psychopathology using EEG-spectral analysis methods.

2. Material and methods

2.1. Participants

Mothers and their children were included in the Geneva Early Childhood Stress Project (GECS-Pro) Phase 2 if they already participated at Phase 1 of the study. During Phase 1 of the project, both groups (IPV-PTSD clinical group and non-PTSD control group) were created according to cutoff criteria on the Clinician Administrated PTSD Scale (CAPS) and the Posttraumatic Symptom Checklist— Short Version (PCL-S): $\geq$55 on the CAPS and $\geq$40 on the PCL-S (Moser et al., 2015).

Mother-child dyads were originally recruited for Phase 1 via flyers advertising a study on stress associated with parenting a young child ages 12–42 months that were posted at the university medical center and psychology departments, and in the general community (i.e. at shopping centers, grocery stores, daycare and preschool programs, and community centers). In order to assure sufficient participation by domestic violence affected mothers, presentations and flyers were offered to the teams of agencies aiding families affected by domestic violence in

Geneva. As an unexpected result, the study recruited an over-representation of mothers who had experienced domestic violence and suffered from related PTSD. The Non-PTSD control group included mothers exposed and not exposed to domestic violence; while 15 mothers who had significant PTSD symptoms due to non IPV events such as medical-surgical trauma or birth-related trauma, natural disasters, and car accidents, were excluded from the study since there was an insufficient number to create a separate control group. Given the over-sampling for mothers with adult exposure to domestic violence, all mothers who had PTSD had experienced at least physical and/or sexual assault over the age of 16 with 70% of mothers having experienced additionally childhood physical and/or sexual abuse and/or exposure to domestic violence (Schechter et al., 2015).

In the present study, children were aged 5–9 years and mother-child dyads were excluded if mothers were actively substance-abusing, suffered from psychotic disorders or if children or mothers were physically and/or mentally impaired in such a way as to render them unable to participate in all research tasks. The institutional ethics committee at the Geneva University Hospitals and Faculty of Medicine approved this research project in accordance with the Helsinki Declaration (World Medical Association, 1999).

Fifty-eight children (34 boys and 24 girls) and their mothers participated following informed consent. Thirty-four of which were children of mothers with IPV-PTSD (mean age = 6.99 years, SD = 1.02). Whereas the non-PTSD controls group comprised of 24 children (mean age = 7.14, SD = 1.15). Resting state EEG recordings were successfully obtained in 57 participants (34 children of IPV-PTSD mothers and 23 children of non-PTSD controls), after exclusion of one participant in the control group due to poor data quality. No children suffered from traumatic brain injury or neurological disorders. There were no statistical differences between groups in age (p = 0.46) or gender (p = 0.86) (see Table 1).

2.2. Clinical assessment

Children and mothers came for two 3-h-long visits on separate days and EEG was recorded during the second visit. During both sessions, mothers and children were assessed by a clinician for maternal and child psychopathology, and maternal-child life-event histories from Phase 1 were updated. Demographic and clinical characteristics are presented in Table 1.

2.2.1. Maternal psychopathology

Since Phase I of the study was from 2010 to 2014 and Phase 2, from 2014 to 2018, with the need for validated French translations of all structured clinical interview materials, the DSM-IV-R criteria for psychopathology were used. Maternal IPV-PTSD was assessed using the Clinician Administered PTSD Scale for the DSM-IV-TR (CAPS (Blake et al., 1995);) and the PTSD Checklist for the DSM-IV-TR- Short Version (PCL-S; (Weathers et al., 2001) during Phase 1 (Schechter et al., 2017). The CAPS assessed lifetime PTSD and the PCL-S assessed current PTSD (Blake et al., 1995). CAPS items had Kappa coefficients higher than 0.63; internal consistency for all CAPS items resulted in a Cronbach's alpha coefficient of 0.97 (Pupo et al., 2011). The PCL-S is similarly a highly reliable measure (Lima et al., 2012) characterized by Cronbach's alpha coefficients that indicate high internal consistency for the total scale (0.91) and for re-experiencing, avoidance, and hyper-arousal symptom clusters as delineated in the DSM-IV (0.83, 0.81, and 0.80) (American Psychiatric Association, 2000).

Mothers who met criteria for DSM-IV-TR PTSD full diagnosis (PCL-S score ≥40 and CAPS Score ≥55) or demonstrated clinically significant symptoms (PCL-S score >25 and CAPS score >35) were included in the same IPV-PTSD group. The second group contained non-PTSD mothers with and without trauma exposure(s). Mothers with PTSD due to reasons other than IPV were excluded from analyses.

Maternal depression was assessed at Phase 1 using the Beck Depression Inventory BDI II (Beck and Steer, 1987), as major depressive disorder (MDD) is often comorbid with PTSD (40–50% of cases (Flory and Yehuda, 2015)). Maternal MDD symptom severity was controlled in our analyses, given the observation that in infants under the age of 6-months, when MDD and PTSD are comorbid as they are generally in 40–60% of women who have IPV with related psychopathology, it is the MDD rather than PTSD that leads to the most deleterious impact on maternal behavior (Muzik et al., 2017).

Table 1
Demographic and clinical variables of IPV-PTSD and non-PTSD mothers and their children. Mean (standard deviation). BDI: Beck Depression Inventory, CAPS: Clinician Administrated PTSD Scale, CBCL: Child Behavior Checklist, IPV: Interpersonal Violence, K-SADS: Schedule for Affective Disorders and Schizophrenia for School-Age Children, PTSD: Post-Traumatic Stress Disorder, VEX-R: Violent Exposure Scale for Children-Revised. P-values from non-parametric Mann Whitney *U* test.

		All	Controls (n = 23)	IPV-PTSD (n = 34)	p-value
Maternal variables	Age of mothers in years	39.64 (5.71)	39.78 (5.53)	39.55 (5.92)	0.45
	Socio-economic status[a]	4.89 (2.00)	4.30 (1.92)	5.30 (1.98)	0.075
	Depression (BDI)	9.16 (7.47)	4.09 (3.27)	12.59 (7.58)	<0.001
	CAPS total score	55.15 (34.71)	19.60 (7.56)	81.29 (20.35)	<0.001
Child variables	Age of children in years	7.08 (1.09)	6.99 (1.02)	7.14 (1.15)	0.32
	% boys	57.9 (33/57)	56.5 (13/23)	58.8 (20/34)	0.33
	PTSD (K-SADS)[b]	2.04 (3.07)	0.82 (1.3)	2.97 (3.67)	<0.05
	Anxious – depressed (CBCL)	5.14 (4.42)	4.17 (4.00)	5.79 (4.62)	0.167
	Aggressive behaviors (CBCL)	9.12 (4.97)	7.87 (4.23)	9.97 (5.3)	0.218
	CBCL total score	35.02 (17.5)	27.39 (13.56)	40.18 (18.14)	<0.01
	Exposure to violence (VEX-R)[c]	−0.004 (1.04)	−0.21 (0.96)	0.13 (1.08)	0.337

%: (n/number of valid data).
[a] Socio-economic status: higher scores reflect lower status; 1 value is missing within the PTSD group.
[b] PTSD (K-SADS): 1 value is missing within the control group, 5 values are missing within the PTSD group.
[c] VEX-R: 1 value is missing within the control group, 1 value is missing within the PTSD group.

2.2.2. Child psychopathology

Child psychopathology was generally assessed using the Child Behavior Checklist (CBCL) (Achenbach et al., 1991), completed by mothers. Child PTSD was specifically measured via the PTSD screening module of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS) (Kaufman et al., 1997). The K-SADS is a clinician-rated semi-structured clinical interview of the child with additional information provided by the parent. Children with 2 or more symptoms on the screening are considered to have PTSD symptoms and typically go on to have a full diagnostic assessment using the K-SADS supplementary measure. We selected specifically the K-SADS PTSD module and number of symptoms as a marker of severity to test our a-priori hypothesis concerning intergenerational transmission within this limited sample. Similarly, we used DSM-IV-R criteria because validated DSM-5 clinical interview materials were not yet available at the start of this second phase of the longitudinal study.

The CBCL is a well-validated parent-report measure for ages 1.5–18

years and thus may contain a potential parental bias, despite the validation of parent report for psychopathology, particularly externalizing behavior (Bied et al., 2017). The CBCL was used in the present study to characterize the sample of children of mothers with IPV-PTSD versus without.

Children also completed the clinician-administered Violent Exposure Scale for Children-Revised (VEX-R) (Fox and Leavitt, 1995). The VEX-R is a 21-item self-report measure administered as an interview to the child about violence exposure, validated for children aged 4–10. Items are divided into statements that probe whether the child witnessed and/or experienced a given type of interpersonal violent event, such as, "I saw someone hit someone else hard" and "Someone hit me hard." We examined the continuous score of number of types and frequency of these events witnessed and experienced combined.

2.3. EEG data acquisition and pre-processing

The electroencephalogram (EEG) was acquired for 3 min at resting state with eyes closed. The data were recorded continuously from scalp electrodes using the HydroCel Geodesic Sensor Net and Net Amps 300 amplifier (Electrical Geodesics, Inc., Eugene, OR (Tucker, 1993);). All electrodes were referenced to the vertex electrode (Cz) for recording and then re-referenced offline to the average reference for data analysis. Electrode impedances were kept below 30 kOhms. Electrodes at neck and jaw were excluded, reducing the number of analyzed electrodes to 204. The data were acquired at 1000 Hz sampling rate, and down-sampled offline to 250 Hz.

Data were processed in MATLAB version 2021a with EEGLAB (The MathWorks, Inc.). The following sequence of steps were performed in order to remove artifactual (i.e. non-cerebral) sources of electrical activity that may contaminate EEG recordings (Bailey et al., 2023). Firstly, EEG data was band-pass filtered at 1–80 Hz. Next, the Zapline method was used to remove the top 6 components around the 50 Hz main line frequency (de Cheveigne, 2020). Then, we removed bad channels using EEG lab's PREP plugin (Bigdely-Shamlo et al., 2015) with default settings and interpolated the rejected channels. After which, Infomax ICA was performed and specific ICA components were rejected related to i) eye blinks/movements using the EyeCatch algorithm default settings (Bigdely-Shamlo et al., 2013), and ii) muscle artifacts flagged by ICLabel at >50% probability (Pion-Tonachini et al., 2019). We then automatically removed additional low-frequency artifacts using wavelet ICA at threshold = 10 and wavelet level = 10 (Castellanos and Makarov, 2006). Finally, remaining EEG artifacts were removed epoch-wise with a z-score based method using the FASTER plug-in (Nolan et al., 2010), rejecting 1-s epochs deviating by more than two standard deviations.

2.4. EEG spectral analysis and estimation of the power-law exponent (PLE)

Absolute power spectral density (PSD) for each EEG channel was computed using the Welch method (Matlab function *pwelch*) for frequencies ranging from 2 to 30 Hz. The Hanning window had an effective size of 2 s and 50% overlap. Individual absolute power values were computed in the alpha (8–12 Hz) frequency ranges for all the 204 electrodes (global power) as well as in frontal, central, parietal, temporal and occipital regions (regional power). To obtain relative power estimates, all absolute power values were divided by the sum of the full spectrum (2–30 Hz).

EEG aperiodic activity has a 1/f-like distribution, whose power decreases exponentially across increasing frequencies. The power-law exponent (PLE) reflects the pattern of aperiodic power across frequencies and is equivalent to the negative slope of the power spectrum. For estimating the PLE we used a similar approach to previous studies (Colombo et al., 2019; Griffiths et al., 2019) using open-source code (https://github.com/milecombo/spectralExponent). More specifically, this first involved calculating the absolute power spectral density (PSD) as described above. Then, the following 3 steps were performed in order to estimate the slope (i.e. spectral exponent α) of the background PSD. The PSD background (i.e. non-oscillatory or aperiodic activity) decays approximately according to an inverse power law PSD (f)$\sim 1/f^{\alpha}$. Firstly, a linear regression line was fit to the PSD using log–log axes. Secondly, frequency bins with positive residuals were considered as containing oscillatory activity and thus removed from subsequent analysis. Thirdly, another linear regression line was then fit on the remaining frequency bins (i.e. those consistent with a 1/f behavior). The slope/gradient of this second line was considered as the estimated spectral exponent α of the PSD background. We estimated the PLE across the lower frequency range (2–25 Hz), where EEG signals have the highest signal-to-noise ratio.

2.5. Statistical analyses

Non-parametric Mann Whitney U tests were performed to compare clinical data across groups. Correlation analyses using Spearman's rank correlation coefficients were then performed between maternal PTSD severity (using CAPS total score) and child PTSD and CBCL scores, as well as EEG spectral measures that were statistically significant at the group level (i.e. child PLE).

Statistical significance of group mean differences in electrophysiological measures were computed using permutation tests (10,000 resamples) in Matlab version 2021a (Mathworks Inc). Permutation tests were used to compare the alpha global and regional power, as well as the overall PLE, between children of IPV-PTSD mothers and non-PTSD controls. All group-wise tests were two-tailed, with significance level at 0.05. Furthermore, we used median absolute deviation (MAD) as a robust way to identify outliers for each outcome measure, with a standard cut-off of MAD >3.

Finally, mediation using regression model was applied using Jamovi version 2.3.21 and therein the jamm 1.2.0 libraries GLM mediation module. Maternal PTSD and PLE were z-standardized prior to mediation analysis. If confounding variables were associated with the outcome (child sex, age, maternal depression, socioeconomic status, and child exposure to violence) they were controlled for by entering them into a multiple linear regression model. For significance calculation, the module first performed 5000 bootstraps with the percentile method. Using these bootstraps, the module calculated confidence intervals and used those to calculate the standard error. Given the standard error and the actual estimate it then calculated z values and corresponding p values. Here we report the completely standardized beta, z and p values without the confidence intervals.

3. Results

3.1. Child spectral power analysis according to maternal psychopathology

Group-wise permutation tests between children of IPV-PTSD mothers and non-PTSD control children showed significant global differences in the aperiodic power-law exponent (PLE). Children of IPV-PTSD mothers demonstrated attenuated PLE values (i.e. flatter power spectrum) compared to controls (Cohen's d = −0.67, p = 0.007), suggesting significantly enhanced cortical excitability and arousal (Fig. 1). On the other hand, group analyses of absolute and relative power in alpha frequency band in the children did not reveal any significant differences between maternal PTSD and non-PTSD groups, either globally (204 electrodes) or regionally (5 regions of interest).

3.2. Association between maternal IPV-PTSD severity and child EEG activity

There was a significant negative correlation between maternal PTSD severity, as measured by the CAPS total score, and child PLE (Spearman's rho = −0.31, p = 0.018). On the other hand, no significant

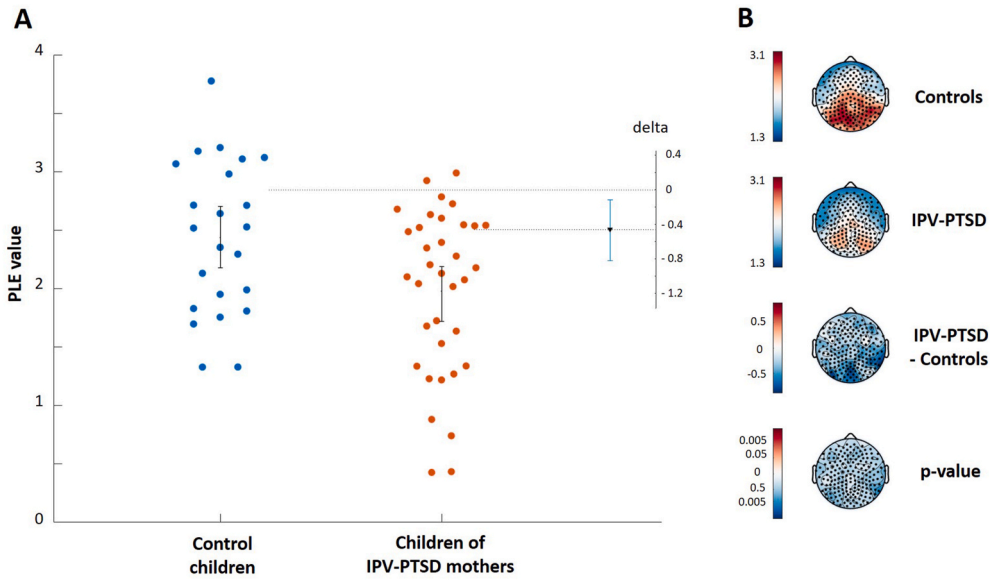

Fig. 1. A. Group strip plots and average PLE values over 204 electrodes in children of IPV-PTSD mothers and control children. On the right, the 'delta value' indicate absolute differences between the group means with 95 % confidence intervals. **B.** Topographic plots of PLE for the children of IPV-PTSD mothers and control children, and unpaired permutation tests (uncorrected, $p < 0.05$).

correlation between maternal PTSD severity and child alpha band absolute or relative power was found.

3.3. Association between maternal IPV-PTSD severity and child psychological symptoms

As noted in Table 1, the total CBCL score significantly differs between maternal PTSD vs non-PTSD groups; yet the aggressive and anxious-depressed subscales individually do not. Children with PTSD symptoms typically show a mix of externalizing and internalizing symptoms (Dehon and Scheeringa, 2006). Even with this significant difference between the 2 groups for the total CBCL score, the PTSD mothers' children are below the clinical threshold of 60 on the CBCL.

Considering potential intergenerational transmission of risk for child psychopathology, the association between mother and child PTSD was studied. The child PTSD module of the K-SADS was examined with respect to the child violent life-events on the VEX-R using crosstabs. The results showed that 10 out of 27 children from mothers with PTSD had 2 or more PTSD symptoms, and all had witnessed and/or experienced IPV; only 3 out of 22 children from mothers without PTSD had at least 2 PTSD symptoms, and all had experienced IPV. The total VEX-R severity score, taking into account the number of different types of IPV witnessed and experienced as well as the frequency of occurrence or number of repetitions of IPV events, was significantly correlated with the number of child PTSD symptoms (Spearman's rho = 0.40, p = 0.004). Moreover, there were significant positive associations between maternal PTSD severity and the number of child PTSD symptoms measured using the K-SADS (Spearman's rho = 0.38, p = 0.006), the child total CBCL score (Spearman's rho = 0.37, p = 0.005), as well as the anxious-depressed CBCL subscale score (Spearman's rho = 0.27, p = 0.04).

3.4. Association between child PTSD symptoms and EEG activity

Significant negative association was found between child PTSD and child PLE (Spearman's rho = −0.32, p = 0.024), as illustrated in Fig. 2.

3.5. Child PLE mediation model between maternal PTSD severity and child PTSD

We created a post hoc model to examine whether the number of child

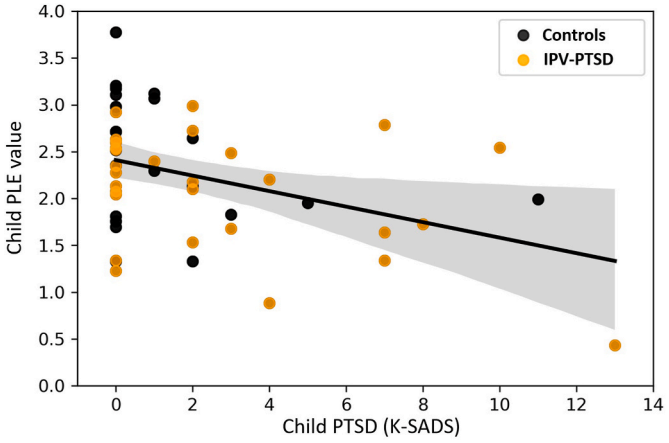

Fig. 2. Significant pooled correlation (Spearman's rho = −0.32, p = 0.024) between child PLE and child PTSD symptoms, in children of IPV-PTSD mothers (orange) and control children (black).

PTSD symptoms as a marker of severity significantly related to both maternal PTSD and child PLE based on Spearman's correlation results. Using child PLE as mediator, the mediation model included maternal PTSD severity (as independent variable) and child PTSD at the K-SADS (as dependent variable). It indicated a total effect of maternal PTSD severity on child PTSD severity (Fig. 3, beta = 0.36, z = 2.72, p = 0.007). However, neither the direct (beta = 0.26, z = 1.61, p = 0.11) nor the mediation (beta = 0.12, z = 1.46, p = 0.14) pathway were solely responsible for this effect. Within the mediation pathway, both components were significant by themselves individually (component 1: beta = −0.37, z = −2.94, p = 0.003, component 2: beta = −0.33, z = −2.00, p = 0.046). We then controlled for child age, sex and child violence exposure as measured by the VEX-R. Controlling for these factors altered the pattern only slightly, and did not do so significantly: the total effect remained significant (beta = 0.31, z = 2.57, p = 0.010) and both direct (beta = 0.21, z = 1.48, p = 0.14) and mediation (beta = 0.09, z = 1.26, p = 0.21) pathways remained below significance threshold. However, only the second component of this pathway (child PLE to child PTSD) lost significance through inclusion of covariates (component 1: beta =

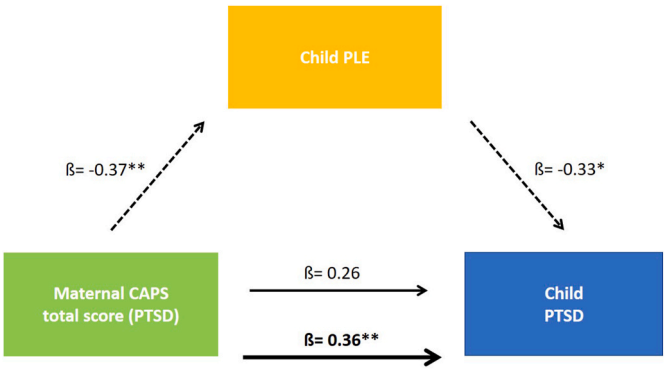

Fig. 3. Model showing maternal PTSD severity and child PTSD being mediated by child EEG power-law exponent (PLE). Bold arrow: total effect (combining direct and mediation effects), plain arrow: direct effect, dashed arrows: mediation component effects. β: Beta values (standardized effect sizes); *p < 0.05, **p < 0.01.

−0.38, z = −2.67, p = 0.008, component 2: beta = −0.24, z = −1.68, p = 0.09). In the full model, both child age (beta = −0.42, t = −2.73, p = 0.009) and child exposure to violence on the VEX-R (beta = 0.47, t = 3.36, p = 0.002) significantly impacted child PTSD.

4. Discussion

4.1. Child resting-state EEG

In the resting-state EEG, analysis of the oscillatory activity in the alpha band did not reveal any significant differences between children of IPV-PTSD mothers and non-PTSD control children. On the other hand, the aperiodic (i.e. non-oscillatory) component, as measured by the PLE, was significantly reduced in children of IPV-PTSD mothers, indicating enhanced cortical excitability in this group (Pani et al., 2022). The absence of significant alpha rhythm differences suggests that PLE seems to be a more sensitive marker of E/I imbalance than periodic oscillations in these children. This is the case despite observations that adult PTSD patients exhibit significantly reduced alpha oscillations that correlate with clinical and physiological measures of cortical arousal (Clancy et al., 2017; Ros et al., 2017; Veltmeyer et al., 2006).

No prior study to our knowledge has longitudinally examined the offspring of mothers with IPV-PTSD using resting state EEG. However, children of IPV-PTSD affected mothers were found in a previous study of the same sample to display increased vigilance to adult emotional expressions as compared to children of non-PTSD controls, marked by higher global field power during a cognitive-affective face-matching task (Perizzolo et al., 2019). Aiming to delve deeper into the neurobiological substrates of this behavioral hyper-arousal, the present study found that elevated E/I (reflected by a reduced PLE) confirmed greater baseline brain arousal in children from mothers with PTSD.

4.2. Child PLE and maternal/child psychopathology

The present study additionally found a significant negative correlation between maternal PTSD severity, as measured by the CAPS total score, and child PLE. This would indicate that the more severe the mother's PTSD symptoms, the lower the child PLE. As such, a reduced PLE has been shown to be an "electrophysiological marker of arousal level" (Lendner et al., 2020) across a wide range of neurobehavioral states (Colombo et al., 2019; Muthukumaraswamy and Liley, 2018). For example, adolescents with attention deficit hyperactivity disorder, who display symptoms of behavioral hyper-arousal, showed reduced aperiodic exponent (i.e. "flattened" EEG power spectrum) relative to their typically-developing peers (Ostlund et al., 2021). These prior studies are compatible with our analyses linking child PLE as a marker of hyper-vigilance among children of mothers with IPV-PTSD.

4.3. Child and maternal psychopathology, and mediation by PLE

The severity of IPV-PTSD in mothers was significantly and positively correlated with the total score and the anxious-depressed subscale score on the maternally reported CBCL. Moreover, severity of maternal IPV-PTSD was significantly and positively associated with child PTSD severity as marked by the number of symptoms on the clinician-rated K-SADS. The child's exposure to trauma as measured by the VEX-R was not significantly associated with maternal PTSD (see also (Glaus et al., 2022)). Early childhood irritability/externalizing symptoms were also reported in this sample (Schechter et al., 2017). By school-age these more externalizing symptoms can attenuate, while anxio-depressive difficulties by parental report have been shown to intensify (Sorcher et al., 2022). We know that maternal psychological functioning in the wake of exposure to a traumatic event, particularly one involving IPV, is in many studies a mediator of effects of the events experienced by the child, on child outcomes such as increased hypervigilance (Glaus et al., 2021; Langevin et al., 2022; Laor et al., 2001; Schechter et al., 2011). We therefore explored the mediation of maternal IPV-PTSD on child PTSD severity by child PLE. This mediation model clearly supports a mechanism where child PLE significantly contributed to the effect of maternal IPV-PTSD on child PTSD severity, even after covarying the effect of direct traumatic-event exposure (i.e. VEX-R). This is evident in the high statistical significance of the model total effect, reflecting the combined contribution of both the direct and mediating pathways. Hence, neither the direct or mediating (i.e. child PLE) factors were the sole determinants, but they contributed together to the intergenerational link between a mother's PTSD and a child's PTSD. Essentially, this means that a marker of a child's brain arousal may be considered a significant mediator, as well as that a part of the direct effect also remains unaccounted for by this particular brain measure.

To our knowledge, this is the first study demonstrating a biomarker of child cortical arousal as being a mediator of the effects of maternal IPV-PTSD severity on child PTSD severity. This appears to occur during an early sensitive period for the development of emotion regulation (ages 1–3.5 years). We wonder, therefore, whether one mechanism at play is increased cortical arousal in the service of adaptation to an expected environment of menace and physical aggression. Previous findings in the same sample support this view, i.e. increased vigilance and faster reaction to adult emotional expressions (Moser et al., 2023; Perizzolo et al., 2019). The latter interpretation supports the notion referred to by clinicians and neurobiologists as "survival-mode", in which the sympathetic branch of the autonomic nervous system, rather than the parasympathetic or vagal branch, predominates (Chemtob et al., 1988; Porges, 2007).

4.4. Clinical implications

How might one be able to apply the findings of this study towards preventive intervention? In response, the potential biomarker PLE uncovered in this study could be used to test the hypothesis that with reduced cortical reactivity or vigilance, in a non-threatening environment, the child would be less likely to aggress peers so automatically. The authors have been developing a targeted brief mother-child psychotherapy for mothers with IPV-PTSD and their toddlers, involving "clinician-assisted video-feedback exposure" towards this end (Schechter and Rusconi Serpa, 2021; Schechter and Rusconi-Serpa, 2013). Further research might explore reducing cortical arousal of affected children by directly targeting the PLE biomarker, e.g. with neurofeedback (Ros et al., 2017).

4.5. Limitations

There are several limitations to this study. Firstly, the sample size is

low, partly due to the fact that data were lost because of poor EEG quality. Secondly, the K-SADS screening measure was used without supplementary section questions for child symptoms (i.e. non-structured, standard clinical diagnosis based on DSM-IV-R criteria was made by the research clinicians). We acknowledge that there have been significant changes in the criteria for diagnosing children when transitioning from DSM-IV-R to DSM-5. This is especially noticeable in the case of PTSD, and it could have an impact on the categorical diagnosis made based on data from the K-SADS and the clinician's judgment after further interview (Scheeringa et al., 2011). However, in our analysis, we relied on Spearman correlations with symptom counts in the K-SADS screening module to measure severity rather than categorical diagnosis. We believe that it is still a valid, possibly more conservative, measure of the relationship with childhood symptomatology, as demonstrated for attentional deficit hyperactivity disorder (Hagstrøm et al., 2024). Thirdly, we used retrospective life event measures for childhood events, which are subject to potential bias.

5. Conclusions

This is the first study to examine the associations between maternal IPV-PTSD, child resting-state EEG and child psychopathological and behavioral outcomes. The aperiodic PLE was significantly reduced in children of IPV-PTSD mothers, indicating cortical hyper-arousal relative to control children. Interestingly, the abnormal PLE mediated the effects of maternal IPV-PTSD on child PTSD. Psycho-neurobehavioral therapy, based on self-regulation of PLE using neurofeedback, may be considered to normalize brain arousal in affected children. Additionally, it is crucial to provide early parent-child psychotherapy to help affected mothers be more physically and emotionally available to their children. This can promote emotional self-regulation and reduce the risk of PTSD, anxiety, and depression in school-age children.

Funding

This work was supported by the NCCR-SYNAPSY [SNSF grant number 51AU40_125759].

CRediT authorship contribution statement

Marie-Pierre Deiber: Writing – review & editing, Writing – original draft, Supervision, Methodology, Formal analysis. **Virginie C. Pointet Perizzolo:** Project administration, Formal analysis, Data curation, Conceptualization. **Dominik A. Moser:** Writing – review & editing, Validation, Software, Methodology, Formal analysis, Conceptualization. **Marylène Vital:** Validation, Project administration, Formal analysis. **Sandra Rusconi Serpa:** Validation, Project administration, Formal analysis. **Tomas Ros:** Writing – review & editing, Writing – original draft, Software, Methodology, Formal analysis, Conceptualization. **Daniel S. Schechter:** Writing – review & editing, Writing – original draft, Validation, Supervision, Resources, Project administration, Funding acquisition, Conceptualization.

Declaration of competing interest

none.

Acknowledgement

We thank Dr. Jennifer Glaus (SUPEA, CHUV, Lausanne, Switzerland) for her assistance with the study.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2024.07.034.

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