Trauma & the reproductive lifecycle in women

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Abstract
Women are at significantly higher risk for developing post-traumatic stress disorder (PTSD) than men, resulting in increased psychosocial burden and healthcare related costs. Recent research has shown complex interactions between the impact of traumatic experiences, and the reproductive lifecycle in women. For example, women suffering from premenstrual dysphoric disorder (PMDD) who also report a history of sexual or physical abuse are more likely to present with different neuroendocrine reactivity to stressors, when compared to premenstrual dysphoric disorder subjects without prior history of trauma or abuse or non-premenstrual dysphoric disorder subjects. In addition, women with a history of abuse or trauma may experience re-emergence of symptoms during pregnancy. Lastly, females who experience miscarriage may present with even higher prevalence rates of post-traumatic stress disorder symptoms.

In this manuscript we examine the existing data on gender differences in post-traumatic stress disorder, with particular focus on psychological and physiological factors that might be relevant to the development of symptoms after exposure to traumatic events associated with the reproductive life cycle. Current options available for the treatment of such symptoms, including group and counselling therapies and debriefing are critically reviewed.

Keywords: Stress disorders, post-traumatic/psychology; Premenstrual syndrome; Pregnancy; Life change events; Women/psychology; Gender identity; Abortion, spontaneous

Resumo
Mulheres estão sujeitas a um maior risco para o desenvolvimento de transtorno de estresse pós-traumático (TEPT) do que os homens, o que acarreta prejuízos e custos significativos do ponto de vista psicossocial e de saúde pública. Estudos recentes mostram interações complexas entre o impacto de experiências traumáticas e o ciclo reprodutivo feminino. Por exemplo, mulheres com transtorno disfórico pré-menstrual (TDPM), que também relatam histórico de trauma ou abuso físico, estão mais sujeitas a apresentar uma resposta neuroendócrina diferenciada após exposição a um fator ou evento estressante, quando comparadas a mulheres com TDPM e sem história de abuso ou mulheres sem TDPM. Além disso, mulheres com histórico de trauma ou abuso podem apresentar recidiva de sintomas durante a gravidez. Por fim, aquelas que sofrerem abortamento estão mais sujeitas ao desenvolvimento de sintomas do transtorno de estresse pós-traumático. Neste artigo, examinamos os dados existentes sobre diferenças de gênero e transtorno de estresse pós-traumático, com enfoque especial nos fatores psicológicos e fisiológicos mais relevantes para o surgimento de sintomas após exposição a eventos traumáticos relacionados ao ciclo reprodutivo feminino. Opções terapêuticas existentes são criticamente revistas, incluindo terapias de aconselhamento e a técnica de debriefing psicológico.

Descritores: Transtornos de estresse pós-traumáticos/psicologia; Síndrome pré-menstrual/psicologia; Gravidez; Acontecimentos que mudam a vida; Mulheres/psicologia; Identidade de gênero; Aborto espontâneo

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Introduction

The past 10 years has witnessed a heightened interest in research on sex differences in posttraumatic stress disorder (PTSD), including differences in hypothalamic-pituitary-adrenal (HPA) axis reactivity, and more recently, the effects of the late luteal phase of the menstrual cycle. Research has shown an association between the negative impact of trauma-related experiences and the reproductive lifecycle in women. For example, there have been studies suggesting a significant co-morbidity between trauma and moderate-severe premenstrual syndrome (PMS) or premenstrual dysphoric disorder (PMDD), \(^1,^2\) or trauma-related symptoms associated with complicated birth experiences. \(^3\) Yet to-date, clinical care devoted specifically to trauma and female reproductive cyclicity has received modest attention.

Sex differences in PTSD

1. Prevalence & risk factors

PTSD is estimated to affect up to 25% of the individuals who have been exposed to traumatic events; the rate increases if the traumatic event is life threatening. \(^4\) The estimated lifetime prevalence of PTSD is 6.8%. \(^5\) Existing data suggest that women have two-fold higher lifetime prevalence than men, even when facing a number of similar stressful situations/exposures. \(^6\) Kessler et al, \(^7\) for example, reported a lifetime prevalence of PTSD in women of 10.4% compared with 5.0% in men, although others have reported rates as high as 19.4% and 7.6% for women and men respectively. \(^8\)

Risk factors for the development of PTSD include: being female; the nature and severity of trauma; the individual's initial psychological reaction to trauma (acute stress); pre-existing psychiatric disorders; a family history of psychopathology; a history of stress, abuse or trauma; insecure attachments; and subsequent exposure to reactivating environmental factors. \(^9\)

2. Symptoms & comorbidity

In a sample of adults (n = 103) diagnosed with PTSD, more than 70% of subjects reported anxiety, insomnia, distressing and recurrent dreams, flashback imagery and intrusive thoughts, irritability, poor concentration, avoidance behaviour and detachment. Low mood, mood lability, and impaired libido - symptoms that are not among the DSM-IV criteria for PTSD, also frequently occurred. \(^10\) Men are significantly more likely to suffer irritability and to use alcohol to excess, \(^11\) whereas women tend to experience more intense psychological reactivity to stimuli that symbolize the trauma, and display restricted affect and exaggerated startle response. \(^11\)

Women have a higher rate of comorbidity associated with PTSD, most often with major depressive disorder, alcohol abuse and dependence, generalized anxiety disorder, and severe PMS/PMDD. \(^1,^2\) A large-scale survey showed the occurrence of PTSD significantly increased the risk for the development of first-onset major depressive disorder, alcohol abuse and dependence in women. \(^13\) The extent to which sex and gender differences in PTSD are driven by hormonal and/or neuroendocrine characteristics has yet to be fully understood; \(^14\) however, such differences are markedly greater if exposure occurred before 15 years of age. \(^13\)

3. Course & burden of illness

PTSD appears to have a long course of illness in women. A community survey revealed the median duration of PTSD symptoms in women was approximately 48 months compared with about 12 months in men. Further, the median time to remission of PTSD symptoms was 35 months for women and 9 months for men. \(^11\) If left untreated, PTSD may become a chronic, disabling disorder, leading to functional impairment and affecting spousal, familial and social relationships. \(^15,^16\)

A recent US study has underscored the enormous costs associated with PTSD in women: healthcare costs more than double for women who endorse a high level of post traumatic stress symptoms compared with those who endorse a low level of symptoms. \(^17\) The initial US National Comorbidity Survey showed that PTSD is associated with nearly the highest rate of service use and possibly the highest per-capita cost of any mental illness. \(^7\)

PTSD & premenstrual syndromes

During the luteal phase of their menstrual cycles, women present with distinct immunological and neuroendocrine responses to stress. \(^18-19\) Studies suggest that hormone alterations at this time of the menstrual cycle might contribute to the development of PTSD symptoms; corroborating this hypothesis, PMDD and PTSD share some clinical characteristics, such as irritability, anxiety, and sleep disturbances. Levels of progesterone, allopregnanolone, and other related neurosteroids drop dramatically during the luteal phase, which may affect the modulation of anxiety-reducing receptors in the brain. \(^20\)

A history of childhood abuse or other types of trauma may be more common in women with PMS or, more specifically, with PMDD. Results of a large-scale prospective longitudinal community survey (n = 1,488) show that women with PTSD have an increased likelihood for secondary PMDD (OR: 11.7, 95% CI: 3.0-46.2). Most PMDD subjects also reported a history of abuse in childhood. \(^7\) Women with sub-threshold PMDD are four times more likely to develop threshold PMDD with the occurrence of a traumatic event than with no occurrence. \(^12\)

Recent studies have shown that subjects suffering from PMDD who also report a history of sexual or physical abuse are more likely to present with different neuroendocrine reactivity to stressors, when compared to PMDD subjects without abuse histories or non-PMDD subjects. A history of trauma may affect HPA-axis responsivity in healthy controls, including blunted cortisol as well as cardiovascular responses to a subsequent stress. \(^21\) Women with a history of PMDD and trauma primarily have altered adrenergic function, including heightened norepinephrine reactivity to speech stress in the luteal phase and greater $\beta_1$ and $\beta_2$ adreno-receptor responsivity, and lower-than-expected plasma norepinephrine throughout the menstrual cycle compared to non-abused PMDD subjects. \(^22\) Additional research also shows increased conversion of T4 to T3 and increased binding of thyroid hormones in women with PMDD and a history of sexual abuse. \(^23\)

PTSD & childbearing

Despite advances in obstetric care, labour and delivery remain painful, frightening, and potentially dangerous experiences for some women. The impact of traumatic or distressing pregnancy or birth experiences for women has only recently been recognized. As a result, there is little knowledge about any aspect of the diagnosis, impact, or treatment of posttraumatic stress disorder following childbirth.

The risk of PTSD is increased in women with unplanned pregnancy, high general anxiety levels prior to labour and/or depressive symptoms during pregnancy, previous pregnancy loss, and a history of mental illness or physical/sexual abuse. \(^24-26\)
With an increasing number of refugee and immigrant women coming to our perinatal clinic, anecdotally, we have noticed that trauma-like symptoms are much more frequent in recent immigrants, especially from developing countries. A recent Canadian report noted that recent immigrant status is a significant predictor of depressive symptoms after delivery.\(^{27}\) Stressful life events (e.g., rape), a lack of social support, and marital strain are highly related to depression and anxiety symptoms in immigrant women.\(^{28}\) Language barriers and health literacy issues may prevent immigrant women from communicating their physical and mental health problems, compounding maternal stress.\(^{29}\)

1. Pregnancy

Being pregnant is a momentous time in a woman’s life, but for some women, the peripartum can be laden with emotional problems, in particular anxiety. The impact on a developing fetus may be dire. High prenatal anxiety during gestation has been linked to higher levels of maternal plasma corticotropin-releasing hormone and pre-term delivery.\(^{30}\) A recent review of 14 independent prospective studies has shown an association between antenatal maternal anxiety/stress and cognitive, behavioural, and emotional problems in the child.\(^{31}\)

Soderquist J et al recently found that in a sample of 1,224 expectant women, 2.3% met DSM-IV criteria for posttraumatic stress disorder (PTSD) and 5.8% fulfilled partial criteria in late pregnancy. Traumatic stress and fear of childbirth correlated significantly.\(^{32}\) The rates may increase in groups of women who are facing hardship and have experienced multiple traumatic events. Loveland Cook et al reported PTSD prevalence of 7.7% in a sample of 744 expectant women enrolled in a supplemental nutrition program in the US mid-west, with a high rate of comorbid major depression and generalized anxiety disorder.\(^{33}\)

Pregnant women with a history of abuse-related trauma frequently report symptoms including intrusive re-experiencing thoughts/somatic symptoms, avoidance and numbing, somatization, dissociation, and interpersonal sensitivity. Other behavioural changes such as substance abuse, eating disturbances, high-risk sexual behaviour, suicidal behavior, and re-victimization have also been reported.\(^{24,33,34}\)

Studies reveal that 10% to 25% of all clinically recognized pregnancies will end in miscarriage (i.e., loss of pregnancy during the first 20 weeks of gestation; also called spontaneous abortion), and about 0.5%-1% of babies are stillborn. Existing literature has shown that about 25% of women who experience pregnancy loss within 20 weeks gestation may present with high prevalence rates of PTSD, with symptoms as severe as those following other traumatic events. About one-third of these cases will also meet criteria for major depression. Recovery is also not immediate; at 4 months post-loss, 7% will still meet criteria for PTSD.\(^{35-38}\)

Further, there is evidence suggesting an increased risk for other types of anxiety disorders in the 6 month-period following a loss.\(^{37}\) A recent report suggests, however, that increased social support may cushion the negative impact of perinatal loss.\(^{38}\)

In a 3-site survey of women who terminated unplanned pregnancies in the first trimester (n = 442), 1% met DSM-III-R criteria for PTSD and 20% of the sample had experienced an episode of depression in the 2 years following termination.\(^{39}\)

2. Postpartum

The development of acute trauma symptoms postpartum is frequently related to a previous history of 2 or more traumatic life events, obstetric complications during birth, premature birth, or stillbirth. Reports from public hospitals indicate that 1 in 3 women report the presence of at least 3 acute trauma symptoms after giving birth, with 2% to 6% of these women meeting DSM-IV criteria for PTSD.\(^{3,37,40-42}\)

A small cross-national survey found that, in a group of women who described traumatic birth experiences, symptoms most often included nightmares and flashbacks of the birth, numbness, confusion, anxiety, anger and depression, and a strong sense of isolation from the world of motherhood.\(^{15}\) Symptoms can onset within 48 hours of delivery and persist up to a year.\(^{43}\)

Notwithstanding, PTSD may also occur following deliveries that were otherwise obstetrically uneventful and deemed as “routine”. The experience of extreme pain, fear of the mother for her life or that of her infant, perception of inadequate intra-partum care during labour, or negative perception of the childbirth experience (e.g., feeling violated, powerless, helpless, a loss of control) are associated with birth trauma.\(^{44}\)

Anecdotal evidence reveals that, during a subsequent pregnancy, postnatal posttraumatic stress symptoms can recur “in full force”.\(^{45}\) Hofberg and Ward describe secondary tokophobia, a pathological dread and avoidance of pregnancy/childbirth as a consequence of a previous, traumatic delivery.\(^{46}\) The sequelae can be profound: women may terminate subsequent, desired pregnancies because they are unable to deal with the idea of another delivery.\(^{47}\)

Trauma & PTSD symptoms in women: the neuroendocrine connection

The accumulating evidence that not everyone copes with traumatic events in the same way has raised questions on what factors could contribute to or better account for such differences. Among these putative factors, the hypothalamic-pituitary-adrenal (HPA) axis and its regulation/dysregulation have been pointed out as a biologic mechanism of vulnerability for the development of a psychiatric problem after a trauma exposure. Yet, only a small percentage of subjects show consistent HPA axis dysregulation due to exposure to trauma, despite evidence of subsequent development of PTSD symptoms.\(^{11}\)

The HPA axis exerts a key role in modulating our response to stressful events. Its activation facilitates a more proactive environment for a fight-or-flight response by mobilizing neuroendocrine, anti-inflammatory and autoimmune systems.\(^{48}\) The exposure to a stressful situation/event leads to the release of corticotropin releasing hormone (CRH) from the hypothalamus, which stimulates the release of adrenocorticotropic releasing hormone (ACTH) from the pituitary gland. ACTH then promotes the production of cortisol, by the adrenal cortex. Cortisol is subsequently secreted into the blood circulation, and will promote the adaptive mechanisms required in a situation of increased stress. It is expected, however, that increased cortisol levels will then induce a negative feedback (inhibition) of the HPA axis via hypothalamus, the pituitary, and the hippocampus, therefore attenuating the stress response. This equilibrium mediated by the negative feedback of the HPA axis avoids the potential negative impact that a prolonged response to stress would exert on the structure and function of different brain regions (e.g., inducing atrophy in cells in the hippocampus, affecting memory and learning).\(^{49-50}\)
An association between PTSD and an enhanced negative feedback regulation of the HPA axis has been demonstrated in several studies. Individuals with PTSD appear to show lower basal cortisol levels and greater suppression of cortisol release following the administration of low doses of dexamethasone, compared to those without PTSD. However, most studies have focused primarily on chronic, highly symptomatic, male patients. Thus, it is still unclear the extent to which the relationship between basal cortisol levels and PTSD symptoms can be modulated by other factors including gender, type of trauma, severity of symptoms, or degree of social functioning.

Studies that included females confirmed that women with PTSD also present with lower cortisol levels compared to healthy controls, and show greater suppression of cortisol release following the administration of dexamethasone. Therefore, the basic neuroendocrine female response to stress appears to be similar to that observed in males. However, the impact of different phases of the menstrual cycle was not taken into account in the majority of these studies. In addition, other factors that may affect cortisol levels have not been adequately controlled for in most of these studies, such as alcohol intake, smoking, and exercise.

Some investigators have postulated that gender differences in HPA axis responses to stress could be partially attributed to differences in ‘threat appraisal’ between men and women. In a laboratory experiment when exposure to stress was modulated by the presence of others (strangers or partners) providing social support, women rated both stranger and partner support attempts more favourably than did men. Men in the partner support condition showed significant attenuation of cortisol responses compared with unsupported or stranger-supported men, whereas women showed no response attenuation under stranger support, and a tendency toward increased cortisol responses when supported by their partners.

The role of sex hormones in the phenomenology of PTSD among females has yet to be fully understood. Several studies suggest the occurrence of increased cortisol reactivity in pre-menopausal women with PTSD. A recent study of 13 PTSD subjects compared to 13 healthy non-traumatized subjects and seven healthy traumatized subjects revealed that plasma levels of dehydroepiandrosterone (DHEA) were increased in the PTSD subjects compared to seven healthy traumatized subjects. DHEA and cortisol responses were higher in the women with PTSD after adrenal activation by ACTH, while progesterone levels and responses were not different among the sub-groups. Interestingly, the peak change in DHEA after ACTH administration, and the DHEA/cortisol ratio were inversely correlated with the severity of PTSD and mood scores observed, respectively. These findings suggest that DHEA release may play an important role in influencing the HPA axis response to stress and modulating the negative impact of stressors on mood and behaviour in pre-menopausal women.

Progesterone is known to have antiglucocorticoid properties and thus interfere with the HPA axis reactivity to stress. In fact, some studies have demonstrated a higher neuroendocrine response to stress (i.e., higher cortisol levels after ACTH administration) in women during the luteal phase of the menstrual cycle, indicating that the negative feedback of the HPA axis may be somewhat affected.

Further research is needed to understand the impact that changes in sex hormone levels may cause on subjects’ behavioural and neuroendocrine ability to respond to stress. It is plausible that abrupt changes in hormone levels (such as that observed in the immediate postpartum period) would alter not only the HPA axis response to a stressful event, but also the negative feedback necessary to avoid potential damages induced by prolonged exposure to ‘stress hormones’.

Current knowledge on treatment options for reproductive-related trauma

1. Counseling therapies

The first line initial treatment strategy for Acute or Chronic PTSD in women is individual psychotherapy with or without adjunct medication, weekly sessions 60 minutes in length. The integration of a traumatic birthing experience at a cognitive and emotional level within a relatively short time frame, however, may be difficult for some women. Additional counseling sessions may be necessary. Many current counseling interventions employ a combination of techniques to abate both fear- (i.e., sense of security) and shame- (i.e., sense of self) based reactions.

Two studies have investigated the effects of counseling on the psychological morbidity of women who experienced a traumatic birth. Gamble et al randomized 50 women to a midwife-led, brief counseling intervention in which the women received face-to-face counseling within 72 hours of delivery and a telephone session 4 to 6 weeks postpartum. Counseling sessions included discussions of the pregnancy and delivery, enhancing social support reinforcing positive approaches to coping, and exploring possible solutions if further therapy is required. The 53 women in the control group received standard postnatal care. Post-traumatic stress symptoms, depression, and anxiety were measured during a third trimester telephone interview for women in the intervention group, and at 3 months postpartum. Women receiving counseling reported a significant decrease in depression, as well as a non-significant decrease in anxiety and self-blame. Only 3 women in the intervention group compared to 9 in the control group met diagnostic criteria for PTSD, although the result was statistically non-significant.

In a second randomized controlled study, Ryding et al compared the efficacy of 3-4 semi-structured, interview-style counseling sessions (n = 50) with standard postnatal care (n = 49) in women who had an emergency cesarean section. The first 2 counseling sessions were scheduled prior to discharge, while the following session(s) took place about 2 weeks postpartum. All participating women completed the W-DEQ, IES, and a version of the Symptoms Check List a few days after delivery, 1 and 6 months postpartum. There was a significant improvement on the 1 and 6 month follow-up questionnaires for the women receiving counseling. However, it should be noted that the counseling was insufficient for the 2 women with the worst cognitive appraisal of delivery and who experienced the most mental distress with post-traumatic stress reactions.

Even though fear of childbirth during pregnancy is fairly common, women with intense fear of pregnancy may experience mental suffering and be prone to having a complicated delivery with concomitant post-traumatic stress reactions (PTSR). Ryding et al recently evaluated the efficacy of midwife-led counseling to decrease post-parturition PTSDs. Fifty-three women identified as vulnerable to PTSR received between 1 and 14 individual sessions of counseling postpartum; they were compared to controls matched for parity and delivery method (i.e. vaginal, emergency c-section or elective c-section). Subjects completed the Wijma Delivery Experience Questionnaire and the Impact of Event Scale. Ten
women in the counseling group and 1 woman in the control group scored > 30 on the Impact of Events Scale, indicative of PTSR. While most of the women reported being satisfied with their care, women in the counseling group as a whole reported a less positive birthing experience than the control group. Yet, without comparison to a group of women deemed vulnerable to PTSR - who did not receive the counselling intervention, it is difficult to ascertain if the intervention had in fact improved their situation.

2. Group therapies

The literature on group therapy for the treatment of reproductive-related trauma is currently rather sparse, although some encouraging notes may be gleaned from the published work.

In a randomized controlled trial Ryding et al investigated the efficacy of group counseling in a sample of women who had undergone an emergency cesarean section.

The counseling intervention group (n = 64) consisted of two group sessions, 2-3 weeks apart, commencing at about 2 months postpartum. Sessions began with women sharing their pregnancy, delivery, and hospital care stories and then proceeded according to the needs of the group. Three questionnaires, measuring delivery expectations and experience, frequency of symptoms related to the traumatic event (i.e. C-section), and depression, were completed 6 months postpartum by all participants. Women in the control group (n = 73) were offered individual consultations to discuss their deliveries after the 6-month questionnaires were completed. While the authors found little difference between the counseling and control group with respect to level of fear following childbirth, symptoms of posttraumatic stress or postnatal depression, subjects reported satisfaction with the group sessions. One participant indicated that she would have liked 1 or 2 additional sessions.

A second study evaluated the efficacy of cognitive group therapy for women who experienced traumatizing provider interactions during their childbearing experience.

Nine women with 21 pregnancies and 19 births underwent cognitive group therapy sessions of 4 hours each, monthly for five months after giving birth. These sessions followed the "Insight: A cognitive enhancement program for women" curriculum in which objectives and activities, including topics of loss/grief, self-esteem and communication, were the focus of assignments and group discussion. Quality of provider interaction, birth perception, posttraumatic childbirth stress, self-esteem, depression, anxiety and loneliness scales were assessed pre and post-intervention. Pre-intervention scores identified perinatal trauma in the women. Post-intervention scores revealed a significant improvement on all the measurements. Notwithstanding, the results should be interpreted with caution since a control group was lacking to account for spontaneous remission of symptoms.

3. Debriefing

Debriefing is a treatment strategy originally used to treat acute combat stress reactions in returning soldiers. The positive results from this intervention prompted similar strategies following a range of adversities in which a traumatic event or 'critical incident' caused unusually intense stress reactions. Critical incident stress debriefing (CISD) is a process that attempts to prevent or limit the development of PTSD by allowing the affected individual to discuss their thoughts and feelings about the event in a controlled and safe environment. The efficacy of this treatment strategy is currently under debate, and current opinion holds there is limited evidence that debriefing prevents PTSD. In fact, there is indication that some vulnerable individuals may experience an increase in symptoms as a result of psychological debriefing.

Two studies have investigated the effectiveness of debriefing after childbirth. Priest et al investigated whether CISD reduced the incidence of postnatal psychological disorders of 1745 women in a single-blind randomized controlled trial. Between 24 and 72 hours after delivery, 875 and 870 women were allocated to the CISD and standard postnatal care respectively; a single, standardized debriefing session was initiated immediately or within the next day. The results showed no difference between groups on the Edinburgh Postnatal Depression or IES at 2, 6, or 12 months postpartum. About 66% of subjects reported that the debriefing session was moderately or greatly helpful.

In the second randomized controlled trial, Small et al investigated the effectiveness of a midwife-led debriefing session after giving birth by caesarean section, forceps, or vacuum extraction. Following delivery, women were randomized to receive a debriefing session (n = 520) to discuss their labour, birth and post-delivery events/experiences prior to discharge or standard care (n = 521), which consisted of a brief visit by the midwife to give the mother a pamphlet on sources of assistance. Maternal depression and overall health status (bodily pain, mental and general health, vitality, physical and social functioning, as well as both physical and emotional role functioning) were measured with the Edinburgh Postnatal Depression Scale and the 36-item short-form health survey (SF-36), respectively, at 6 months postpartum. Of the debriefing and standard care groups respectively, 467 and 450 women completed the follow-up questionnaires. Women allocated to the debriefing group were more likely to score as depressed and had poorer health status on 7 of the 8 SF-36 subscales, although the results were only significant for the emotional role functioning subscale. Although 94% of the women perceived the debriefing sessions as "helpful", the intervention in this study was ineffective in reducing maternal morbidity and possibly contributed to emotional health problems.

Two additional randomized trials have studied the effects of debriefing on postpartum psychological morbidity and emotional adaptation, respectively. Lavender & Walkinshaw found that women who received an unstructured debriefing intervention had decreased anxiety and depression scores at 3 weeks postpartum compared to women who had no intervention. However, participants comprised of healthy women who had an uneventful delivery. Lee et al employed psychological debriefing at 2 weeks postpartum in a group of women who miscarried. At the four-month follow-up, there were no significant differences between women who received the intervention and those who received standard postnatal care with respect to emotional adaptation.

Based on the assumption that individuals wish to talk about a traumatic event, early intervention therapies, like debriefing, have been implemented as a requirement for post-incident coping and adjustment, especially in fields with a high degree of trauma exposure (emergency care, military personnel, etc.). Yet, this assumption is being questioned. In a survey of 217 emergency services officers, participants rated the helpfulness of talking about a traumatic event at various time periods. The results showed that 51% of subjects reported talking...
immediately after the event was the most helpful. Discussing the event within 24 hours of the incident was considered helpful by 41.5%; this decreased to 37.8% after 24 hours had elapsed. Although these numbers suggest that the optimum time to discuss an event is earlier rather than later post-incident, they should be interpreted with caution since the nature of the traumatic event and the structure of the discussions were not controlled. In the context of traumatic births, debriefing too soon may interfere with the natural mechanisms that process the trauma and allow the individual to accept the event in a non-traumatic way.28

Quite often, psychological perinatal trauma includes both shame and fear-based reactions.29 Thus, unimodal therapy strategies are likely inadequate for individuals who are trying to cope with both fear and shame experiences. This may account for the ambiguous results purported by studies investigating debriefing as an early intervention after traumatic births.

Conclusions
Existing data suggest a higher vulnerability to develop PTSD symptoms associated with traumatic experiences in the context of reproductive life cycle events (e.g., miscarriage, PMDD). However, much has yet to be learned on how different biological and psychological factors may play a role in modulating this increased risk, and about effective pharmacological and non-pharmacological treatment options.

References


