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Research paper

Prenatal insomnia and childbirth-related PTSD symptoms: A prospective population-based cohort study

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ABSTRACT

Background: Certain populations are at high risk of experiencing a traumatic event and developing post-traumatic stress disorder (PTSD). Yet, primary preventive interventions against PTSD are lacking. It is therefore crucial to identify pre-traumatic risk factors, which could be targeted with such interventions. Insomnia may be a good candidate, but studies on civilians are sparse. Furthermore, the mechanisms at stake in the relationship between pre-traumatic insomnia and PTSD symptoms are unclear.

Methods: This prospective population-based cohort study (n = 1,610) examined the relationship between insomnia symptoms at 32 weeks of pregnancy and childbirth-related PTSD (CB-PTSD) symptoms at eight weeks postpartum. Postnatal insomnia symptoms, prenatal psychological symptoms (depression, anxiety, PTSD, fear of childbirth), subjective birth experience (SBE) and birth medical severity were included as covariates in the analyses, which were based on a Piecewise Structural Equation Modelling approach.

Results: The relationship between prenatal insomnia and CB-PTSD symptoms was mediated by negative SBE and postnatal insomnia symptoms. All relationships involving insomnia symptoms had small or very small effect sizes.

Limitations: This study used self-report questionnaires. Postnatal insomnia and CB-PTSD symptoms were concurrently measured.

Conclusion: Prenatal insomnia symptoms may impair the ability to cope with a difficult birth experience and contribute to postnatal insomnia, a risk factor for CB-PTSD. Thus, prenatal insomnia symptoms may be a promising target for CB-PTSD primary preventive interventions, although other prenatal psychological symptoms could also be considered. Even beyond the perinatal context, future studies on pre-traumatic insomnia and PTSD should include post-traumatic insomnia as a covariate.

Abbreviation: ABC, Akershus Birth Cohort; BIS, Bergen Insomnia Scale; CB-PTSD, Childbirth-related Post-Traumatic Stress Disorder; CES, Combat Exposure Scale; DSM, Diagnostic and Statistical Manual of Mental Disorders; ECS, Emergency caesarean section; EPDS, Edinburgh Postnatal Depression Scale; FOC, Fear Of Childbirth; GPCM, Generalised Partial Credit Model; IES, Impact of Event Scale; IES-R, Impact of Event Scale-Revised; IRM, Item Response Model; MINI, Mini International Neuropsychiatric Interview; MVC, Motor Vehicle Accident; PCA, Principal Component Analysis; RESI, Robust Effect Suze Index; SCL, Hopkins Symptom Checklist; SEM, Structural Equation Modelling; SBE, Subjective Birth Experience; VIF, Variance Inflation Factor.

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1. Introduction

Triggered by a traumatic experience, post-traumatic stress disorder (PTSD) is a mental health disorder with a lifetime prevalence of 3.9% (Koenen et al., 2017). Its four symptoms clusters, which have to be present at least one month post-trauma, are: re-experiencing symptoms (including distressing trauma-related dreams), avoidance of trauma-related stimuli, alterations in arousal (including sleep disturbances), and negative cognitions and mood (American Psychiatric Association, 2013). Certain populations, such as service members, are at increased risk of experiencing a traumatic event and developing PTSD (Stefanovics et al., 2020). This is also the case for women during the perinatal period, given that childbirth-related PTSD (CB-PTSD) affects 4% of mothers in community samples, and 18.5% in high-risk samples (Yildiz et al., 2017). Overall, these groups would benefit from primary preventive interventions for PTSD, but they are lacking (Howlett and Stein, 2016). To develop such evidence-based interventions, it is crucial to identify modifiable risk factors, at stake before trauma exposure.

In that respect, sleep disturbances could be a promising intervention target. It is well documented that they are associated with impaired mental health (Freeman et al., 2020) and disrupted physiological functioning (e.g., blunted diary cortisol trajectory, increased risk of hypertension, and higher body mass index) (Bei et al., 2017; Medic et al., 2017). Insomnia, in particular, which is characterised by difficulty falling or staying asleep and associated with distress or functional impairment (American Psychiatric Association, 2013), may contribute to PTSD development. As an illustration, pre-deployment insomnia predicted post-deployment PTSD in service members (Gehrman et al., 2013; Wang et al., 2019), although one study did not find such a relationship (van Liempt et al., 2013). In civilians, pre-traumatic insomnia symptoms also predicted PTSD(-like) symptoms following a traumatic injury (Bryant et al., 2010), an analogue trauma (Short et al., 2020), or a motor vehicle collision (MVC) (via, in the latter case, acute stress disorder) (Neylan et al., 2021). Importantly, the relationship between insomnia and PTSD seems to remain after controlling for other psychological symptoms, such as pre-traumatic depression, anxiety, and PTSD (e.g., Wang et al., 2019). This suggests that pre-traumatic insomnia may not only be a symptom of other psychopathologies: it could also be an independent predictor of PTSD symptom development (Cox et al., 2017; Kartal et al., 2021), albeit contradictory findings have been reported in service members (van Liempt et al., 2013),

One of the proposed mechanisms to explain this relationship is that insomnia fosters maladaptive responses to the trauma (Bryant et al., 2010; Cox et al., 2017) by disturbing emotional regulation (Tempesta et al., 2018), executive functions (Cox and Olatunji, 2016), and encoding (Cousins and Fernández, 2019; Krause et al., 2017), thus increasing the stressor's psychological impact (Bryant et al., 2010). This could explain why combat-related stress severity partially moderated the relationship between pre-deployment sleep disturbances and subsequent PTSD – although, importantly, this was not found in the replication sample (Acheson et al., 2019). One study also reported that subjective peritraumatic distress mediated the relationship between prior insomnia and PTSD-like symptoms after exposure to an analogue trauma (Short et al., 2020), although, in another study, pre-traumatic insomnia did not appear to predict peritraumatic distress in MVC survivors (Neylan et al., 2021). Overall, these mixed results suggest that it is important to take into account both the stressor severity and the subjective response to it when investigating the potential link between pre-traumatic insomnia symptoms and PTSD.

Given the chronicity of insomnia, a second hypothesis could be that individuals with pre-traumatic insomnia also have *post*-traumatic insomnia, which has been shown to directly contribute to the aggravation or maintenance of PTSD symptoms (Biggs et al., 2020; Garthus-Niegel et al., 2015; Zeng et al., 2020). Post-traumatic insomnia involvement in PTSD pathogenesis may be linked with 1) the disruption of memory consolidation (Azza et al., 2020; Cousins and Fernández, 2019; Sopp et al., 2021), which corresponds to the stabilisation of the memory trace into long-term memory and is suspected to be impaired in PTSD (van Marle, 2015), and 2) the disruption of fear extinction or safety learning prevention (Colvonen et al., 2019; Seo et al., 2021), both allowing for a reduction of the fear response. Thus, post-traumatic insomnia emerges as a crucial factor, explaining by itself the relationship between pre-traumatic insomnia and PTSD. To our knowledge, however, post-traumatic insomnia has not been taken into account in previous studies. Clarifying its role therefore seems important to conclude on the relevance of treating pre-traumatic insomnia to prevent PTSD.

Furthermore, in civilians, pre-traumatic sleep and covariates are usually retrospectively measured, as it is difficult to recruit participants before trauma exposure (e.g., Bryant et al., 2010; Neylan et al., 2021). However, retrospective measurements, in particular following a traumatic event, may be subject to memory bias. Moreover, the literature on pre-traumatic insomnia and PTSD symptoms sometimes relies on sleep measurements reconstituted from different instruments or single-item questions (e.g., Gehrman et al., 2013; van Liempt et al., 2013), which reduces their validity. Finally, most studies focus on service members, yet the weight of PTSD risk factors varies across groups (Brewin et al., 2000), thus the evidence collected in this population may not be valid in other groups. In addition, the vast majority of service members are men, whereas insomnia prevalence is higher in women (Suh et al., 2018; Zhang and Wing, 2006), and sex could affect the relationship between sleep and PTSD (Kobayashi et al., 2007; Kobayashi and Delahanty, 2013). To develop appropriate preventive interventions, it would therefore be crucial to conduct studies in new populations.

In this study, we examined the prospective relationship between prenatal insomnia symptoms and subsequent CB-PTSD symptoms. Beyond the fact that CB-PTSD is relatively common (Yildiz et al., 2017) and that women are underrepresented in the literature on pre-traumatic sleep and PTSD, pregnancy follow-up allows prospective measurement of pre-traumatic (i.e., pre-natal) insomnia symptoms and other covariates. Furthermore, traumatic childbirth represents a relatively standardised real-life trauma (homogenous population; mostly taking place in a care setting) and thus a good study model for PTSD. Hence, studying the relationship between insomnia and PTSD in the perinatal context may help to overcome the limitations identified in the current literature.

Previous research found that 39.7% of women suffer from insomnia during the third trimester of pregnancy (Sedov et al., 2021), and that it is also common postnatally (Dorheim et al., 2014; Sivertsen et al., 2015). Prenatal insomnia may, inter alia, be caused by pregnancy-related nocturnal ruminations (Swanson et al., 2020), potentially reflecting fear of childbirth (FOC). FOC has shown to be more intense in pregnant women with insomnia than in those without (Osnes et al., 2020), and is a risk factor for CB-PTSD (Ayers et al., 2016; Garthus-Niegel et al., 2014). Prenatal insomnia has also well documented links with prenatal anxiety and prenatal depression symptoms (Emamian et al., 2019; Osnes et al., 2020; Sedov and Tomfohr-Madsen, 2021). Its prospective relationship with CB-PTSD, however, has received little attention so far, although insomnia at eight weeks postpartum has been identified as a maintaining factor of CB-PTSD at two years postpartum (Garthus-Niegel et al., 2015).

In summary, pre-traumatic insomnia symptoms may be a risk factor for PTSD symptom development. If so, it could be a relevant target for PTSD primary preventive intervention. However, current evidence is mixed and most of the available studies concern male service members or suffered from significant limitations, which limits the results' generalisability. The objective of this prospective population-based cohort study was to examine the relationship between prenatal insomnia symptoms at 32 weeks of pregnancy and CB-PTSD symptoms at eight weeks postpartum, taking postnatal insomnia symptoms into account and controlling for prenatal psychological symptoms (depression, anxiety, and PTSD symptoms as well as FOC) and childbirth-related factors (subjective birth experience (SBE), birth medical severity). We hypothesised that prenatal insomnia symptoms would predict CB-PTSD

symptoms, but that this relationship would be mediated by postnatal insomnia symptoms and SBE.

2. Method

2.1. Design and study population

This study was derived from the Akershus Birth Cohort (ABC), a large

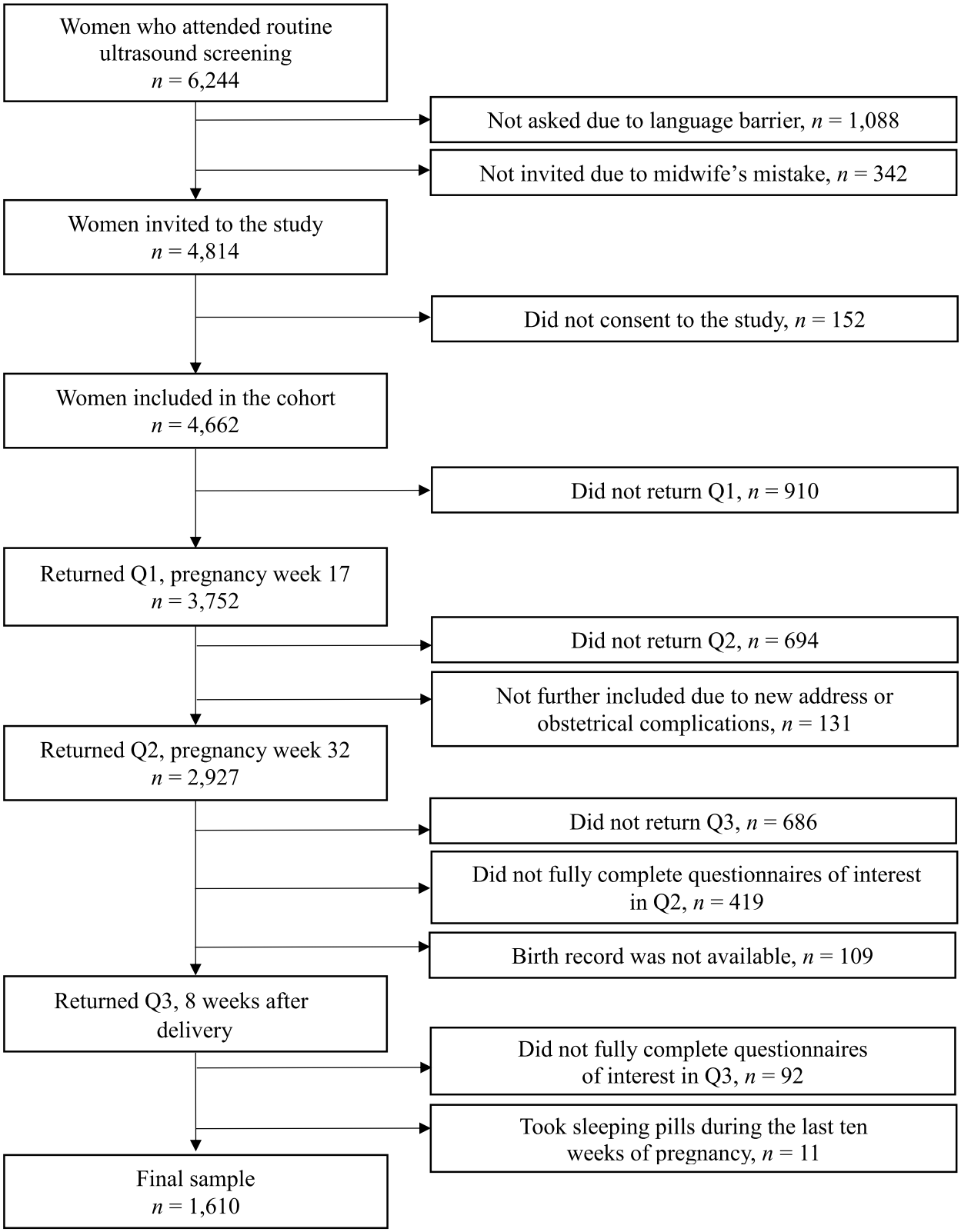

Fig. 1. Responses to the study questionnaires.

population-based prospective cohort study. The ABC targeted all women planning to give birth between November 2008 and April 2010 at Akershus University Hospital (Norway), which serves around 350,000 inhabitants. Women were eligible to participate if they were able to complete questionnaires in Norwegian. They were recruited at 17 weeks of pregnancy, during their routine foetal ultrasound examination. Participation involved completing paper questionnaires at 17 weeks and 32 weeks of pregnancy, and at eight weeks and two years postpartum. Participants who delivered between May 2009 and September 2010 answered additional questions about SBE at 48 hours postpartum. Obstetrical information was retrieved from hospital birth records. The ABC was approved by the Regional Committee for Medical and Health Research Ethics (approval number S-08013a); all participants provided written informed consent.

In this study, we used data from the questionnaires at 17 weeks and 32 weeks of pregnancy, as well as the 8-week postpartum questionnaires and the hospital birth record. Of the eligible women, 80% ($N = 3,752$) agreed to participate and returned the 17-week questionnaires, of whom 131 were excluded from the cohort study due to new address or obstetrical complications (see Garthus-Niegel et al. (2018a) for detailed information on participants). Women were further excluded from the current study for the following reasons: having taken sleeping pills during the last ten weeks of pregnancy (because it could have biased the insomnia measures), missing hospital birth record (rendering birth medical severity assessment impossible) or missing data in the questionnaires of interest (rendering computation of total scores impossible). The final sample consisted of 1,610 participants (Fig. 1).

2.2. Measures

Because a Structural Equation Modelling (SEM) approach was planned, and in order to obtain numeric scores, all questionnaires were statistically treated using item response models (IRM) or factor techniques.

2.2.1. Childbirth-related PTSD symptoms

CB-PTSD symptoms were measured at eight weeks postpartum, with the Impact of Event Scale (IES) (Horowitz et al., 1979). Participants were instructed to complete it in relation to their last childbirth. The IES is a 15-item self-rating questionnaire that has been validated in postpartum women (Olde et al., 2006). Each item has four response categories (usually recoded with the following weightings: 0 = "not at all", 1 = "rarely", 3 = "sometimes", and 5 = "often"). The sum score of the overall scale ranges from 0 to 75, with scores above 34 indicating probable PTSD (Neal et al., 1994). In this study, however, the ordinal nature of the response scale was taken into account by using a polytomous IRM (Hambleton et al., 1991) to compute a total IES score (see Supplementary material, section 1.1.).

2.2.2. Insomnia symptoms

Insomnia symptoms were self-reported at 32 weeks of pregnancy and eight weeks postpartum through the Bergen Insomnia Scale (BIS), a six-item self-rating scale which has been validated against polysomnographic data (Pallesen et al., 2008). Four items assess sleep impairment (which corresponds to criterion A for insomnia in DSM-IV-TR (American Psychiatric Association, 2000)), and two items respectively assess daytime impairment linked with sleepiness or sleep-related dissatisfaction (criterion B for insomnia in the DSM-IV-TR (American Psychiatric Association, 2000)). In the original scoring, items are scored from 0 to 7, reflecting the frequency of each symptom per week, over the past month. The BIS total score ranges from 0 to 42, with higher scores reflecting more severe insomnia symptoms. Because previous validation studies have retained one or two factor solutions depending on the calibration sample, a Principal Component Analysis (PCA) was run to check for dimensionality in the present data. A one-factor solution was finally retained (see Supplementary material, section 1.2.).

2.2.3. Prenatal depression symptoms

Prenatal depression symptoms over the past week were measured by the 10 item self-report Edinburgh Postnatal Depression Scale (EPDS) (Cox et al., 1987), at 32 weeks of pregnancy. The EPDS has been validated in pregnant women (Bergink et al., 2011; Bunevicius et al., 2009). Each item has four response categories, ranging from 0 to 3, yielding a total score from 0 to 30, with a higher score indicating more severe depression symptoms. In this study, given the categorical nature of responses, an IRM was adopted for score computation (see Supplementary material, section 1.3.).

2.2.4. Prenatal anxiety symptoms

Prenatal anxiety symptoms were reported at 32 weeks of pregnancy through the 10-item anxiety scale (SCL-A) of the Hopkins Symptom Checklist (SCL-25) (Derogatis et al., 1974). Each item assesses one anxiety symptom over the past week and has four response categories, from 1 = "not at all" to 4 = "extremely". The total score in the original scoring ranges from 10 to 40, higher scores reflecting more severe anxiety symptoms. Given the categorical nature of the data and previous successful item response modelling of the SCL (Kleppang and Hagquist, 2016), responses to the SCL-A were also analysed with an IRM (see Supplementary material, section 1.4.).

2.2.5. Prenatal PTSD symptoms

Prenatal PTSD symptoms were assessed at 17 weeks of pregnancy. Women reported whether and which of eight PTSD symptoms they experienced over the past month, in relation to a dramatic or terrifying event they potentially experienced. This PTSD symptom checklist was based on the Mini International Neuropsychiatric Interview (MINI) (Sheehan et al., 1998), see Garthus-Niegel et al. (2018a) for a full description of this scale. Symptoms were scored as 0 = "absent" or 1 = "present"; with the total score ranging from 0 to 8. The prenatal PTSD symptoms score was computed in this study with a dichotomous IRM (see Supplementary material, section 1.1.).

2.2.6. Fear of childbirth

FOC was assessed at 32 weeks of pregnancy, with the 33-item self-report Wijma Delivery Expectancy/Experience Questionnaire version A (W-DEQ) (Wijma et al., 1998). The W-DEQ evaluates women's cognitive appraisal of the forthcoming birth, with six response categories scored from 0 to 5, and a total score ranging from 0 to 165 in the original scoring. In this study, scores were computed as a factor score on the first component of a standard PCA (see Supplementary material, section 1.5.), were higher values indicated more severe FOC.

2.2.7. Subjective birth experience

SBE was measured at eight weeks postpartum, with three items ("How frightened were you during the birth?"; "What was your overall experience of the birth?", and "To what degree did you feel taken care of during the birth?"). The first two were answered on a 10-point scale from 0 = "not frightened at all"/"very good", respectively to 10 = "extremely frightened"/"extremely bad", respectively. The last item was answered on a 4-point scale, from 1 = "very good" to 4 = "very bad". SBE has been shown to be a major risk factor for CB-PTSD (Ayers et al., 2016), notably when measured with these three items (Garthus-Niegel et al., 2013). In the present study, a PCA revealed a strong one-factor solution, with high scores reflecting a negative experience (see Supplementary material, section 1.6.).

2.2.8. Birth medical severity

Birth medical severity was expressed with a dichotomous variable, coded 1 if the birth involved vacuum, forceps, or an emergency caesarean section (ECS), or 0 if not. This information was retrieved from hospital birth records. The objective of the variable was to reflect birth-related stress exposure, comparable to the Combat Exposure Scale (CES) used in military populations to assess combat-related stress exposure

(Cox et al., 2018). In the absence of a brief validated equivalent for childbirth, we chose operative birth as a surrogate. Indeed, although birth can be stressful in many respects, operative birth is an important indicator of the urgency of the birth and can be particularly stressful (Gamble and Creedy, 2005)

2.2.9. Parity

Based on participants' report at 17 weeks of pregnancy, parity was coded 0 = "nulliparous" and 1 = "parous".

2.2.10. Sociodemographic characteristics

Marital status (1 = "married/cohabiting" and 0 = "single"), education (0 = "elementary school", 1 = "high school", and 2 = "higher degree"), and maternal age at the time of birth were extracted from hospital birth records.

2.3. Data analysis

2.3.1. Sample description

Descriptive analyses were conducted with IBM SPSS 27. The difference between prenatal and postnatal insomnia scores was calculated with a Wilcoxon Signed-Ranks test, given that the data were skewed.

2.3.2. Piecewise structural equation modelling

Because several causal hypotheses involving a set of potentially mediating/moderating variables were to be tested in this study, a Structural Equation Modelling approach was adopted (Ullman and Bentler, 2003) using R version 4.0.4 (R Core Team, 2021). As many women reported no CB-PTSD symptoms, a particularity of the data was a strong bimodal zero-inflated distribution of the CB-PTSD variable (see Figure S1 in Supplementary material, Section 2). This was taken into account by assuming a Tweedie distribution for this variable, which is appropriate for positive non-symmetric data, and can take a zero-inflated bimodal shape for some parameter values (Jorgensen, 1987). This particularity precluded a traditional SEM analysis of the variance-covariance matrix. Thus, a piecewise SEM approach (Shipley, 2000), using the piecewiseSEM R package (Lefcheck, 2016), was chosen as it can flexibly accommodate non-gaussian cases. Piecewise SEM allows connecting a set of generalised linear models within a network of hypothesised relationships. Apart from the Tweedie submodel on CB-PTSD symptoms, all other pieces in the model were standard gaussian linear models. The global fit of the piecewise SEM was examined through a Fisher's C statistic. Admissible but non-included relationships were tested through a set of directed separation tests (Shipley, 2013) for each model.

Three layers of variables were connectable in the model: prenatal variables, birth-related variables (birth medical severity, SBE), and postnatal variables. Because of this temporal structure, not all orientated connections were possible, which helped to define a maximal model including all admissible connections, with all variables from a given layer predicting all variables in subsequent layers.

A good predictive model of CB-PTSD symptoms was predefined as: 1) being inclusive: it includes, and potentially extends, a minimal set of relationships that are well-documented in previous studies; 2) being parsimonious: it includes only the minimal set of relationships required to reach acceptable fit; 3) being strongly connected: it only includes statistically significant relationships, and does not include relationships for which conditional independence tests given the current model are non-significant; and 4) showing a good fit, in terms of Fisher's C statistic. Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) were also computed, as they allow for comparison between models that have the same numbers of parameters (e.g., models 11 and 13 in Table 2).

Seven theoretically expected relationships were taken as a starting point in the modelling sequence. The following causal influences were a priori assumed: 1) prenatal depression symptoms to prenatal insomnia symptoms (Dorheim et al., 2012; Sedov and Tomfohr-Madsen, 2021); 2) prenatal anxiety symptoms to prenatal insomnia symptoms (Osnes et al., 2020; Sedov and Tomfohr-Madsen, 2021); 3) FOC to prenatal insomnia symptoms (Swanson et al., 2020); 4) prenatal insomnia symptoms to CB-PTSD symptoms; 5) SBE to CB-PTSD symptoms (Garthus-Niegel et al., 2013); 6) birth medical severity to CB-PTSD symptoms (Ayers et al., 2016); and 7) birth medical severity to SBE (Garthus-Niegel et al., 2013). Because they are repeated measurements, it also appeared statistically meaningful to add 8) prenatal insomnia symptoms to postnatal insomnia symptoms. All eight relationships were gathered into a first basic structural model (M1, Table 2).

3. Results

3.1. Characteristics of the study sample

At birth, mean maternal age was 31.29 years ($SD = 4.54$) (Table 1). The majority of women had a higher education degree and were married/cohabiting. Half of them were nulliparous, and 20.3% of the deliveries involved forceps, vacuum, or ECS. At eight weeks postpartum, 1.9% of them had probable CB-PTSD according to the original IES scoring. Insomnia original scores at 32 weeks of pregnancy were significantly higher than at 8 weeks postpartum, $Z = -6.303$, $p < .001$ (Table 1).

3.2. Prediction of CB-PTSD symptoms

The first basic model did not in itself show acceptable global fit (Fisher's $C = 946.444$, d.f. 36, $p < .001$) but all regression coefficients, in particular the prenatal insomnia symptoms – CB-PTSD symptoms relationship, were significant ($p < .001$). Examination of the D-separation tests suggested that several relationships ignored in the first model had a statistical significance, and could thus be included to improve the global fit. Prenatal insomnia symptoms ceased to directly predict CB-PTSD symptoms once anxiety symptoms were included as direct predictors of CB-PTSD. Sequentially including all relationships with a significant D-separation test (high criterion values first) resulted in an acceptable model fit (M12: Fisher's $C = 20.458$, d.f. 14, $p = .116$), once the prenatal insomnia – SBE relationship was added. Within M12, only the prenatal insomnia – CB-PTSD symptoms regression coefficient was non-significant ($\beta = -0.0016$, s.e. = 0.0160, d.f. 1603, $t = -0.1021$, $p = .919$). A reduction of deviance test, comparing M12 with a reduced model discarding this link, was non-significant ($\chi^2(2) = 0.397$, $p = .82$). The final model (M13) met all four prior-mentioned quality criteria, in particular global fit (Fisher's $C = 20.855$, d.f. 16, $p = .184$) and strong connectivity (Table 3). In the final model, prenatal insomnia symptoms predicted CB-PTSD symptoms through SBE and postnatal insomnia symptoms (Table 4; Fig. 2).

The robustness of M13 was tested by beginning the sequence from the full model including all admissible connections, and then removing, in turn, all non-significant terms. This backward procedure yielded the same final model as M13. Furthermore, removing any link in this model resulted in a non-acceptable fit of the modified model; a significant conditional dependence test on the removed term, and a significant relative increase in deviance (as measured by a reduction of deviance test). In order to ensure that the presence of two sleep disturbance-related items (one in the EPDS, one in the prenatal PTSD symptoms checklist) was not inflating the correlations of these questionnaires with insomnia measures (thus potentially affecting model fit and structure), the whole analysis was re-run without these two items. The very same structural model emerged, with almost identical parameter values and effect sizes. A choice was made to keep the model obtained from the original scales.

Table 1
Characteristics of the study sample (n = 1,670).

Sample characteristics (*time point measured or range*)	Frequency (%)	Mean (*SD*)
Sociodemographic characteristics		
Age (*at birth*)		31.29 (4.54)
Education (*at birth*)		
Elementary	43 (2.7[a])	
High school	439 (27.3[a])	
Higher degree	1.072 (66.6[a])	
Marital status (*at birth*)		
Married or cohabiting	1,565 (97.2[a])	
Single	35 (2.2[a])	
Prenatal psychological symptoms		
Insomnia symptoms (*pregnancy week 32*)		
Score[b] (*0–42*)		17.45 (10.36)
PCA score[c] (*-1.58–2.67*)		0.19 (1.05)
Depression symptoms (*pregnancy week 32*)		
Score[b] (*0–24*)		4.90 (4.11)
IRM score[c] (*-0.69–0.37*)		0.01 (0.15)
Anxiety symptoms (*pregnancy week 32*)		
Score[b, d] (*10–32*)		12.80 (3.10)
IRM score[c] (-0.98–*2.81*)		-0.03 (0.83)
Prenatal PTSD symptoms (*pregnancy week 17*)		
Score[b] (*0–8*)		0.25 (0.77)
IRM score[c] (*-0.25–3.18*)		-0.04 (0.54)
Fear of childbirth (*pregnancy week 32*)		
Score[b] (*2–145*)		57.44 (19.64)
PCA score[c] (*-2.74–4.44*)		0.02 (0.98)
Birth-related variables		
Parity (*pregnancy week 17*)		
Nulliparous	812 (50.4)	
Parous	798 (49.6)	
Subjective birth experience (8 weeks postpartum)		
Score [b]		
"How frightened" (*0–10*)		2.96 (2.90)
"Overall experience" (*0–10*)		2.89 (2.69)
"Taken care of" (*1–4*)		1.37 (0.63)
PCA score[c] (*-1.14–3.81*)		0.00 (0.99)
Birth medical severity (forceps, vacuum, ECS)	327 (20.3)	
Postnatal psychological symptoms		
Insomnia symptoms (*8 weeks postpartum*)		
Score[b] (*0–42*)		15.57 (8.92)
PCA score[c] (*-1.58–2.67*)		-0.03 (0.91)
CB-PTSD symptoms (*8 weeks postpartum*)		
Score[b] (*0–52*)		7 (8)[d]
Score > 34, probable CB-PTSD[b]	30 (1.9)	
IRM score[c] (*0–3.94*)		1.31 (0.89)

Note. PCA = Principal Component Analysis; IRM = Item Response Model; CB-PTSD = Childbirth-related PTSD.
[a] Total of % does not equal 100 because of missing values (n = 1,554 for education; n = 1,600 for marital status).
[b] Scores derived from the original scoring. Reported for descriptive purpose only.
[c] Scores computed using a PCA or an IRM, used in the analyses.
[d] Computed on n = 1601 because of missing items.

4. Discussion

In this population-based prospective cohort study, the direct relationship between prenatal insomnia symptoms at 32 weeks of pregnancy and CB-PTSD symptoms at eight weeks postpartum was initially significant, but disappeared when controlling for postnatal insomnia symptoms, prenatal psychological symptoms, and childbirth-related factors. More precisely, it was not significant anymore once the prenatal anxiety – CB-PTSD relationship was added to the model, suggesting that it was mostly the anxious component of prenatal insomnia that predicted CB-PTSD symptoms. Prenatal psychological symptoms (depression, anxiety, PTSD, and FOC) all predicted prenatal insomnia symptoms. Except for depression symptoms, they also predicted postnatal insomnia symptoms, thus confirming their relevance as covariates.

Pursuant to our hypothesis, both negative SBE and postnatal insomnia symptoms mediated the relationship between prenatal insomnia and CB-PTSD symptoms. In addition, postnatal insomnia partially or fully mediated the relationship between prenatal psychological symptoms (except depression) and CB-PTSD, thus suggesting that postnatal insomnia symptoms may be one of the pathways through which prenatal psychopathology renders women vulnerable to CB-PTSD. Importantly, all relationships involving insomnia symptoms had small or very small effect sizes. The four other predictors of CB-PTSD symptoms were birth medical severity, SBE, and prenatal anxiety and depression symptoms, which is in line with the existing evidence (Ayers et al., 2016; Garthus-Niegel et al., 2013). However, in contrast with previous research (Ayers et al., 2016; Grekin et al., 2021), neither prenatal PTSD symptoms nor FOC directly predicted CB-PTSD symptoms, although Grekin et al. (2021), Garthus-Niegel et al. (2013) and Garthus-Niegel et al. (2014) also found that SBE partially or fully mediated the relationship between FOC and CB-PTSD symptoms.

The fact that prenatal insomnia symptoms predicted CB-PTSD through a negative SBE suggests that they are likely to reduce psychological resources for coping with a difficult childbirth. Hence, they may be one of the vulnerability factors explaining why some women show a maladaptive stress response to a difficult childbirth and develop CB-PTSD symptoms. As for postnatal insomnia symptoms, they had already been identified as predictors and maintaining factors of CB-PTSD symptoms in the postpartum period (Garthus-Niegel et al., 2015). However, our results add an important element to the picture, as they show these symptoms also mediate the relationship between prenatal insomnia and CB-PTSD. Therefore, prenatal insomnia symptoms could both reduce the resources for coping with a traumatic birth and hinder post-traumatic recovery by increasing the likelihood of

Table 2
Model construction and comparison.

Model	Links	AIC	BIC	Fisher C	Model d.f.	Model p value	Fisher C difference	D.f. diff.	*p* value
M1[a]	Basic model	976.444	1057.204	946.444	36	< 0.001			
M2	Fear of childbirth → Subjective birth experience	629.995	716.139	597.995	34	< 0.001	348.449	2	< 0.001
M3	Prenatal anxiety symptoms → CB-PTSD symptoms	405.227	496.755	371.227	32	< 0.001	226.768	2	< 0.001
M4	Prenatal anxiety symptoms → Postnatal insomnia symptoms	279.334	376.246	243.334	30	< 0.001	127.893	2	< 0.001
M5	Prenatal anxiety symptoms → Subjective Birth Experience	211.702	313.998	173.702	28	< 0.001	69.632	2	< 0.001
M6	Prenatal PTSD symptoms → Prenatal insomnia symptoms	185.038	292.718	145.038	26	< 0.001	28.664	2	< 0.001
M7	Fear of childbirth → Postnatal insomnia symptoms	152.329	265.393	110.329	24	< 0.001	34.709	2	< 0.001
M8	Subjective birth experience → Postnatal insomnia symptoms	129.439	247.887	85.439	22	< 0.001	24.89	2	< 0.001
M9	Prenatal PTSD symptoms → Postnatal insomnia symptoms	110.732	234.564	64.732	20	< 0.001	20.707	2	< 0.001
M10	Prenatal depression symptoms → CB-PTSD symptoms	91.309	220.525	43.309	18	.001	21.423	2	< 0.001
M11	Postnatal insomnia symptoms → CB-PTSD symptoms	80.206	214.806	30.206	16	.017	13.103	2	.001
M12	Prenatal insomnia symptoms → Subjective birth experience	72.458	212.442	20.458	14	.116	9.748	2	.008
M13[b]	Prenatal insomnia symptoms → CB-PTSD symptoms (removed)	70.855	205.455	20.855	16	.184	0.397	2	0.82

Note. CB-PTSD = Childbirth-related PTSD.
[a] M1 includes the eight basic links mentioned in the data analysis section.
[b] M13 is the last and best model. It removes the non-significant Prenatal insomnia symptoms → CB-PTSD symptoms relationship from the previous one, M12.

Table 3
D-separation tests of relationships not included in the final model.

Independence claim	D.f.	Criterion	*p* value
Fear of childbirth → CB-PTSD symptoms	1603	0.7267	.468
Prenatal insomnia symptoms → CB-PTSD symptoms	1601	-0.2624	.793
Prenatal PTSD symptoms → CB-PTSD symptoms	1603	1.4421	.150
Prenatal depression symptoms → Subjective birth experience	1604	1.6788	.093
Prenatal PTSD symptoms → Subjective birth experience	1604	-0.8283	.408
Prenatal depression symptoms → Postnatal insomnia symptoms	1603	1.3867	.166
Birth medical severity → Postnatal insomnia symptoms	1603	1.0776	.281
Prenatal insomnia symptoms → Birth medical severity	1604	-1.0346	.301

Note. CB-PTSD = Childbirth-related PTSD. The reported criterion values are conditional tests that a relationship is significant, given all links in the final model (M13).

experiencing postnatal insomnia. These two mediations were cumulative in our final model and appeared to connect, as negative SBE predicted postnatal insomnia symptoms. It is possible that this relationship results from ruminations, anxiety, infant-related worries, or even acute stress disorder (Neylan et al., 2021) caused by a negative SBE, which would trigger insomnia.

Clinically, our results tentatively suggest that targeting prenatal insomnia symptoms may, by improving SBE and reducing postnatal insomnia, be a primary prevention pathway for CB-PTSD. However, it should be remembered that the effect sizes observed were all small or very small, thus further studies are needed to clarify whether interventions focusing on prenatal insomnia do yield significant benefits for CB-PTSD prevention or not. Reducing prenatal insomnia symptoms may also have other benefits, as insomnia puts pregnant women at risk of adverse obstetrical outcomes (Palagini et al., 2014) and may affect long-term infant development (Adler et al., 2021; Monk et al., 2019). In that respect, cognitive behavioural therapy (CBT) for insomnia is recommended in the general population (Trauer et al., 2015) and shows good acceptability during pregnancy (Sedov et al., 2017). According to the final model, CB-PTSD prevention may also involve treating prenatal depression and anxiety symptoms. Reducing the latter could help to decrease pre- and postnatal insomnia, and to improve the SBE. Moreover, interventions targeting prenatal insomnia symptoms and FOC may help to prevent a negative SBE. As for CB-PTSD prevention or treatment *after* childbirth, targeting postnatal insomnia symptoms may be a beneficial strategy, echoing a recent meta-analysis indicating that CBT for sleep disturbances leads to PTSD symptom reduction (Ho et al., 2016). Overall, the final model, like the existing literature (e.g., Ayers et al., 2016), suggests that there are several potential targets for CB-PTSD prevention (e.g., depression symptoms or FOC), some of which may be more clinically relevant than pre-traumatic insomnia.

With regard to sleep, it is true that the perinatal population has some specificities: for instance, pregnant women report more nocturnal ruminations than non-pregnant women, and the hormonal changes and pregnancy-related physical discomfort are likely to affect their sleep (Swanson et al., 2020). Furthermore, prenatal insomnia may be perceived as more normative and transient than pre-traumatic insomnia in other groups. Despite these particularities, we believe the results of this study are of interest beyond the perinatal context. The mediating role of SBE and prenatal insomnia, in particular, supports the hypotheses of reduced peritraumatic psychological resources and impaired post-traumatic recovery.

This study has important strengths. It is the first, to our knowledge, to prospectively look at the relationship between pre-traumatic insomnia symptoms and PTSD symptoms in a large population-based cohort of civilians. It is also the first to study this relationship while taking post-traumatic insomnia into account, and the first to consider these variables in the perinatal context. However, it has several limitations. Firstly, except for birth medical severity, all measures were self-reported. This may be problematic, as individuals with insomnia symptoms tend to underestimate their sleep duration (Bianchi et al., 2013). The BIS, however, has been validated against polysomnographic data (Pallesen et al., 2008) and thus represents the self-report questionnaire of choice. Secondly, operative delivery was used as a proxy for

Table 4
Coefficients of the final model.

Predictor	Explained variable	Estimate	Standard error	D.f.	Criterion	*p* value	Standard estimate	Effect size (S^2 / f^2)[a]	VIF
Prenatal anxiety symptoms	Prenatal insomnia symptoms	0.1448	0.0387	1605	3.7444	< 0.001	0.1143	0.0132	1.8021
Prenatal depression symptoms	Prenatal insomnia symptoms	0.2785	0.0365	1605	7.6334	< 0.001	0.2358	0.0589	1.8443
Fear of childbirth	Prenatal insomnia symptoms	0.0832	0.0267	1605	3.1167	.002	0.0776	0.0061	1.1970
Prenatal PTSD symptoms	Prenatal insomnia symptoms	0.2383	0.0465	1605	5.1273	< 0.001	0.1226	0.0153	1.1059
Birth medical severity	Subjective birth experience	0.4142	0.0516	1605	8.0217	< 0.001	0.1832	0.0347	1.0141
Fear of childbirth	Subjective birth experience	0.2670	0.0246	1605	10.8448	< 0.001	0.2651	0.0756	1.1618
Prenatal anxiety symptoms	Subjective birth experience	0.1541	0.0299	1605	5.1600	< 0.001	0.1295	0.0171	1.2256
Prenatal insomnia symptoms	Subjective birth experience	0.0618	0.0227	1605	2.7215	.007	0.0658	0.0043	1.1382
Prenatal insomnia symptoms	Postnatal insomnia symptoms	0.2875	0.0206	1604	13.9843	< 0.001	0.3313	0.1233	1.1670
Prenatal anxiety symptoms	Postnatal insomnia symptoms	0.1032	0.0277	1604	3.7286	< 0.001	0.0939	0.0089	1.3175
Fear of childbirth	Postnatal insomnia symptoms	0.0812	0.0228	1604	3.5656	< 0.001	0.0872	0.0077	1.2443
Subjective birth experience	Postnatal insomnia symptoms	0.1012	0.0219	1604	4.6258	< 0.001	0.1095	0.0121	1.1650
Prenatal PTSD symptoms	Postnatal insomnia symptoms	0.1479	0.0391	1604	3.7835	< 0.001	0.0877	0.0078	1.1161
Subjective birth experience	CB-PTSD symptoms	0.2176	0.0140	1604	15.5420	< 0.001	-	(*) 0.1789	1.1561
Birth medical severity	CB-PTSD symptoms	0.1111	0.0327	1604	3.4014	< 0.001	-	(*) 0.0072	1.0515
Prenatal anxiety symptoms	CB-PTSD symptoms	0.1341	0.0229	1604	5.8512	< 0.001	-	(*) 0.0217	1.7738
Prenatal depression symptoms	CB-PTSD symptoms	0.0781	0.0220	1604	3.5543	< 0.001	-	(*) 0.0073	1.7757
Postnatal insomnia symptoms	CB-PTSD symptoms	0.0559	0.0167	1604	3.3376	< 0.001	-	(*) 0.0068	1.1381

Note. CB-PTSD = Childbirth-related PTSD, VIF = Variance Inflation Factor.
[a] Cohen's (1988) f^2 indices were used to measure effect sizes, except for predictors of CB-PTSD (marked with an asterisk). As CB-PTSD symptoms is a Tweedie-distributed zero-inflated variable, squared Robust Effect Size Indices, readable on a Cohen's f^2 scale (small effect: 0.02, medium effect: 0.15, strong effect: 0.35), were computed (Vandekar et al., 2020).

birth medical severity but only partially reflects the traumatic stressor intensity. It would have been preferable to use a validated scale but, to our knowledge, there is no brief scale available in obstetrics. Thirdly, it was impossible to exclude women who took sleeping pills after the delivery, as this information was not available. Given that only 11 women took sleeping pills during the last weeks of pregnancy, it seems unlikely, however, that this affected many participants. Fourthly, SBE was measured retrospectively, at eight weeks postpartum, although two out of the three SBE items were also completed at 48 hours postpartum and showed strong correlations with the 8-week ratings (see Supplementary material, section I.6.). Fifthly, a measure of exposure to trauma would have been helpful, given that it may be both associated with sleep disturbances (Brock et al., 2019) and an important risk factor for CB-PTSD (Andersen et al., 2012). Sixthly, the measurement of (CB-)PTSD symptoms could have been improved by 1) using the Impact of Event Scale-Revised (IES-R) (Weiss and Marmar, 1997), given that it contains items on hyperarousal symptoms, including on trauma-related sleep disturbances - however, one study found that the IES-R was not superior to the original IES to measure CB-PTSD(Olde et al., 2006); and 2) using the same instrument to measure prenatal and postnatal symptoms. Finally, the sample may lack representativeness. Indeed, in this cohort study, women having depressive symptoms at 17 weeks of pregnancy were more likely to drop out (Garthus-Niegel et al., 2017, 2018b). Importantly, at the same time point, neither anxiety nor PTSD symptoms predicted drop-out, and the sample was well representative of the Norwegian population in terms of obstetric complications (Storksen et al., 2013).

As for the model, while it allows to identify relationships between variables, the causal directions within a given time point are not determined statistically but according to existing scientific evidence. For example, although there are good reasons to postulate that prenatal anxiety and depression predict pre-natal insomnia, as reflected in the pregnancy layer of our model, the reverse relationship may exist. This particularly concerns the link between postnatal insomnia and CB-PTSD. Although postnatal insomnia was identified as a major predictor of CB-PTSD up to two years postpartum in the same study sample (Garthus-Niegel et al., 2015), and this relationship was reported as unidirectional in other populations (Wright et al., 2011), the relationship between postnatal insomnia and CB-PTSD symptoms may be bidirectional (Kartal et al., 2021; Richards et al., 2020).

Future studies should more systematically include post-traumatic insomnia symptoms as a covariate in the analyses: as our results illustrate, it seems to play a central role in the pre-traumatic sleep – PTSD relationship. One might wonder whether this direct relationship, which has been observed in other studies not controlling for post-traumatic insomnia (Gehrman et al., 2013; Neylan et al., 2021; Wang et al., 2019) but was not found in our sample, would persist when taking post-traumatic insomnia into account. Answering this question would help to determine whether the differences in results are due to the specificities of the perinatal context or the oversight of post-traumatic insomnia. If the former was true, this would reinforce the importance of studying more diverse samples. To better disentangle the effects of post-traumatic insomnia on the development of PTSD, it would also be necessary to measure insomnia symptoms before PTSD, rather than

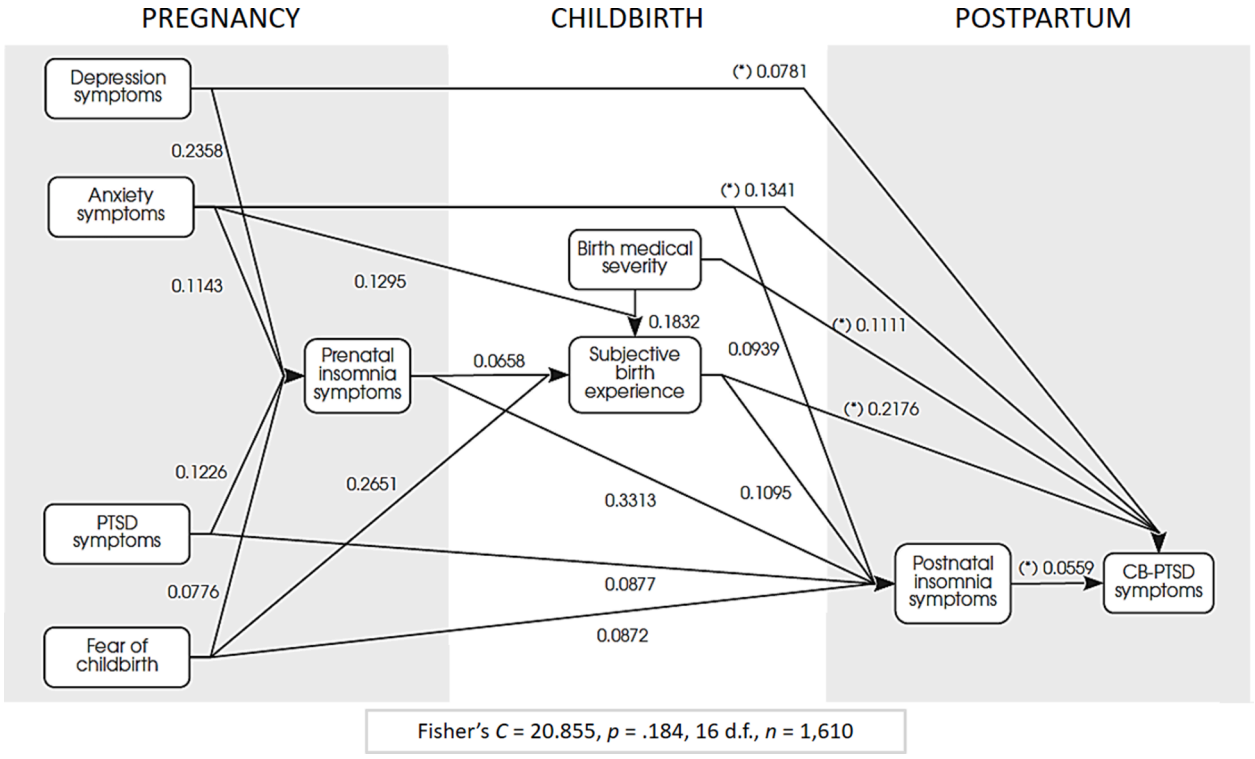

Fig. 2. Final model describing prospective associations between prenatal insomnia symptoms and CB-PTSD (derived from piecewise SEM analyses).
Note. CB-PTSD = Childbirth-related PTSD. Subjective birth experience is negatively orientated (higher scores reflect negative birth experience). Only the statistically significant pathways are shown. For all predictors of CB-PTSD symptoms, for which a log link function was used in the Tweedie model, only the unstandardised coefficients (marked with an asterisk (*)) are reported.

concurrently. Finally, complementing subjective measures of insomnia symptoms with objective measurements, such as polysomnography and actigraphy (e.g., smart watches), would help to consolidate the present findings.

5. Conclusion

Prenatal insomnia symptoms predicted CB-PTSD through SBE and postnatal insomnia symptoms, thus representing a vulnerability factor that may disrupt both maternal response during a traumatic childbirth and postpartum recovery. As a result, they may be a relevant target for CB-PTSD primary prevention, although it should be noted that effect sizes in our model were all small or very small. Finally, given that postnatal insomnia symptoms mediated the prospective relationship between prenatal insomnia and CB-PTSD symptoms, they should be more systematically included in future studies.

Contributors

C. Deforges, S. Garthus-Niegel and A. Horsch conceptualised the study. Y. Noël performed all the analyses, in collaboration with C. Deforges. C. Deforges drafted the initial manuscript, with the contribution of Y. Noël for the statistics. M. Eberhard-Gran designed and coordinated the original cohort study. All authors approved the final manuscript as submitted, and agreed to be accountable for all aspects of the work. A. Horsch is the PhD supervisor of C. Deforges.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Supplementary materials

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