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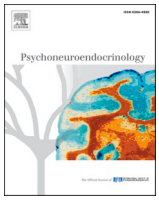

Hair glucocorticoids during pregnancy in the context of trauma exposure and their predictive value for the development of childbirth-related posttraumatic stress disorder symptoms

S. Steudte-Schmiedgen [a,*,1], S. Schälicke [a,b,1], L. Bergunde [a,b], M. Karl [a,b], V. Weise [b], J. Junge-Hoffmeister [a], S. Schumacher [c], T. von Soest [d], K. Weidner [a], C. Kirschbaum [e], S. Garthus-Niegel [b,f,g]

[a] Department of Psychotherapy and Psychosomatic Medicine, Faculty of Medicine of the Technische Universität Dresden, Dresden, Germany
[b] Institute and Policlinic of Occupational and Social Medicine, Faculty of Medicine of the Technische Universität Dresden, Dresden, Germany
[c] Clinical Psychology and Psychotherapy, Faculty of Health, Health and Medical University, Potsdam, Germany
[d] PROMENTA Research Center, Department of Psychology, University of Oslo, Oslo, Norway, and Norwegian Social Research, OsloMet – Oslo Metropolitan University, Oslo, Norway
[e] Institute of Biological Psychology, Faculty of Psychology of the Technische Universität Dresden, Dresden, Germany
[f] Institute for Systems Medicine (ISM) and Faculty of Medicine, Medical School Hamburg MSH, Hamburg, Germany
[g] Department of Childhood and Families, Norwegian Institute of Public Health, Oslo, Norway

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ABSTRACT

Background: Childbirth-related posttraumatic stress disorder (CB-PTSD) is gaining attention as a mental disorder with negative sequela for mothers and their offspring. Maternal trauma history is a well-known vulnerability factor for CB-PTSD symptoms (CB-PTSS). Furthermore, alterations of the hypothalamus-pituitary-adrenal axis have been linked to both trauma exposure and PTSD development. Hence, we investigated whether trauma history was associated with long-term glucocorticoid (GC) levels during pregnancy and their predictive role for CB-PTSS. Further, we examined whether GCs act as a mediator in the relationship between trauma history and CB-PTSS and whether this was moderated by the subjective birth experience.

Methods: 212 women participating in the prospective cohort study $DREAM_{HAIR}$ provided hair samples for quantification of long-term integrated cortisol and cortisone levels prior to their anticipated birth date accompanied by measures of trauma history. CB-PTSS and subjective birth experience were assessed two months postpartum.

Findings: Trauma history predicted elevated hair cortisol and hair cortisone during the third trimester of pregnancy, however associations did not remain significant when Bonferroni correction due to multiple testing was applied. Trauma history also predicted higher CB-PTSS. Hair GC levels during pregnancy neither predicted CB-PTSS two months after birth nor mediated the relationship between trauma history and CB-PTSS. The subjective birth experience moderated the relationship of hair cortisol and cortisone with CB-PTSS.

Conclusion: Our data suggest that a history of trauma contributes to a higher risk to develop CB-PTSS and elevated long-term GC levels during the third pregnancy trimester. Further, the predictive role of hair cortisol and cortisone levels for CB-PTSS may depend on subjective birth experience. This highlights the need to consider the latter in future investigations when examining the role of stress-related biomarkers in more severely affected samples.

* Corresponding author.
E-mail address: Susann.Schmiedgen@tu-dresden.de (S. Steudte-Schmiedgen).
[1] shared first authorship

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1. Introduction

Research shows that following childbirth, 12.3% of women develop symptoms of childbirth-related posttraumatic stress disorder (CB-PTSS; Heyne et al., 2022) which negatively impact maternal and offspring health (e.g., Garthus-Niegel et al., 2017). To inform prevention efforts, identifying factors that put women at risk for CB-PTSS is pivotal. Ayers et al.'s (2016) meta-analytically founded diathesis-stress model displays how pre-birth vulnerability factors, birth-associated risk factors, and postnatal aspects interact to predict the development of childbirth-related posttraumatic stress disorder (CB-PTSD). Among others, a negative subjective birth experience (SBE) was found to be one of the strongest predictors (Ayers et al., 2016) and trauma exposure history was highlighted as a key vulnerability factor for CB-PTSS (Grekin and O'Hara, 2014) and general PTSD (e.g., Kessler et al., 2017). The sensitization effect may explain the latter, postulating that individuals who experienced at least one prior traumatic event may be less well equipped to deal with subsequent stressors (e.g., Breslau et al., 1999), for instance a traumatic birth. This also aligns with the well known *building block effect* showing that PTSD incidence and severity increase with the number of prior traumatic events experienced (Kolassa et al., 2010).

To advance our understanding of CB-PTSS etiology, examining alterations of the hypothalamic-pituitary-adrenal (HPA) axis, our central stress response system, appears promising. Dysregulated HPA axis functioning has been suggested to link the experience of stressful and traumatic events with adverse mental and physical health outcomes, such as PTSD (e.g., Morris et al., 2016). Regarding basal cortisol levels in PTSD, the majority of cross-sectional research reported reduced circulating cortisol levels, albeit with heterogeneity in findings (meta--analysis: Schumacher et al., 2019). Similarly, a meta-analysis of prospective studies assessing HPA axis alterations immediately after trauma found no consistent relationship with subsequent PTSS development (Morris et al., 2016). Among others, heterogeneity may be attributable to most studies having employed traditional cortisol measurement methods (e.g., in blood, saliva, or urine), which capture short-term hormone concentrations and are affected by various situational factors (e.g., Stalder et al., 2017).

Increasing evidence highlights the potential of hair glucocorticoid (GC) analysis offering a reliable and valid assessment of *long-term* hormone integration for periods up to several months (Stalder et al., 2017) to elucidate the relationship between traumatization, cortisol regulation, and PTSD development (Steudte-Schmiedgen et al., 2016). Specifically, based on previous hair cortisol data, Steudte-Schmiedgen et al. (2016) proposed a model suggesting a dose-dependent increase of cortisol levels following traumatization which may chronically attenuate below baseline. In their meta-analysis, Khoury et al. (2019) found that adversity is related to both increased and attenuated hair cortisol levels (i.e., hypercortisolism and hypocortisolism), depending on moderators such as clinical status, timing of adversity, and ethnicity.

Considering research documenting an effect of lifetime trauma exposure (i.e., at least one traumatic event as well as the number of traumatic events) on hair cortisol (e.g., Schumacher et al., 2022; Steudte et al., 2013), it has been suggested that HPA axis alterations could be a consequence of prior trauma exposure rather than representing a correlate of PTSD status, thereby contributing to the risk of developing PTSD after subsequent trauma exposure (Steudte-Schmiedgen et al., 2016). This assumes HPA axis dysregulation as a premorbid vulnerability factor suggesting an endocrine mechanism for the building block/stress sensitisation effect. Studies assessing hair cortisol as a retrospective index shortly after trauma exposure (Pacella et al., 2017; Petrowski et al., 2020), as an indicator of pre-trauma cortisol functioning, revealed hypercortisolism to be linked with PTSS development. Yet, interpretation of findings is limited by samples taken *after* the index trauma potentially being affected by peri- and immediate posttraumatic phases (e.g., Colding-Jørgensen et al., 2020). In a truly prospective study among male military personnel, lower hair cortisol *prior to* deployment was predictive of PTSS increase when accounting for *new-onset* trauma exposure (Steudte-Schmiedgen et al., 2015). In the above integrative model it was thus proposed that long-term cortisol dysregulation may partly mediate the link between previous trauma history and risk for PTSD upon additional trauma exposure (Steudte-Schmiedgen et al., 2016). However, a recent study assessing duty-related trauma exposure among firefighters did not find a predictive effect of baseline hair cortisol for PTSS 6 and 12 months later (Sopp et al., 2021). The fact that this study did not consider the presence of new-onset traumatic events might be an explanation for their null-result.

These findings may, however, not generalise to pregnant women as the peripartum period shows unique physical and endocrine changes (e.g., Marceau et al., 2020). Studies using hair analyses found that pregnant women who had experienced lifetime traumatic events showed elevated hair cortisol levels (Swales et al., 2018; Schreier et al., 2016). Also abuse (Schreier et al., 2015) and traumatic events (Swales et al., 2018) in childhood were found to be related to increased hair cortisol levels among pregnant women, albeit not consistent (Schury et al., 2017). Importantly, hair samples of the above studies (except for Swales et al., 2018) were taken after delivery, thereby potentially being affected by birth itself (e.g., Colding-Jørgensen et al., 2020). These findings align with recent reviews showing that in pregnancy early life adversity was associated with elevated diurnal cortisol parameters regardless of current stress level, whereas a link with higher tonic cortisol levels (including hair cortisol) was only evident for pregnant women with greater current stress or psychological symptoms (e.g., Epstein et al., 2021).

Besides cortisol, its inactive form cortisone has been suggested as a more robust GC index (e.g., Perogamvros et al., 2010). Moreover, the ratio of cortisone and cortisol is considered an indirect estimate of 11-β-hydroxysteroid dehydrogenase type 2 (11β-HSD2) activity – an enzyme known to convert cortisol into inactive cortisone. As 11β-HSD2 plays a role in maternal prenatal mental health and protecting the fetus from excess cortisol, the above GC estimates might be promising biomarkers during pregnancy (Scharlau et al., 2018) and are thus implemented in the current study.

The aims of this prospective study were to assess (i) the association between prior trauma exposure and hair GC estimates measured *before birth*, (ii) the prospective relationship between hair GCs and subsequent postpartum CB-PTSS, and (iii) whether hair GCs mediate the relationship between trauma exposure and CB-PTSS. We expected prior trauma exposure to predict elevated hair GCs (e.g., Schreier et al., 2016) and CB-PTSS (Grekin and O'Hara, 2014). While this is the first study in the context of CB-PTSD, we hypothesized hair GCs to predict CB-PTSS based on evidence outside the realm of childbirth (Steudte-Schmiedgen et al., 2015). Finally, we combine previous evidence highlighting SBE as one of the strongest predictors for CB-PTSD (Ayers et al., 2016) and findings showing a predictive value of hair cortisol only when accounting for new-onset trauma exposure including the subjective response to the event (i.e., DSM-IV A2 criterion, Steudte-Schmiedgen et al., 2015). Based on this, we exploratively investigated whether SBE moderates the mediating effect of hair GCs by interacting with prior trauma exposure and hair GCs to predict CB-PTSS.

2. Methods

2.1. Study design and participants

This investigation is part of the prospective cohort study DREAM (Dresden Study on Parenting, Work, and Mental Health) and especially its endocrine sub-study $DREAM_{HAIR}$ (Kress et al., 2019). $DREAM_{HAIR}$ examines long-term endocrine determinants of the relationship between perinatal stress and mental health-related outcomes in both mothers, partners, and their offspring, including measurements at five time points (T1 $DREAM_{HAIR}$ – T6 $DREAM_{HAIR}$, see Fig. 1).

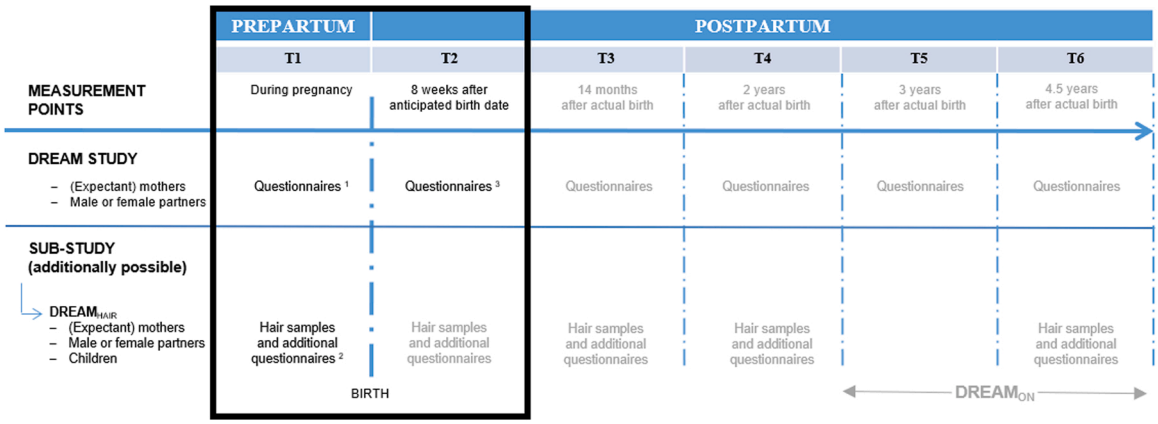

Fig. 1. Assessment waves of the DREAM study and its endocrine sub-study $DREAM_{HAIR}$. *Note.* Grayed assessments are not relevant to this investigation. [1]At T1 DREAM (range: 12 – 36 weeks pregnant) mothers completed questionnaires (e.g., FOBS and EPDS). [2] At T1 $DREAM_{HAIR}$ (*range*: 0 – 6 weeks before the anticipated birth date) mothers completed trauma questionnaires and provided hair samples. [3] At T2 DREAM mothers completed questionnaires (e.g., SBE, OBE, IES-R).

Expectant parents were recruited in two steps. For the basic DREAM study, expectant parents were recruited from June 2017 to the end of 2020 mainly at birth information evenings in obstetrical clinics and in midwife practices in Dresden, Germany. Here, measurements at T1 DREAM, which only included the assessment of questionnaire data, took place during pregnancy (T1 DREAM: $M = 27.47$ weeks pregnant, $SD = 5.06$ weeks, *range* = 12 – 36 weeks pregnant). Those who completed T1 DREAM questionnaires at least four weeks prior to the anticipated birth date were recruited for the substudy $DREAM_{HAIR}$ via a telephone screening. Hair samples for $DREAM_{HAIR}$ were taken 4 ± 2 weeks before the anticipated birth date (T1 $DREAM_{HAIR}$: $M = 2.96$ weeks before the anticipated birth date, $SD = 1.17$ weeks, *range* = 0 – 6 weeks). For T2, questionnaires from the basic DREAM study were sent by post eight weeks after the anticipated birth date (T2 DREAM: $M = 8.31$ weeks, $SD = 1.50$ weeks, *range* = 5 – 13 weeks). The current investigation focuses exclusively on T1 DREAM, T1 $DREAM_{HAIR}$, and T2 DREAM and includes only data of expectant mothers.

Basic DREAM study inclusion criteria were being currently pregnant, resident in Dresden (Germany), and adequate German language skills. Additional inclusion criteria for $DREAM_{HAIR}$ were a minimal hair length of 2 cm, no hair loss or baldness, no serious disease in the last five years, and no use of GC containing medication in the last three months. Fulfilment of further inclusion criteria was required for this investigation: no multiples due to different pregnancy and birth experience, no preterm birth, and no behavior likely to alter hair GCs (e.g., alcohol, smoking, or use of psychotropic drugs in the last three months; Stalder et al., 2017). Failure to return material at T2 DREAM and laboratory analyses not being possible (e.g., due to insufficient amount of hair) or delayed (i.e., hair samples sent to laboratory in a later batch) led to participant exclusion. Further, one participant was excluded due to completion of T2 DREAM within four weeks postpartum. In this time frame, acute stress disorder symptoms would be assessed rather than PTSS (American Psychiatric Association, 2013). The final sample consisted of N=212 expectant mothers (see Fig. 2). Sample size varied slightly between analyses due to missing data.

Dropout analyses with Welch-Test and Fisher's exact/Chi-square test were conducted to investigate whether completers (participated at T1 DREAM, T1 $DREAM_{HAIR}$, and T2 DREAM; $n = 213$) differed from non-completers (T2 DREAM not completed; $n = 17$). Comparisons were made for all study variables. Groups differed only regarding depressive symptoms during pregnancy, such that non-completers had slightly higher depressive symptoms ($t(20.71) = 2.51$, $p = .020$, Cohen's $d = 0.50$).

Written informed consent according to the Declaration of Helsinki was given by all participants for DREAM and $DREAM_{HAIR}$ and ethical approval was received by the Ethics Committee of the Faculty of Medicine of the Technische Universität Dresden (No: EK 278062015).

2.2. Measurements

2.2.1. Sociodemographic and hair characteristics

Sociodemographic characteristics (e.g., age, BMI prior to pregnancy, academic degree, preterm birth (birth 21 days or more before the anticipated birth date)), medication intake, and physical illness were assessed at T1 DREAM. Self-reported hair-related characteristics (i.e., weekly hair washes, sunlight exposure, any hair treatment such as tint, perm, or coloring in the last three months) as well as health-related questions (i.e., smoking, alcohol, and drugs in the last three months) were assessed with an in-house hair protocol at T1 $DREAM_{HAIR}$ (Stalder et al., 2014).

2.2.2. Clinical and psychological measures

CB-PTSS as the main dependent variable were measured at T2 DREAM using the German version of the Impact of Event Scale – Revised (IES-R; Maercker and Schützwohl, 1998; Weiss and Marmar, 1996). The subscales *intrusion*, *avoidance,* and *hyperarousal* were measured in relation to childbirth. Participants were asked to rate 22 items on a 4-point Likert scale from *not at all* (0) to *often* (5) (total score: 0 – 110; Rosner and Hagl, 2008). The clinical cut-off of ≥ 35 of the original Impact of Event Scale (total score: 0 – 75) was employed (Neal et al., 1994). In our sample, internal consistency for total IES-R score was acceptable (Cronbach's $\alpha = 0.77$).

Previous traumatic events and trauma exposure according to DSM-IV were assessed through self-report based on the Trauma Checklist of the Posttraumatic Diagnostic Scale (Foa et al., 1997) at T1 $DREAM_{HAIR}$. Participants indicated which (if any) out of 12 traumatic event types (including an open category "other") they had experienced so far. All events mentioned under "other" that were related to childbirth were subsequently categorized as birth traumatization. Finally, if traumatic events had been experienced, participants were asked to indicate the most upsetting event. With regards to this event, criterion A1 (i.e., injury to oneself or others, thinking one's own life or the life of someone else is in danger) and criterion A2 (i.e., feelings of helplessness, fear, and horror) of the DSM-IV (American Psychiatric Association, 2000) were assessed by six yes−no questions. Expectant mothers were classified as trauma-exposed according to DSM-IV (coding 0 vs 1) if criterion A1 and A2 were fulfilled. The number of lifetime traumatic events was calculated as the sum of traumatic event types reported (*range* = 0 – 12).

Beyond lifetime trauma exposure, we also examined childhood maltreatment, measured by the 28-item Childhood Trauma Questionnaire short-form (CTQ; Bernstein et al., 2003) at T1 DREAM as a predictor of hair GCs and CB-PTSS. The CTQ provides a total score (good

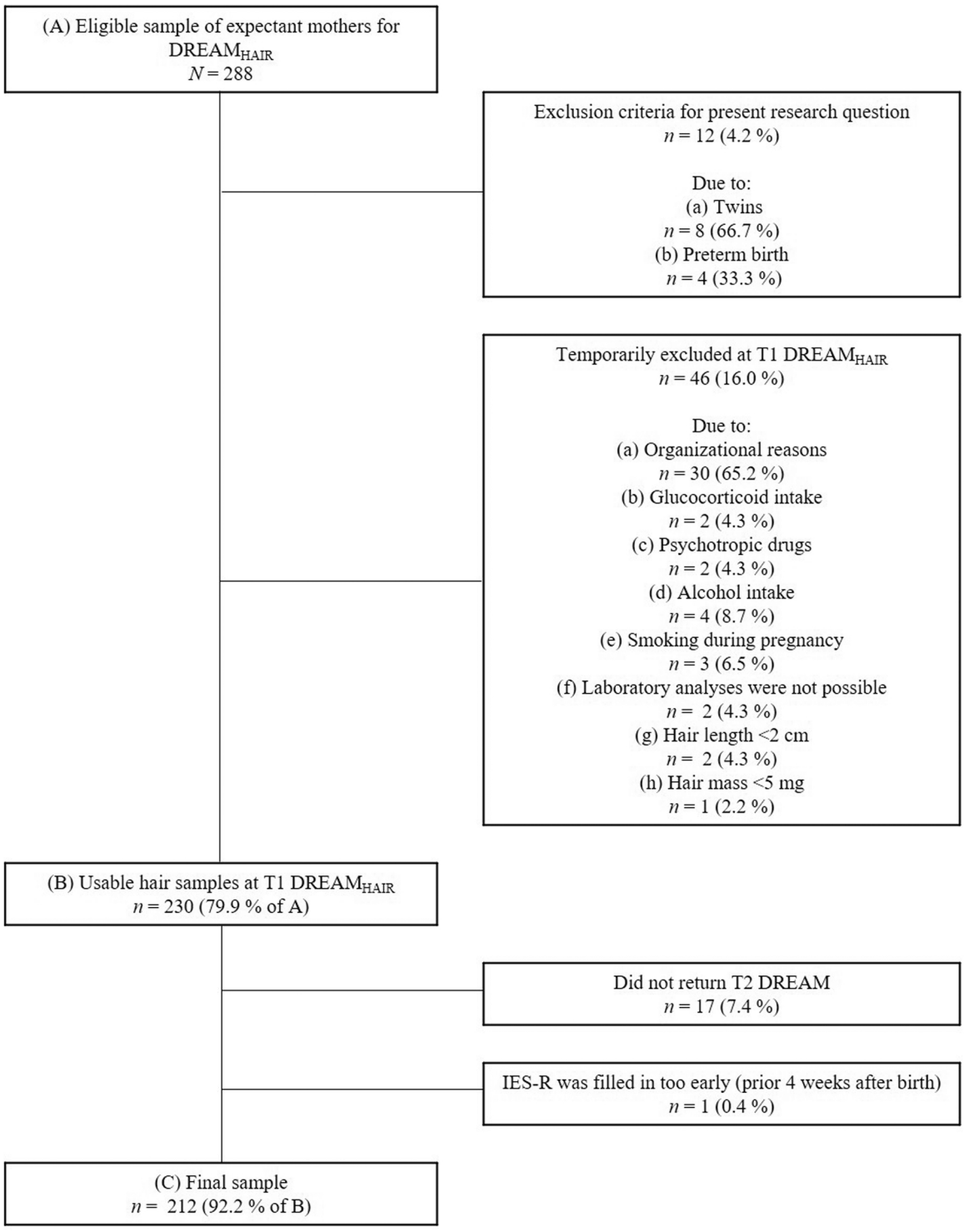

Fig. 2. Flowchart of Retention Rate and Exclusion Criteria Resulting in Final Sample *Note*. Data until end of February 2020 (version 7 of the quality-assured data files, prospective data collection ongoing). The last hair sample in the final sample was taken on the 27th of November 2019, i.e., before the COVID-19 pandemic. T1 DREAM$_{HAIR}$ = 4 ± 2 weeks prior to anticipated birth date; T2 DREAM = 8 weeks after anticipated birth date. IES-R = Impact of Event Scale – Revised.

internal consistency, α = 0.88) as well as cut-off scores for none or low-moderate and moderate-severe exposures to five trauma categories including emotional abuse (EA), physical abuse (PA), sexual abuse (SA), emotional neglect (EN), and physical neglect (PN) (Bernstein et al., 2003). Based on these cut-offs, participants were categorized as having had no or low-moderate childhood maltreatment or moderate-severe childhood maltreatment in at least one trauma category (EA ≥ 13, PA ≥ 10, SA ≥ 8, EN ≥ 15, PN ≥ 10).

SBE as a moderator was measured at T2 DREAM based on the single item "How was your overall experience of the birth?" rated on a numeric scale ranging from 0 (*positive*) to 10 (*negative*). This was recoded so that higher values represent a more positive SBE. This measure has shown high predictive value for CB-PTSS in previous research (r = 0.39; Garthus-Niegel et al., 2014).

An index of objective birth experience (OBE; based on Garthus-Niegel et al., 2013) was included as a control variable in analyses

predicting CB-PTSS. The 16-item OBE index was constructed using information from maternity records and self-generated questions at T2 DREAM. Maternal (*no progress in 2nd stage of labor, premature placental abruption or difficulties with placental abruption, extensive blood loss, tears (vaginal, labial, or perineal), instrumental birth, active phase of labor > 12 h*) and infant (*breech or transverse presentation, pathological heart sounds, umbilical cord complications, green amniotic fluid, Apgar score <7 at 5 min*) complications were treated as dichotomous variables (0 = had not occurred; 1 = occurred).

Depressive symptoms during pregnancy and fear of childbirth (FOC) were also included as confounding variables. Depressive symptoms during pregnancy were assessed by means of the German version of the Edinburgh Postnatal Depression Scale (EPDS; Bergant et al., 1998) at T1 DREAM. The EPDS inquires the degree of severity of ten symptoms, rated on a four-point scale from *zero* to *three* in the past seven days (total score: 0 – 30). For sample characteristics, cut-off scores of ≥ 10 for minor depression and ≥ 13 for major depression were used (Bergant et al., 1998). In this sample, Cronbach's alpha showed good internal consistency (α = 0.85). FOC at T1 DREAM was measured using the Fear of Birth Scale (FOBS; Haines et al., 2011), a highly economical two item assessment. Based on the question "How do you feel right now about the approaching birth?" participants estimated on a scale ranging from 0 to 100 how calm (*calm* to *worried*) and fearful (*no fear* to *strong fear*) they felt. The average of the two scores formed a total score, where higher values indicated more fear. Internal consistency in the present sample was high (Cronbach's α = 0.89).

2.2.3. Hair GC analyses

Two strands of hair (minimum length of 2 cm; collective diameter of 3 mm) were collected mainly by trained staff at the scalp-near posterior vertex position. Some participants were sent the materials by post with detailed instructions and a video tutorial for self-sampling. Hair samples were stored in a dry and dark place at room temperature in aluminum foil and sent to the laboratory at the Technische Universität Dresden in two batches (storage time: $M = 49.96$ weeks, $SD = 15.48$, *range* = 21 – 80). Hair cortisol (HairF) and cortisone (HairE) were determined in the scalp-near 2 cm hair segment. Based on an average growth rate of 1 cm per month, this represents GC secretion of the two months prior to hair sampling, reflecting approximately the third pregnancy trimester in our sample. Laboratory analyses performed liquid chromatography tandem mass spectrometry according to a published protocol demonstrating high sensitivity, reliability, and specificity (Gao et al., 2013).

2.3. Statistical analyses

Analyses were conducted with IBM SPSS Statistics 28 (IBM Corp, 2021). For IES-R, EPDS, and CTQ, if at least 80% of items were completed, mean replacement with the individual's mean of that scale was used for EPDS ($n = 1$; 10% missing), IES-R ($n = 9$, max. 9.09% missing), and CTQ ($n = 4$; max. 4% missing). As expected, HairF and HairE were not normally distributed and thus were log transformed to reduce skewness. Two expectant mothers with non-detectable values for HairF and participants with outliers with ± three standard deviations from the mean of each GC were excluded (HairF: $n = 5$; HairE: $n = 4$), resulting in a final sample for analyses of $n = 207$ (HairF), $n = 208$ (HairE), as well as $n = 203$ (HairE/HairF ratio).

To reduce bias (Lee, 2015), we controlled for the following confounders which were chosen based on theoretical considerations indicating their influence on trauma history, hair GCs, and CB-PTSS: age, parity, academic degree, depressive symptoms during pregnancy, and FOC (e.g., Ayers et al., 2016; Marteinsdottir et al., 2021; Stalder et al., 2017; Ursache et al., 2017). For the relationship between trauma history and hair GCs, confounders for GCs as proposed by previous evidence (BMI, storage time, hair treatment, sunlight exposure, gestational week at T1 $DREAM_{HAIR}$, hair washes per week) were also investigated and included in subsequent analyses if they were significantly associated with hair GCs in our sample (e.g., Braig et al., 2015; Marceau et al., 2020; Stalder et al., 2017). Relevant confounders for hair GCs (i.e., that correlated significantly with hair GCs) in the current sample were: BMI (HairF: r_s=0.22, 95% CI [0.08,0.35], p=.002; HairE: r_s=0.18, 95% CI [0.04,0.31], p= .01; HairE/HairF ratio: r_s= −0.14, 95% CI [−0.28, −0.00], p=.042), storage time (HairF: r_s= −0.42, 95% CI [−0.53, −0.30], p<.001; HairE: r_s= −0.36, 95% CI [−0.48,−0.23], p< .001; HairE/HairF ratio: r_s=0.39, 95% CI [0.26,0.51], p < .001), hair treatment (HairF: r_s =.–15, 95% CI [−0.29,−0.01], p=.029, HairE/HairF ratio: r_s =0.14, 95% CI [0.00,0.28], p= .040, but not HairE: r_s = −0.13, 95% CI [−0.26,0.02], p= .072), and sun exposure per week (HairF: r_s=0.22, 95% CI [0.08,0.350], p=.002; HairE: r_s =0.16, 95% CI [0.02, 0.30], p=.021; HairE/HairF ratio: r_s = −0.19, 95% CI [−0.32,−0.05], p= .008). Hair wash frequency per week was only significantly associated with the HairE/HairF ratio (r_s = −0.15, 95% CI [−0.28,−0.01], p=.037).

Spearman Rank correlations were estimated between key study variables. Then linear multiple regression models with 95% bias-corrected and accelerated bootstrap confidence intervals based on N=2000 bootstrap resampling procedures were carried out to examine the relationships between 1) trauma history and hair GCs, 2) hair GCs and CB-PTSS, 3) trauma history and CB-PTSS. For illustrative purposes, an ANCOVA was conducted to investigate differences in hair GCs between trauma-exposed and non-trauma-exposed mothers. Subsequently, using the Preacher and Hayes PROCESS macros (Hayes, 2018), we conducted mediation (model 4) and moderated mediation (model 15 with SBE as a moderator) analyses, one for each hair GC estimate (HairF, HairE, HairE/HairF ratio).

While testing model assumptions for multiple regression, one multivariate outlier was detected in diagnostical approaches (e.g., case-by-case diagnosis, studentized deleted residuals, and Cook's distances). Closer inspection showed this outlier had the highest IES-R sum score as well as very high FOC, however responses overall were not illogical nor indicative of a clearly different population. Therefore the outlier was included in reported analyses and we conducted sensitivity analyses with and without this outlier, however no changes in significance occurred. Significance was determined with $p \leq .05$ and confidence intervals (CIs) not containing zero indicating significance. Bonferroni correction was used for analyses of hypothesis one to correct for multiple testing, resulting in a significant p-value of ≤ .008.

3. Results

3.1. Sample characteristics and preparatory analyses

Sample characteristics (Table 1) showed that 62 expectant mothers (29.2%) fulfilled both trauma criterion A1 and A2, leading to nearly one third of participants being classified as trauma-exposed according to DSM-IV. Of the trauma exposed women, six (9.7%) experienced the traumatic event in the year prior to hair sampling. At T1 DREAM, 16 (7.5%) and 14 (6.6%) women fulfilled the cut-off for minor and major depression, respectively. At T2 DREAM, only two women (0.9%) had clinically relevant CB-PTSS (≥ 35).

Table 2 displays Spearman correlations between key study variables. CB-PTSS showed positive correlations with trauma exposure ($p = .004$), lifetime trauma load ($p = .010$), depressive symptoms ($p < .001$), and FOC ($p = .020$), however only a trend-level correlation with SBE ($p = .072$). No significant association with childhood maltreatment (p's > .130), the objective birth experience ($p = .599$), or any of the hair GCs (p's > .120) emerged. A positive correlation between trauma exposure, however not lifetime trauma load, and HairF as well as HairE emerged (r_s's > .17, p's < .05). As expected, all hair GCs were significantly associated with one another (p's < .001). Associations between childhood maltreatment (both continuous and dichotomous measures) and the main outcome variable CB-PTSS (p's > .130) and the hair GCs (p's > .310) were not significant. Hence, information on regression and

Table 1
Sample characteristics.

Variables	
Sociodemographic characteristics	
Age in years (*M, SD,* Range)[a, 1]	30.55 ± 3.81 (18 – 42)
Body mass index (kg/m^2; *M, SD, Range*)[a, 1]	23.66 ± 4.23 (16.60 – 40.90)
Mother Language German (*n*, %) [1]	203 (95.8%)
Marital status[a, 2]	
Married/ registered same sex partnership (*n*, %)	91 (43.1%)
Unmarried (*n*, %)	114 (54.0%)
Divorced (*n*, %)	6 (2.8%)
Parity[a, 1]	
Primiparous (*n*, %)	170 (80.2%)
Multiparous (*n*, %)	42 (19.8%)
Academic degree[a, 2]	
No academic degree (*n*, %)	77 (36.5%)
Academic degree (*n*, %)	134 (63.5%)
Hair GCs	
HairF concentrations (pg/mg; *M, SD, Range*)[b, d, 3]	8.74 ± 7.43 (0.99 – 45.03)
HairE concentrations (pg/mg; *M, SD, Range*)[b, d, 4]	28.91 ± 23.40 (4.03 – 153.19)
HairE/HairF ratio (pg/mg; *M, SD, Range*)[b, d, 5]	1.86 ± 0.76 (1.13 – 7.79)
Hair-specific characteristics	
Hair wash frequency per week[b, 1]	2.94 ± 1.31 (0.25 – 7)
Sunlight exposure (minutes/week)[b, 2]	776.60 ± 540.84 (9 – 3960)
At least one hair treatment[b, 1]	81 (38.2%)
Trauma-related and clinical/psychological variables	
Number of prior potentially traumatic events (PDS; *M, SD, Range*)[b, 3]	0.81 ± 1.04 (0 – 5)
At least one traumatic event according to DSM-IV[1]	62 (29.2%)
DSM-IV traumatic event within the past year	6 (9.7%)
Type of the most upsetting traumatic event[6]	
Accident	24 (40.0%)
Natural disaster	5 (8.3%)
Violent attack	10 (16.7%)
Sexual attack	6 (10.0%)
Physical illness	3 (5.0%)
Birth-related	2 (3.3%)
Other	10 (16.7%)
Childhood Maltreatment load (CTQ; *M, SD, Range*)[b, 3]	31.91 ± 7.67 (25 – 72)
Childhood Maltreatment above the cut-off[b, 3]	31 (14.6%)
CB-PTSS (IES-R total score; *M, SD, Range*)[c, 1]	13.66 ± 9.79 (0 – 58)
SBE (*M, SD, Range*)[c, 7]	7.58 ± 2.45 (0–10)
OBE (*M, SD, Range*)[c, 1]	1.46 ± 1.12 (0 – 6)
Depressive symptoms (EPDS; *M, SD, Range*)[a, 1]	5.08 ± 4.14 (0 – 21)
FOC (FOBS; *M, SD, Range*)[a, 2]	33.91 ± 22.17 (0–100)

Note. Raw Hair GC data was used. HairF = Hair cortisol; HairE = Hair cortisone; PDS = Trauma Checklist of the Posttraumatic Diagnostic Scale; CTQ = Childhood Trauma Questionnaire; CB-PTSS = Childbirth-related Posttraumatic Stress Symptoms; IES-R = Impact of Event Scale-Revised. SBE = Subjective birth experience. OBE = Objective birth experience; EPDS = Edinburgh Postnatal Depression Scale; FOC = Fear of Childbirth; FOBS = Fear of Birth Scale; [a] T1 DREAM (in pregnancy). [b] T1 DREAM$_{HAIR}$ (4 ± 2 weeks prior anticipated birth date). [c] T2 DREAM (eight weeks after anticipated birth date). [d] Glucocorticoid specific outliers (± 3 SD) were excluded.
[1] $n = 212$; [2] $n = 211$; [3] $n = 207$; [4] $n = 208$; [5] $n = 203$, [6] $n = 60$ due to $n = 2$ missing values; [7] $n = 210$.

mediation and moderated mediation findings with childhood maltreatment are provided in the supplements (see supplementary materials).

3.2. Main analyses

3.2.1. Associations between trauma history and hair GCs during pregnancy

Among expectant mothers, linear regression analyses, controlling for age, parity, academic degree, FOC, depressive symptoms, BMI, storage time, sunlight exposure, hair treatment (only for HairF and HairE/HairF ratio), and hair wash frequency (only for the HairE/HairF ratio) confirmed correlational findings by revealing previous trauma exposure as a significant positive predictor of HairF ($B = 0.11$, $\beta = 0.15$, $SE = 0.05$, 95% BCa CI [0.01,0.20], $p = .033$) and HairE ($B = 0.08$, $\beta = 0.15$, $SE = 0.04$, 95% BCa CI [−0.00, 0.17], $p = .0499$; see Fig. 3). However, neither finding survived Bonferroni correction with a significant p-value of < 0.008. The relationship between trauma exposure and the HairE/HairF ratio was not significant, neither in correlation analysis ($r_s = -0.11$, $p = .14$), nor in the regression model ($B = -0.13$, $\beta = -0.08$, $SE = 0.12$, 95% BCa CI [−0.35, 0.07], $p = .249$). Further, lifetime trauma load was not associated with or predictive of any of the hair GCs in correlational (r_s's = −0.00 to 0.11, p's $> .11$) and regression analyses (B's = −0.02 to 0.01, p's $> .652$).

3.2.2. Predictive associations between trauma history measures and CB-PTSS

Linear regression, controlling for age, parity, academic degree, FOC, OBE, and depressive symptoms, confirmed the positive relationship found in correlational analyses between trauma exposure (yes/no) and CB-PTSS ($B = 3.89$, $\beta = 0.19$, $SE = 1.53$, 95% BCa CI [1.02, 6.78], $p = .017$) and lifetime trauma load and CB-PTSS ($B = 1.90$, $\beta = 0.20$, $SE = 0.60$, 95% BCa CI [0.77, 3.16], $p < .001$).

3.2.3. Predictive associations between hair GCs and CB-PTSS

None of the hair GCs was associated with CB-PTSS in correlational analyses ($p > .05$, see Table 2). This was confirmed by linear regression analyses controlling for age, parity, academic degree, FOC, depressive symptoms, OBE, and trauma exposure (HairF: $B = -1.08$, $\beta = -0.04$, $SE = 2.11$, 95% BCa CI [−5.17, 2.99], $p = .612$; HairE: $B = 2.07$, $\beta = 0.06$, $SE = 2.68$, 95% BCa CI [−3.13, 7.32], $p = .439$; HairE/HairF ratio: $B = 0.36$, $\beta = 0.03$, $SE = 1.11$, 95% BCa CI [−1.46, 3.31], $p = .731$). In regression analyses controlling for relevant confounders and lifetime trauma load rather than trauma exposure, also no significant results emerged ($B = -1.10$ to 2.49, p's $> .337$).

3.2.4. Mediation analyses

To examine the mediating effect of hair GCs in the relationship between trauma history and CB-PTSS, PROCESS Model 4 was employed with the following covariates: age, parity, academic degree, FOC, OBE and depressive symptoms. In line with results from linear regression, previous trauma exposure, but not lifetime trauma load, was predictive of HairF and HairE during pregnancy. None of the hair GCs significantly predicted CB-PTSS. The positive effect of previous trauma exposure on CB-PTSS was significant in all models. However, this effect did not become significantly smaller after inclusion of any hair GCs as a mediator. Accordingly, none of the indirect effects were significant (HairF: $B = -0.16$, $SE = 0.33$, *95% CI* [−0.91, 0.44]; HairE: $B = 0.23$, $SE = 0.33$, *95% CI* [−0.36, 0.94]; HairE/HairF ratio: $B = -0.07$, $SE = 0.24$, *95% CI* [−0.68, 0.31]). We repeated mediation analyses with lifetime trauma load as a predictor. Only the effect of lifetime trauma load on CB-PTSS was significant ($p < .05$). All other effects ($p > .05$) and the indirect effects (HairF: $B = -0.03$, $SE = 0.08$, *95% CI* [−0.24, 0.11]; HairE: $B = 0.05$, $SE = 0.09$, *95% CI* [−0.10, 0.29]; HairE/HairF ratio: $B = -0.03$, $SE = 0.08$, *95% CI* [−0.21, 0.12]) were non-significant.

3.2.5. SBE as a moderator of the relationship between trauma history, hair GCs, and CB-PTSS

PROCESS Model 15 with mean-centering was used to explore the effect of SBE as a moderator of the indirect relationship between trauma history and CB-PTSS via hair GCs (see Fig. 4). Analyses were conducted for both measures of trauma history (previous trauma exposure and lifetime trauma load). Analyses with previous trauma exposure as the predictor and HairE as the mediator revealed two significant interaction effects (Fig. 4A). SBE significantly moderated the association between trauma exposure and CB-PTSS, such that the effect of prior trauma exposure on CB-PTSS was stronger among mothers who had more negative SBE, compared to mothers with more positive SBE. Johnson-

Table 2
Spearman correlations [95% CI [a]] among primary study variables (N = 212 [b]).

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. CB-PTSS (IES-R)		**.21*** [.07, .34]	**.18*** [.04,.31]	.05 [-.14,.15]	.11 [-.04,.24]	-.13 [-.26,.02]	.04 [-.10,.17]	**.23*** [.09,.36]	**.16*** [.02,.29]	-.07 [-.20,.08]	.06 [-.08,.20]	.11 [-.03,.25]
2. Trauma exposure (yes/no; PDS)			**.71*** [.63,.77]	.14 [-.01, .27]	**.19*** [.05,.32]	-.08 [-.22,.07]	-.01 [-.16,.13]	.13 [-.02,.27]	.07 [-.07,.22]	**.17*[c]** [.03,.31]	**.18*[c]** [.03,.31]	-.11 [-.25,.04]
3. Lifetime trauma load (PDS)				**.23*** [.09,.35]	**.32*** [.19,.44]	.00 [-.14,.14]	-.10 [-.23,.05]	**.17*** [.03,.30]	.07 [-.07,.21]	.07 [-.07,.21]	.11 [-.03,.25]	-.00 [-.14,0.14]
4. Childhood trauma load (CTQ)					**.59*** [.48,.67]	**-.14*** [-.28,.00]	-.10 [-.23,.05]	**.22*** [.08,.35]	.04 [-.10,.18]	.05 [-.09,.19]	-.06 [-.20,.08]	-.07 [-.21,.07]
5. Childhood trauma (yes/no; CTQ)						-.02 [-.16,.12]	-.08 [-.21, .07]	**0.22*** [.09,.35]	.04 [-.10,.18]	-.03 [-.17,.11]	-.00 [-.14,.14]	.02 [-.13,.16]
6. SBE							**-0.32*** [- .44, - .19]	-.13 [-.27,.01]	**-.23*** [-.36, .09]	.00 [-.14,.15]	.06 [-.08,.20]	.02 [-.12,.17]
7. OBE								-.10 [-.23,.04]	-.01 [-.15,.13]	.06 [-.08,.20]	-.02 [-.16,.12]	-.06 [-.20,.08]
8. Depressive symptoms (EPDS)									**.45*** [.33,.55]	.00 [-.14,.14]	.02 [-.12,.16]	.008 [-.13,.15]
9. FOC (FOBS)										.07 [-.08,.20]	.09 [-.05,.23]	-.07 [-.21,.07]
10. HairF											**.78*** [.71,.82]	**-.90*** [-.93, .87]
11. HairE												**-.46*** [-.56,.34]
12. HairE/HairF ratio												

Note. HairF, HairE and HairE/HairF ratio levels were log-transformed. CB-PTSS = Childbirth-related Posttraumatic Stress Symptoms; IES-R = Impact of Event Scale – Revised. PDS = Trauma Checklist of the Posttraumatic Diagnostic Scale. CTQ = Childhood Trauma Questionnaire. SBE = Subjective birth experience. OBE = Objective birth experience. EPDS = Edinburgh Postnatal Depression Scale. FOC = Fear of Childbirth; FOBS = Fear of Birth Scale. [a] Values in brackets show the 95% confidence interval for each correlation. [b] *n* varied slightly due to missing data and outlier removal and ranged from 195 to 212. [c] did not survive Bonferroni correction ($p > .008$). * $p \leq .05$ (two-tailed).

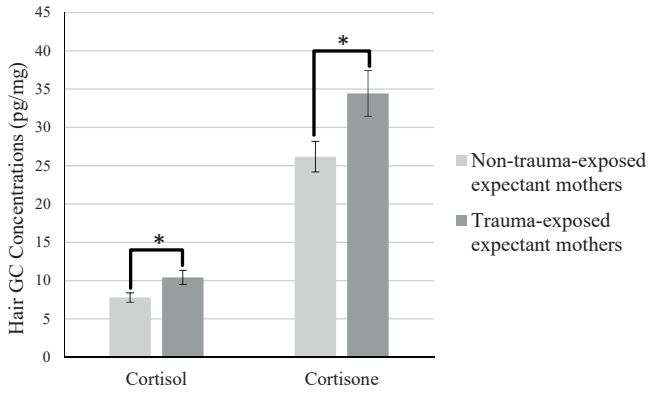

Fig. 3. Mean hair cortisol and cortisone concentrations of trauma-exposed and non-trauma-exposed expectant women during the third trimester of pregnancy. *Note.* Mean (±1SEM) raw hair cortisol and cortisone concentrations of expectant mothers who had experienced a traumatic event (trauma-exposed) and expectant mothers who had not experienced a traumatic event (non-trauma-exposed). For illustrative purposes results are based on ANCOVA calculations controlling for relevant confounders using non-log transformed hair GCs ($p < .05$).

Neyman interaction probing showed that the effect of prior trauma exposure on CB-PTSS became significant for SBE values of 0.08 SDs above the mean and lower (i.e., more negative). The interaction effect between HairE and SBE indicated that HairE significantly predicted CB-PTSS only when SBE was positive, namely more than 0.6 SDs above the mean. The overall index of moderated mediation was not significant (Index: 0.27 [−0.04, 0.65]). For HairF and the HairE/HairF ratio neither the interaction terms (i.e., previous trauma exposure x SBE and hair GCs x SBE) nor the index of moderated mediation were significant (HairF: 0.18, [−0.11, 0.53] and HairE/HairF ratio: −0.04; [−0.45, 0.24]).

Analyses with lifetime trauma load as a predictor revealed similar findings (Figs. 4B and 4C). The index of moderated mediation (HairF: 0.05 [−0.05, 0.17]; HairE: 0.06 [−0.07, 0.21]; HairE/HairF ratio: 0.002 [−0.14, 0.07]) and the interaction between lifetime trauma load and SBE ($p > .07$) were not significant in any of the models. However, a significant interaction effect of HairE and HairF with SBE in predicting CB-PTSS emerged. Johnson-Neyman interaction probing indicated that for negative SBE at least 2.2 SDs or more below the mean, low HairE values were significantly associated with greater CB-PTSS, whereas for positive SBE at least 0.6 SDs above the mean, high HairE values predicted greater CB-PTSS. Regarding HairF, results showed that low HairF was predictive of CB-PTSS upon negative SBE of at least 0.9 SDs below the mean.

4. Discussion

To our knowledge, this is the first study investigating the relationship between trauma history, long-term integrated GCs during pregnancy, and the development of CB-PTSS two months postpartum among expectant mothers. Our results revealed that women who had experienced at least one traumatic event according to DSM-IV prior to birth (i.e., trauma-exposed) showed elevated HairF and HairE during the third pregnancy trimester as well as higher levels of CB-PTSS two months postpartum. Having had experienced a greater number of prior traumatic events also predicted elevated CB-PTSS. Findings showed no association between hair GCs during pregnancy and CB-PTSS and hair GCs did not mediate the relationship between trauma history and developing CB-PTSS. However, results tentatively suggest that the risk for CB-PTSS associated with HairF and HairE during pregnancy may depend on how birth was subjectively experienced.

Consistent with our hypothesis, data showed that prior trauma exposure was associated with elevated HairF and HairE among expectant mothers. This suggests that if an expectant mother experienced at least one traumatic event fulfilling both the A1 and A2 criterion of DSM-IV at some point in her life, this was associated with long-term endocrine alterations evident during the third pregnancy trimester. This association, however, did not survive correction for multiple testing and should

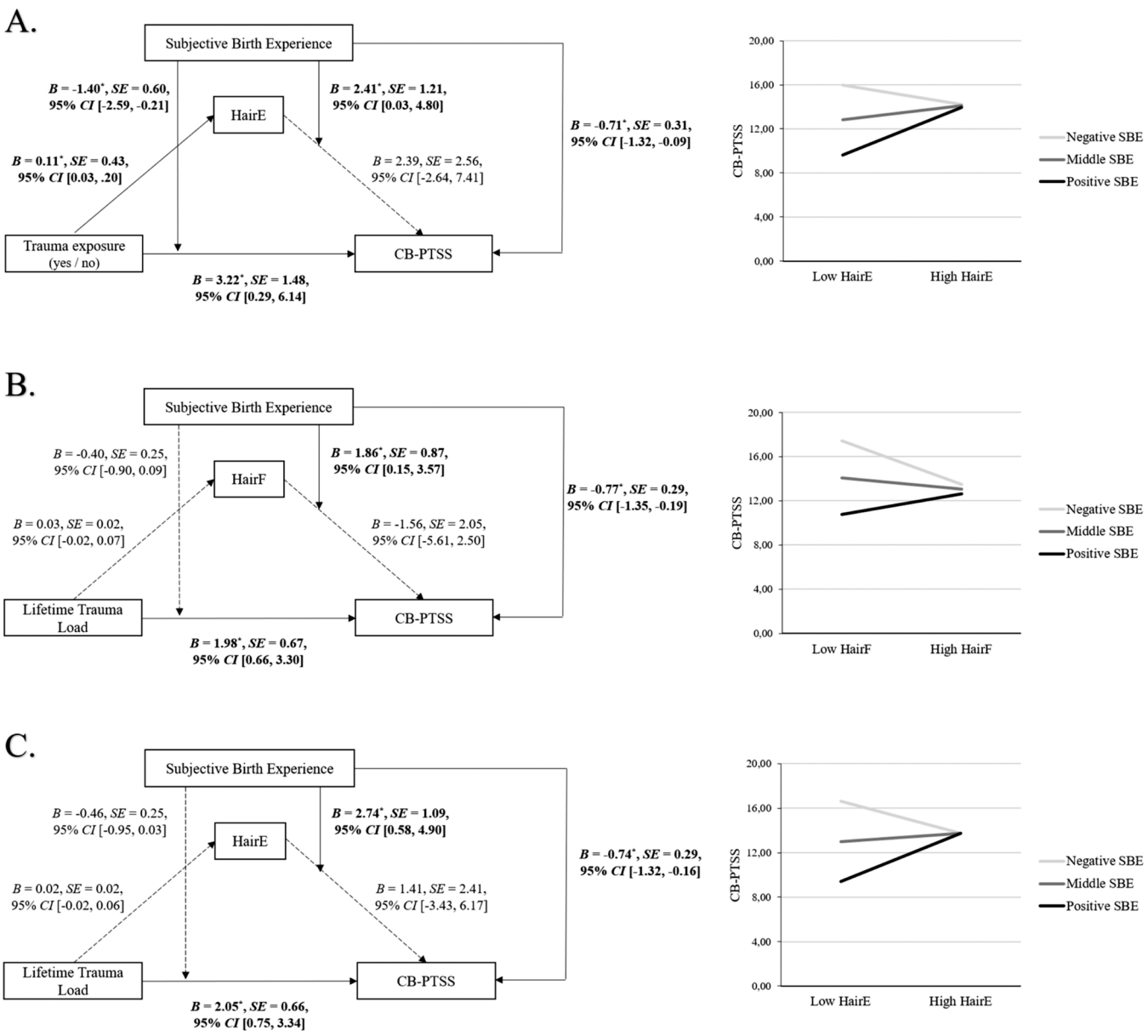

Fig. 4. Moderated Mediation Analysis predicting CB-PTSS. *Note.* CB-PTSS = Childbirth-related posttraumatic stress symptoms. HairF = Hair cortisol. HairE = Hair cortisone. SBE = Subjective birth experience. Predicting CB-PTSS based on a moderated mediation analysis with A) previous trauma exposure, HairE, and subjective birth experience B) lifetime trauma load, HairF, and subjective birth experience and C) lifetime trauma load, HairE, and subjective birth experience in PROCESS Model 15 with 5000 bootstrap resampling procedures. Covariates as described in text were included. The overall index of moderated mediation was not significant in any of the models (p's $> .05$). Graphs on the right show the significant hair GC x SBE interaction with positive/high and negative/low values corresponding to ± 1 SD from the mean.

therefore be interpreted cautiously and replicated in future studies. Moreover, the relationship was not observed for the number of lifetime traumatic events. Findings partly align with previous studies among pregnant women where adulthood traumatic events predicted elevated long-term cortisol secretion in the last pregnancy trimester (Swales et al., 2018; Schreier et al., 2016) as well as with studies using traditional cortisol assessment methods (Epstein et al., 2021). Besides lifetime trauma history, our data did not reveal a significant effect of childhood maltreatment on maternal HairF and HairE during pregnancy, which is in line with Schury et al. (2017), but contrasts with Swales et al. (2018). Even when controlling for prenatal depressive symptoms and FOC the effect of lifetime traumatic events on hair GCs remained robust, suggesting that irrespective of prenatal psychopathology, prior trauma exposure may alter HPA axis functioning. This fits with research among pregnant women where trauma exposure predicted HairF regardless of depression or PTSD status (Schreier et al., 2016). However, as we did not measure PTSS prior to birth, we could not account for pre-existing PTSS, and we could also not confirm their positive association between the *number of lifetime traumatic experiences* and HairF during pregnancy. Yet, our findings show that experiencing at least one lifetime traumatic event, regardless of degree of traumatization, affects hair GCs during pregnancy.

Although largely consistent with studies among pregnant women (e.g., Swales et al., 2018), research among non-pregnant mixed-sex samples is more heterogenous, with some studies finding a negative association of both trauma measures with HairF (e.g., Steudte et al., 2013), and meta-analytic findings reporting both elevated and reduced HairF in relation to traumatic experiences (Khoury et al., 2019). However, we note that Khoury et al. (2019) mixed childhood and lifetime trauma and included clinical samples, thereby potentially contributing

to heterogeneity in findings. From reviews of existing literature outside pregnancy, the endocrine building block effect has been proposed (Steudte-Schmiedgen et al., 2016), positing that traumatic experience initially leads to increased cortisol secretion, followed by a decrease below initial baseline levels with time. Based on this, our finding of a *positive* relationship between trauma exposure and hair GCs would indicate that study participants were in the initial post-trauma phase of increased cortisol secretion. However, as only six participants experienced their DSM-IV traumatic event within the year prior to hair sampling, *recent* traumatic exposures cannot explain the positive relationship. Instead, considering the immense physiological changes during pregnancy, it is conceivable that the pregnant state represents a unique stressful period in which effects of traumatic experiences on HPA axis functioning present themselves differently (e.g., Epstein et al., 2021). The building block effect also defines a dose-response relationship between the number of traumatic experiences and cortisol levels, such that cortisol levels attenuate even further below baseline with additional traumatic experiences (Steudte-Schmiedgen et al., 2016). Our findings oppose this idea as results showed no relationship between maternal lifetime trauma load and hair GCs.

Importantly, in accordance with studies investigating hair GCs in other research contexts (e.g., Scharlau et al., 2018; Steudte-Schmiedgen et al., 2021; Valk et al., 2022), we also examined HairE and the HairE/HairF ratio in addition to HairF in pregnant women in the context of trauma history and CB-PTSS. Our findings indicate elevated HairE, the inactive form of HairF, during the third pregnancy trimester may also be a biological correlate of trauma exposure while this was not evident for the ratio of cortisone and cortisol. This indicates that maternal trauma history may not impact the activity of the enzyme 11ß-HSD2 during pregnancy, suggesting other factors, such as current stress, may be more relevant for this enzyme (Scharlau et al., 2018). However, additional analyses regarding the effect of childhood maltreatment, indicated a negative relationship between CTQ score and HairF/E ratio, suggesting that mothers with history of increased childhood maltreatment showed increased enzymatic activity of 11ß-HSD2. This preliminary finding, which did not survive Bonferroni correction, needs to be confirmed by future investigations.

Overall, the finding that trauma exposure is linked to increased hair GCs during pregnancy is indicative of a potential biological mechanism of the sensitization effect, whereby changes to HPA axis functioning may explain how prior traumatic experience increases susceptibility to negative effects of subsequent traumatic experiences (Breslau et al., 1999). Results lend support to the idea that prior traumatic stress exposure affects long-term changes in GC secretion evident during pregnancy. Considering that maternal HPA axis functioning during pregnancy may impact both maternal (e.g., mood disorders) and fetal (e.g., birth weight) outcomes (Duthie and Reynolds, 2013), findings suggest maternal trauma history should be considered in future prevention strategies.

However, contrary to our hypotheses, hair GCs during pregnancy did not predict CB-PTSS two months postpartum. Being the first to assess this relationship with CB-PTSS, we discuss our null findings in the context of prospective research from the general PTSD literature. In their meta-analysis, Morris et al. (2016) found no link between cortisol levels measured in saliva, plasma/serum, or urine in the *acute post-traumatic phase,* and subsequent PTSS. More recent studies assessing cortisol levels via hair analysis measured *shortly after* the traumatic event found hypercortisolism to predict PTSS risk following traumatic brain injury (Pacella et al., 2017), and avoidance behaviour symptoms in motor vehicle crash victims (Petrowski et al., 2020). Variations could be due to our design involving hair samples being taken *prior to* trauma exposure (i.e., childbirth), thereby limiting confounding influences of the trauma itself (e.g., Colding-Jørgensen et al., 2020). While a prospective study among firefighters with duty-related trauma found no predictive value of HairF for PTSS development (Sopp et al., 2021), the only study assessing HairF prior to *new-onset* trauma exposure found that attenuated pre-traumatic HairF predicted a greater increase in PTSS in male soldiers who had experienced at least one traumatic event during military deployment (Steudte-Schmiedgen et al., 2015). Whilst their design is most closely comparable to ours, the outcome measure was PTSS *increase*, whereas we only assessed CB-PTSS once postpartum and therefore could not account for pre-existing PTSS. Pre-existing differences in symptom load may have obscured effects, however we did control for prenatal depression which is closely linked to CB-PTSS (Ayers et al., 2016). Moreover, in Steudte-Schmiedgen et al.'s (2015) study, the new-onset traumatic event was determined using a standardized clinical interview. In contrast, the new-onset traumatic event in relation to which we investigated PTSS in our study was childbirth, which is experienced as traumatic only by about a third of women (Heyne et al., 2022). As less than 1% of our sample scored above the clinical cut-off for CB-PTSS, it could be that we found no effect because our sample did not include enough women traumatised by their childbirth.

Our exploratory findings lend initial support to the idea that SBE represents an important factor to consider for the above research question. While none of the hair GCs were significant mediators of the relationship between trauma history and CB-PTSS, when SBE was included as a moderator, interaction effects emerged. Both HairF and HairE significantly interacted with SBE to predict CB-PTSS. Findings tentatively suggest that for individuals who had a negative SBE, lower HairE and HairF levels during the third trimester of pregnancy were associated with higher CB-PTSS. However, when hair GC levels were high, SBE did not contribute to differential CB-PTSS. Interestingly, the former is partly in line with our previous data on military personnel suggesting a predictive value of lower HairF for developing PTSD after trauma exposure and supports the assumption that the effect of HPA axis alterations for (CB)-PTSS may depend on whether a new-onset, negative (and potentially traumatic) event was experienced (Steudte-Schmiedgen et al., 2015). In this previous study, however, trauma exposure history was also reflected in lower baseline cortisol levels, whereas the current study revealed increased hair GCs in trauma-exposed expectant mothers. Although our findings concerning the role of long-term GC levels, trauma history, and developing CB-PTSS are complex, our data provide tentative support for the necessity to consider SBE in future studies in this research context. It should be considered that our one-item measure of overall birth experience could not confirm whether the childbirth experience fulfilled the diagnostic criterion for a traumatic experience. Hence, future research is needed to confirm the current findings using more detailed assessments to more accurately capture the nature and complexity of a traumatic birth experience according to diagnostic criteria. Here, the recently developed City Birth Trauma Scale allowing a detailed assessment of PTSD symptoms including the trauma criterion according to DSM-5 (Weigl et al., 2021) might be of particular relevance.

While the overall indices of moderated mediation were non-significant, results also showed that expectant mothers with trauma history reported greater subsequent CB-PTSS and that this effect became stronger the more negative the SBE, confirming the diathesis-stress-model of CB-PTSD with trauma history as a vulnerability factor for CB-PTSS (Ayers et al., 2016). This also aligns with the sensitization effect, such that prior trauma exposure may reduce the ability to successfully deal with future potentially traumatic experiences, thereby increasing risk for PTSD development (Breslau et al., 1999). Yet the assumption of HPA axis alterations as a biological explanation of this effect was not fully supported as evidenced by non-significant mediation effects. Consistent with meta-analytic research highlighting SBE as a potent risk factor for CB-PTSS (Ayers et al., 2016), SBE correlated positively with CB-PTSS at trend level and was a significant predictor of CB-PTSS in moderated mediation models. Again, our data support the consideration of SBE in future studies in this research context which is further underlined by the fact that in addition to its relevance for CB-PTSS, SBE has been shown to play a key role in other perinatal outcomes, such as bonding (Junge-Hoffmeister et al., 2022). While the above-mentioned moderated mediation findings showed tentative

effects for lifetime trauma history on hair GCs and CB-PTSS when considering SBE, this was not observed for childhood maltreatment in the present study (see supplements). This contrasts with previous assumptions suggesting early adverse experiences to be biologically embedded and increase risk for later psychopathology (Brückl and Binder, 2017).

Concerning design of future studies, measurements of GCs prior to pregnancy as well as across pregnancy appear promising. Particularly since GC levels change naturally during pregnancy (Scharlau et al., 2018), taking intra-individual changes across pregnancy into account could offer new insights into the relationship between hair GCs and CB-PTSS. Also, our findings support HairE, but not necessarily the HairE/HairF ratio, as a robust marker that should be included in future investigations. Nonetheless, some limitations should be considered when interpreting our findings. The relatively small sample size of the current study may have impeded the detection of robust effects, especially when considering that the main analyses were adjusted for numerous confounders. Further, generalization of findings is limited by our sample demonstrating above average health levels with less than 1% reporting clinically-relevant CB-PTSS – markedly less than the 3.1%–4.7% reported in meta-analyses (Grekin and O'Hara, 2014; Heyne et al., 2022). Moreover, women who dropped out had more depressive symptoms, indicating risk of self-selection bias. Thus, reported findings may apply to community samples in particular, and the development of largely *subclinical* CB-PTSS. Future research may benefit from examining these relationships in samples with greater variability regarding for instance education, ethnicity, and prior clinical status as well as among fathers, where studies report between 0% and 5% to develop CB-PTSS (e.g. Heyne et al., 2022; Kress et al., 2021). In addition, it should be noted that SBE was assessed two months postpartum. Ideally, SBE should be measured as soon as possible after birth. However, it is often not feasible to administer questionnaires to mothers immediately after they have given birth (Garthus-Niegel et al., 2013). Thus, it is conceivable that other factors (e.g., depressive symptoms) may have affected mothers' retrospective SBE ratings. However, we have partly accounted for a potential confounding effect through statistical control of prenatal depressiveness that has been shown to be related with postnatal depressiveness (Hutchens & Kearney, 2020). Finally, it should be mentioned that despite that the current research included assessments of trauma history and childhood maltreatment, no information on prior PTSD symptomatology due to other traumatic events were obtained. Hence, we cannot rule out that women may have suffered from PTSD symptoms in relation to a trauma, irrespective of childbirth. Future studies should thus include measurements of PTSD symptomatology separately for a birth and non-birth-related index trauma.

5. Conclusion

Overall, findings indicate that elevated HairF and HairE during the third pregnancy trimester correlate with previous trauma exposure, albeit this was not revealed when correcting for multiple testing. Specifically, expectant mothers with a history of trauma showed elevated HairF and HairE during pregnancy and reported greater CB-PTSS. Our data provided preliminary evidence that the predictive value of HairF and HairE for CB-PTSS was dependent upon the level of SBE, suggesting hair GCs during the third trimester may be a valuable biological predictor of subsequent CB-PTSS when considering SBE. Future research should examine the predictive value of hair GCs measured prenatally and throughout pregnancy accompanied by assessment of SBE in more severely affected samples to determine additional risk biomarkers and inform biological prevention strategies.

CRediT authorship contribution statement

Susann Steudte-Schmiedgen: Conceptualization, Resources, Supervision, Writing – review & editing, Project administration. **Sarah Schälicke**: Conceptualization, Investigation, Formal analysis, Writing – original draft, Writing – review & editing, Visualization. **Luisa Bergunde**: Conceptualization, Investigation, Formal analysis, Writing – review & editing, Visualization. **Marlene Karl**: Investigation, Data curation, Writing – review & editing. **Victoria Weise**: Investigation, Data curation, Writing – review & editing. **Juliane Junge-Hoffmeister**: Resources, Writing – review & editing. **Sarah Schumacher:** Writing – review & editing. **Tilmann von Soest:** Formal analysis, Writing – review & editing. **Kerstin Weidner**: Resources, Writing – review & editing. **Clemens Kirschbaum**: Resources, Writing – review & editing. **Susan Garthus-Niegel**: Conceptualization, Resources, Supervision, Writing – review & editing, Project administration, Funding acquisition.

Conflict of interest

The authors report no conflict of interest.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2022.105973.

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