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Sensory Modulation Difficulties and PTSD: A Prospective Study During and After Rocket Attacks

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Abstract

Objective: Civilians in war may suffer from distress not only during the peritraumatic phase, manifested in early trauma-related symptoms, but also after the trauma ends, as reflected in Posttraumatic Stress Disorder (PTSD). Evidence has suggested that early trauma-related symptoms underlie the development of PTSD. Additionally, research has revealed relations between sensory responsiveness and both early trauma-related symptoms and PTSD, thus implying that individuals with sensory modulation difficulties may exhibit elevated symptomatology at the peritraumatic phase, and may be at risk for PTSD. Nevertheless, the cross-sectional design of former studies allowed neither identifying the directionality of the relationship between sensory modulation and PTSD nor exploring the role of early trauma-related symptoms within this relationship. **Methods**: The current prospective study, which was aimed at bridging these knowledge gaps, was conducted among a convenience sample of Israeli adults (n=209) during rocket attacks (T1) and 40 to 71 days after ceasefire (T2). Background variables, sensory modulation difficulties, early trauma-related symptoms, and PTSD symptoms were assessed online via self-report measures. **Results**: Results revealed that high sensory responsiveness was related to early trauma-related symptoms, and predicted PTSD symptoms of hyperarousal, intrusion, and negative alterations in mood and cognitions. Moreover, early trauma-related symptomatology mediated the relations between high sensory responsiveness and PTSD symptoms of intrusion and negative alterations in mood and cognitions. **Conclusion**: The current findings suggest that high sensory responsiveness is a risk factor for PTSD, and that early trauma-related symptomatology may serve as a mechanism underlying the relationship between high sensory responsiveness and PTSD.

Keywords: early trauma-related symptoms; PTSD; sensory modulation; war; rocket attacks

Clinical Impact Statement: The present findings indicated that high sensory responsiveness predicted PTSD symptoms, and early trauma-related symptomatology mediated the relations between high sensory responsiveness and PTSD symptoms. Thus, assessment of sensory modulation may be beneficial for identifying individuals who may be particularly vulnerable to PTSD, and implementing sensory modulation strategies during the peritraumatic phase may decrease the risk of future PTSD among this vulnerable population.

Introduction

Exposure of citizens to mass trauma (Hoffman & Kruczek, 2011) such as war may lead to a plethora of detriments (American Psychiatric Association, 2013). War has been found to be related to an elevated prevalence of psychopathology among civilians, manifested, among other ways, in anxiety, depression, and Posttraumatic Stress Disorder (PTSD; Morina et al., 2018; Murthy & Lakshminarayana, 2006). During and shortly after exposure to war, namely, during the peritraumatic phase, some individuals may exhibit peritraumatic reactions, such as dissociation, fear of dying, and tachycardia (Agorastos et al., 2013). Furthermore, ongoing exposure to a series of traumatic events, such as happens during war, may lead to the development of initial symptomatology that is often ascribed to the posttraumatic phase. Former studies among civilians have documented these early trauma-related symptoms, which consist of re-experiencing of the trauma, avoidance of stimuli associated with the trauma, hyperarousal, and negative changes in mood and cognitions (Gelkopf et al., 2017, 2019).

Although many individuals exhibit resilience and do recover after the traumatic event is over (Bonanno, 2004; Murthy & Lakshminarayana, 2006), others may suffer from symptomatology at the posttraumatic phase, manifested in Posttraumatic Stress Disorder (PTSD). According to the DSM-5 (APA, 2013), PTSD comprises four clusters that last for over a month and impair functioning: intrusion (e.g., intrusive memories, nightmares, flashbacks), avoidance (e.g., efforts to avoid trauma-related thoughts, feelings, or external reminders), negative changes in mood and cognitions (e.g., inability to recall a crucial detail of the trauma, negative thoughts about oneself and others), and arousal symptoms (e.g., hypervigilance, irritability, or aggression). Evidence has revealed several risk factors for PTSD, among them early symptomatology, gender, and age, so that individuals who suffer from elevated early trauma-related symptoms (Gelkopf et al., 2019; Lapid Pickman et al., 2017; Neria et al., 2010), women (Christiansen & Berke, 2020; Kolltveit et al., 2012; Luxton et al., 2010), and younger individuals have been found to be at risk for PTSD symptomatology (e.g., Reynolds et al., 2016; Sommer et al., 2021).

Another potential risk factor for PTSD is the individuals' perception of the internal (bodily) and external (environmental) sensations (Harricharan et al., 2021). Sufficient sensory modulation--the ability to produce an adjusted response to sensory stimuli while filtering out irrelevant stimuli-- enables an efficient and adaptive performance (Miller et al., 2007). Further, modulating the intensity and type of responding to sensory cues across sensory modalities helps maintain an appropriate level of arousal (Harricharan et al., 2021; Porges, 2009), which is necessary for adaptive emotional and social behavior (Sievers et al., 2019). Research has documented two main patterns of sensory difficulties. The first reflects high sensory responsiveness, manifested in responding to daily non-painful sensations with higher intensity and for longer duration, while reporting these to be aversive and even painful (Bar-Shalita et al., 2019; Miller et al., 2007). High sensory responsiveness co-occurs with anxiety from early childhood (Carpenter et al., 2019) and is linked to neuroticism (Bar-Shalita & Cermak, 2020). The second pattern consists of lower sensory responsiveness, requiring robust sensory cues to provoke responses and is therefore characterized by tendencies that include failing to detect sensory signals, appearing apathetic, having low inner drive, and being withdrawn and inattentive (Miller et al., 2007).

Sensory modulation difficulties, which have been found to be related to psychological distress in the general population (Bar-Shalita & Cermak, 2016), appear to increase one's vulnerability in the face of trauma (Charny et al., 2023). A traumatic event, by definition, involves aversive stimuli, while specific reactions to trauma-related stimuli are inherent to PTSD (e.g., re-experience or avoidance of trauma-related stimuli; American Psychiatric Association, 2013). Therefore, individuals with sensory modulation difficulties may be at risk for trauma-related symptomatology (Yochman & Pat-Horenczyk, 2020).

Research has supported this line of thought, suggesting that sensory modulation difficulties may be a risk factor for symptomatology at both the peritraumatic and the posttraumatic phases. Previous studies have indicated relations between sensory modulation difficulties and PTSD symptomatology (Engel-Yeger et al., 2013; R. Joseph et al., 2022; R. Y. Joseph et al., 2021; Kimball, 2022; Yochman & Pat-Horenczyk, 2020). For example, a study among 30 individuals diagnosed with PTSD and 30 healthy controls revealed that those with PTSD had a higher frequency of both high and low sensory responsiveness (Engel-Yeger et al., 2013). Additionally, a study among children exposed to rocket fire during war found that these children exhibited behaviors typical of both high sensory responsiveness and low sensory responsiveness (Yochman & Pat-Horenczyk, 2020).

Research on the relations between sensory modulation patterns and early symptoms arising at the peritraumatic phase, on the other hand, has been highly limited. In fact, to the best of our knowledge, only one study, conducted recently by this research group (Charny et al., 2023) explored this subject matter. Nonetheless, the results of that study revealed the same trend, so that high sensory responsiveness was related to elevated early trauma-related symptoms and that the risk for elevated symptomatology during the peritraumatic phase was doubled for each increase in the high sensory-responsiveness score (Charny et al., 2023).

Despite this evidence, several key questions remain. First the directionality of the relationship between sensory modulation and PTSD symptoms is unclear. Given that studies thus far have been cross-sectional (Engel-Yeger et al., 2013; Yochman & Pat-Horenczyk, 2020), their results cannot indicate whether sensory modulation patterns predict PTSD or the other way around, so that PTSD hampers the ability to efficiently modulate sensory input. Second, the role of initial symptomatology at the peritraumatic phase within the relations between sensory modulation and subsequent PTSD in unknown. Specifically, it is unclear whether individuals with sensory modulation difficulties exhibit vulnerability at the peritraumatic phase, manifested in elevated early trauma-related symptoms, which, in turn, predict their subsequent PTSD. Therefore, a comprehensive understanding of the linkages between sensory modulation and PTSD symptoms necessitates further longitudinal research that not only has the potential to enhance the identification of individuals susceptible to PTSD, but also to deepen our insights into the mechanisms underlying this vulnerability.

The current prospective study bridged these gaps. This study assessed Israeli civilians' trauma-related symptoms in the face of rocket (i.e., unguided military missile) attacks as part of Operation Guardian of the Walls. The operation lasted 12 days (May 10, 2021 through May 21, 2021), during which period 4,360 rockets were fired at Israel. The study relied on data collected during rocket attacks (the peritraumatic phase, T1) and 40 to 71 days after ceasefire (the posttraumatic phase, T2). Two main objectives were set: (1) to explore the relationship among sensory modulation difficulties at T1, early trauma-related symptoms at T1, and PTSD symptoms at T2, and (2) to explore the mediating role of early trauma-related symptoms at T1 within the relationship between sensory modulation difficulties at T1 and PTSD symptoms at T2.

Methods

Participants and procedure. The current research is part of a larger prospective study that focuses on the implications of rocket attacks on civilians' well-being (e.g., Charny et al., 2023; Dokkedahl et al., 2024; Dokkedahl & Lahav 2023a, 2023b). This study employed a convenience sample of Israeli adults ≥ 18 years old and living in Israel, using an online survey. Data was collected at two time points: 1) The peritraumatic phase (T1)-- May 14, 2021 to May 21, 2021 (fifth day of the operation to several hours after the ceasefire); 2) The posttraumatic phase (T2)-- June 30, 2021 to July 31, 2021 (40 to 71 days after the ceasefire).

The Tel Aviv University institutional review board (IRB) approved all procedures and instruments. The survey was administered utilizing Qualtrics software (QualtricsLabs, inc., Provo, UT, US). Clicking on the link to the survey guided potential respondents to a page that provided information about the purpose of the study, the nature of the questions, and a consent form (i.e., participants were informed that the survey was voluntary; respondents could skip any questions or quit at any time). Participants were also provided with contact information for the research team and for several Israeli organizations that provide support/treatment, in case they experienced discomfort/distress and need help. A built-in option of the Qualtrics platform ('Prevent Ballot Box Stuffing') was used in order to avoid duplicative entries to the survey. In addition, to detect potential fraudulent entries from bots, records attached to IP addresses that were duplicated in the dataset were discarded.

At T1, a Facebook advertisement was used to recruit participants. Facebook users were eligible for this study if they were ≥ 18 years old and living in Israel. The Facebook advertisement included a headline, main text, and link to the survey. At the end of the survey, participants were provided with the option to leave their email address in case they were willing to take part in the second measurement of the study. To protect participants' privacy, their email addresses were kept in a secured file protected by password, separately from the dataset. At T2, participants who provided their email addresses were contacted via email inviting them to participate in the second measurement. The invitation consisted of a headline, main text, and link to the survey. The questionnaire completion time ranged between 15 and 30 minutes at both T1 and T2.

At T1 a total of 794 responses were collected, of which 739 (93%) reported being exposed to rocket attacks and answered some of the survey's questionnaires, and 488 participants provided data regarding the study variables. Of these, 315 (64.5%) provided an email address in order to be invited to take part in the second assessment. At T2, all 315 were invited via email to participate in the study. Of them, 236 participants entered the survey's link and answered some of the survey's questionnaires, and 209 provided data regarding the study variables. Therefore, the sample in the current study consisted of 209 participants who participated at both T1 and T2 and provided data regarding the study variables. Participants' ages ranged from 20 to 75 (M=37.8, SD=10.4), and most participants reported being secular (68.5%), being in intimate relationships (61.5%), having an undergraduate or graduate degree (65%), and earning an average or above average income (56%).

Measures

Background variables. Participants completed a brief demographic questionnaire that assessed age, gender, education, relationship status, religiosity, and income.

Sensory responsiveness (T1). High and low sensory responsiveness were measured via the Sensory Responsiveness Questionnaire-Intensity scale (SRQ-IS; Bar-Shalita et al., 2009). This scale consists of 58 scenarios typical in daily life, each of which was phrased in a manner that attributes either a hedonic or an aversive valence (e.g., "I enjoy being in a place that is brightly lit"; "Washing my face bothers me"; "Going down an escalator bothers me";

“Smelling perfume bothers me”). Each item refers to one sensory stimulus in one of the following modalities: auditory; visual; gustatory; olfactory; vestibular; and somatosensory, excluding pain. Participants were asked to rate the intensity of their hedonic/aversive response to the scenario on a 5-point Likert scale ranging from 1 (not at all) to 5 (very much), yielding the SRQ-Aversive and SRQ-Hedonic scores indicating high and low sensory responsiveness, respectively. The SRQ-IS demonstrates high internal consistency and test-retest reliability (Bar-Shalita et al., 2009). Internal consistency reliabilities in this study for SRQ-Aversive and SRQ-Hedonic scores were good ($\alpha = 0.77$, 0.73, respectively).

Early trauma-related symptoms (T1). Trauma-related symptoms during rocket attacks were measured via a modified version of the PTSD Checklist (PCL-5; Weathers et al., 2013). In this 20-item self-report measure, participants were asked to indicate the extent to which they experienced each PTSD symptom (e.g., “Repeated, disturbing dreams of the stressful experience?”; “Loss of interest in activities that you used to enjoy?”; “Feeling jumpy or easily startled?”), on a 5-point Likert scale ranging from 0 (*not at all*) to 4 (*extremely*). Items corresponded to the newly approved PTSD symptom criteria in the Diagnostic and Statistical Manual of Mental Disorders (DSM–5; American Psychiatric Association, 2013). The original version was adapted such that the timeframe for experiencing each symptom was changed from “in the past month” to “during the current rocket attacks,” and the index event was the rocket attacks that were part of Operation Guardian of the Walls. Although not a definitive diagnostic measure, preliminary research suggests a cutoff score of 33 is a useful threshold to indicate symptomatology that may be at clinical levels (Bovin et al., 2016). The PCL-5 demonstrates high internal consistency and test-retest reliability (Bovin et al., 2016). This study had good internal consistency reliabilities for intrusion, avoidance, negative alterations in mood and cognitions, and hyperarousal clusters ($\alpha = 0.87$, 0.78, 0.84, 0.84, respectively).

Posttraumatic Stress Disorder Symptoms (T2). Symptoms of PTSD in response to the rocket attacks were measured via the PCL-5 (see the description above; Weathers et al., 2013), 40 to 71 days after ceasefire. The original version was adapted, such that the timeframe for experiencing each symptom was changed from "in the past month" to "since ceasefire," and the index event was the rocket attacks during the Operation Guardian of the Walls. This study had good internal consistency reliabilities for intrusion, avoidance, negative alterations in mood and cognitions, and hyperarousal clusters (α = 0.90, 0.88, 0.91, 0.86, respectively).

Control variables. Given that age and gender are known to be risk factors for PTSD, they were treated as control variables.

Analytic Strategy

The analysis was performed using R software (R Core Team, 2022). Pearson correlation tests (for continuous variables), independent samples t-tests, and Chi square tests with Yate's continuity correction (for the dummy variable of gender) were conducted to assess the associations between the study variables.

To test for the mediating role of early trauma-related symptoms at T1 within the association between sensory modulation at T1 and PTSD symptoms at T2, we applied a mediation analysis analytic scheme, using age and gender as control variables in the analysis. For each cluster of symptoms in T1 and T2, we screened for multivariate outliers using the Mahalanobis distance measure for our raw data, in order to detect and omit observations that might severely distort parameter estimation (Leys et al., 2018). The data was also tested for the standard OLS regression linearity and homoscedasticity assumptions (Tabachnick et al., 2007). Since the data presented some heteroskedasity and right-skewness, we applied a quasi-parametric mediation analysis method, which is more robust to such violations. The analysis was performed using the *robmed* package (Alfons, Nüfer, et al., 2022). We applied the median regression approach, as suggested by Yuan and Mackinnin (2014), which uses the Least Absolute Deviations estimation method, and better controls for type I error. Since our dependent variables are not absolutely continuous (i.e., higher-order ordinal), estimation of the conditional median could lead to convergence issues of the maximum score estimator (Machado & Santos Silva, 2005). When such issues arose in our sample, we applied a Jittering technique, which imposes an artificial density correction on the dependent variable (Geraci & Farcomeni, 2022). We measured each model's goodness of fit using Koenker and Machado's (1999) pseudo-R^2 measure, which represents the improvement of the model of interest from an intercept-only null model. We followed the suggestion of Alfons et al. (2022) and reported point estimated produced using the bootstrapped samples. The significance of the estimates of direct and total effects was calculated using the normal approximation bootstrap z-tests, using the mean and the standard deviation over the bootstrap replicates. Confidence intervals for the estimates of the indirect effects of sensory modulation on PTSD symptoms were calculated using the biased-corrected accelerated method (Davison & Hinkley, 1997)

Power Analysis

While recruitment efforts were made based on our initial pool of 315 participants who agreed to be contacted for participation at T2, we also performed a power analysis in order to validate the sufficiency of the data gathered. Since the indirect effect's significance is determined using a bootstrapping procedure, we adopted a Monte-Carlo simulation approach for the analysis, as suggested by Schoemann et al. (2017). The analysis derived a sample size of 186 observations, for a power of 0.8, in a 5000 replicates procedure, given the estimates of the correlation coefficients from our data.

Results

Early Trauma-related Symptoms and PTSD Symptoms

At T1, 39.2% (n = 82) reported at least one intrusion symptom, 26.8% (n = 56) reported at least one avoidance symptom, 52.6% (n = 110) reported at least one symptom reflecting negative alterations in mood and cognitions, and 77.0% (n = 161) reported at least one hyperarousal symptom. Furthermore, 18.7% (n = 39) of the participants had an early trauma-related symptoms total score of 33 or above, indicating that their symptoms were clinically significant.

At T2, 45.5% (n =95) of the participants reported at least one intrusion symptom, 30.1% (n = 63) reported at least one avoidance symptom, 48.8% (n = 102) reported at least one symptom reflecting negative alterations in mood and cognitions, and 69.7% (n = 146) reported at least one hyperarousal symptom. Furthermore, 18.2% (n = 38) of the participants had a PTSD symptoms total score of 33 or above, indicating that their symptoms were clinically significant. Comparing symptom levels via Wilcoxon's signed-rank test, indicated higher mean-rank score at T2 than T1 for all symptom clusters ($p_s<0.05$).

Associations Between Sensory Modulation Difficulties, Early Trauma-related Symptoms, and PTSD Symptoms

As can be seen in Table 1, high sensory responsiveness was associated with elevated levels of early trauma-related symptoms measured at T1, and PTSD symptoms measured at T2. Conversely, low sensory responsiveness had non-significant associations with both early trauma-related symptoms and with PTSD symptoms. In addition, as can be seen in Table 1, early trauma-related symptoms were associated with PTSD symptoms: the higher the levels of early trauma-related symptoms, the higher the levels of PTSD symptoms.

Sensory Modulation Difficulties and PTSD Symptoms: The Mediating Role of Early Trauma-related Symptoms

Mediation analyses indicated that low sensory responsiveness did not predict either PTSD symptoms or early trauma-related symptoms, and no mediation effects were found within these relations. Conversely, the total effect of high sensory responsiveness on PTSD symptoms of hyperarousal, intrusion, and negative alterations in mood and cognitions at T2 was significant overall ($\hat{\beta} = 2.6, pv < 0.001$; $\hat{\beta} = 0.74, pv = 0.03$; $\hat{\beta} = 1.09, pv = 0.01$, respectively), namely, higher levels of sensory over-responsiveness in T1 predicted these PTSD-clusters symptoms.

Furthermore, the results indicated that early trauma-related symptoms mediated the relationship between high sensory responsiveness and intrusion, and negative alterations in mood and cognitions clusters of PTSD symptoms (see Table 2 and Figures 1-2). Specifically, whereas the indirect effect of high sensory responsiveness was non-significant for the hyperarousal cluster, it was significant for the intrusion and negative alterations in mood and cognitions clusters ($\hat{\beta} = 0.58$, with LL>0 for confidence intervals of 97% confidence; $\hat{\beta} = 0.71$, with LL>0 for confidence intervals of 99% confidence, respectively), indicating that high sensory responsiveness at T1 was positively and significantly associated with intrusion and negative alterations in mood and cognitions clusters of early trauma-related symptoms at T1 ($\hat{\beta} = 1.61, pv = 0.01$; $\hat{\beta} = 2.36, pv = 0.004$, respectively), and that the latter positively and significantly predicted their respective clusters of PTSD in T2 ($\hat{\beta} = 0.36, pv < 0.001$; $\hat{\beta} = 0.3, pv < 0.001$, respectively).

The indirect effect of high sensory responsiveness was also significant for the avoidance cluster ($\hat{\beta} = 0.14$, with LL>0 for confidence intervals of 97% confidence; see Table 2 and Figure 3); however, it had no direct effect on these clusters at either T1 or T2. Such statistical results are somewhat debatable in the literature, and should be interpreted with caution (Baron & Kenny, 1986; Hayes & Rockwood, 2017).

Discussion

The current prospective study aimed to explore, for the first time, the contribution of sensory modulation in predicting PTSD and the mediating role of early trauma-related symptoms within these relations. Our findings indicated that high sensory responsiveness was associated with elevated levels of all PTSD symptoms, and predicted PTSD symptoms of hyperarousal, intrusion, and negative alterations in mood and cognitions one to two months after rocket attacks, above and beyond age, gender, and early trauma-related symptoms. Furthermore, our findings indicated that the relations between high sensory responsiveness and PTSD symptoms of intrusion and negative alterations in mood and cognitions, were mediated by trauma-related symptomatology during the peritraumatic phase.

The current results revealed that high sensory responsiveness was associated with early trauma-related symptoms and predicted PTSD symptoms of hyperarousal, intrusion, and negative alterations in mood and cognitions. These findings are in line with previous evidence, which pointed to high sensory responsiveness may be a risk factor for psychological distress in the face of trauma (Engel-Yeger et al., 2013; Yochman & Pat-Horenczyk, 2020). Deficient executive functions (Engel-Yeger & Rosenblum, 2021) and impeded functioning (Dunn et al., 2016; Engel-Yeger & Rosenblum, 2021), which have been documented among individuals who are characterized by high sensory responsiveness, may limit the ability to adaptively cope with traumatic events, and thus may intensify vulnerability to trauma-related symptomatology not only during trauma exposure, but, also, after the trauma ends. In addition, it might be that high sensitivity to stimuli, which characterizes high sensory responsiveness, may negatively color appraisals of the event, so that individuals with high sensory responsiveness evaluate the traumatic event as more harmful and aversive (Charny et al., 2023), and therefore suffer from substantial emotional detriments.

The current analyses further indicated that the relations between high sensory responsiveness and PTSD symptoms of intrusion and negative alterations in mood and cognitions, were mediated by their respective early trauma-related symptoms at the peritraumatic phase. These results suggest that, although most individuals exhibit recovery after the trauma ends (Bonanno, 2004), early symptomatology during trauma exposure may be responsible for the vulnerability high sensory responsiveness poses for developing subsequent PTSD.

Individuals with high sensory responsiveness appear to be particularly distressed during trauma, and to suffer from early symptoms of intrusion and negative alterations in mood and cognitions, which in turn may take their toll and put them at risk for PTSD. Re-experiencing thoughts and memories concerning the trauma may entrench the painful experience, and suffering from negative moods and pessimistic views may hinder reprocessing of the trauma. Together, these negative implications of early trauma-related symptoms may substantially affect individuals with high sensory responsiveness-- who exhibit elevated sensitivity to stimuli and difficulties in habituation to begin with (Bar-Shalita et al., 2019)-- hampering their capacity to flexibly move from emergency to routine, and to recover after the trauma ends. In this way, early trauma-related symptoms may not only pose a burden on these individuals in the short run, but may serve as mechanism underlying their later PTSD symptomatology. Indeed, in healthy individuals, sensory information is relayed via the anterior insula to the prefrontal cortex responsible for multisensory integration enabling the signature of the sensory experience that is supported by executive function processing. However, in PTSD these processes are limited (Harricharan et al., 2021).

Whereas the mediation analyses for the avoidance cluster produced debatable findings and thus were suppressed from our discussion, the analyses for the hyperarousal cluster revealed interesting findings. Our results indicated that, although high sensory responsiveness was related to elevated hyperarousal symptoms during both the peritraumatic and posttraumatic phases, and although early hyperarousal symptoms predicted PTSD hyperarousal symptoms, the relations between high sensory responsiveness and hyperarousal PTSD symptoms were not mediated by early hyperarousal symptoms. One may suggest that the present findings are rooted in the way that early hyperarousal symptoms are perceived and experienced by individuals with high sensory responsiveness. Specifically, since high sensory responsiveness is coupled with hyperarousal (Sievers et al., 2019), the latter type of symptomatology, as opposed to other early trauma-related symptoms, does not serve as a mechanism underlying PTSD symptoms. One should note, however, that the present study did not explore perceptions of early hyperarousal symptoms among individuals with high sensory responsiveness. Therefore, we have offered only speculations that warrant future research.

As opposed to high sensory responsiveness, the current findings indicated that low sensory responsiveness was unrelated to either early trauma-related symptoms or PTSD symptoms, and had a non-significant effect in explaining these symptoms. The present findings are inconsistent with former evidence that has indicated low sensory responsiveness to be associated with PTSD symptoms (Engel-Yeger et al., 2013; Yochman & Pat-Horenczyk, 2020). Nevertheless, these studies had cross-sectional designs and could not reveal the directionality of the relationship between low sensory responsiveness and PTSD. Therefore, it might be that low sensory responsiveness does not shape one's reaction to trauma, and that earlier results indicating associations between low sensory responsiveness and PTSD reflect, in fact, the effects of PTSD symptoms on the sensory domain (Warner et al., 2013), which result in hyposensitivity to stimuli.

Alternatively, it might be that the implications of low sensory responsiveness on trauma-related symptomatology become apparent only after a long time has passed since exposure to the trauma. According to this explanation, individuals with low sensory responsiveness may tend to present a delayed response (Miller et al., 2007), and may exhibit symptomatology long after trauma exposure. Therefore, the present study, which assessed symptoms during trauma exposure and 40 to 71 days after the trauma ended, could not detect these effects. To explore this supposition, there is a need for longitudinal studies that would assess the implications of low sensory responsiveness among trauma survivors over a long period of time.

The current study suffered from several limitations. First, our sample was gathered online, potentially leading to a self-selection bias. Furthermore, this study relied on convenience sampling and was conducted among Israeli participants, which could limit the generalizability of the present results to other populations. Second, this study was based on self-reported data, which may be subject to response biases. Third, although the second assessment, which took place 40 to 71 days after the trauma, was in line with the time frame for PTSD symptoms as suggested by the DSM-5 (i.e., within the first 3 months after the trauma; APA, page 276), it might have been limited in detection of later recovery or delayed expression of symptomatology. Thus the present results concerning levels of posttraumatic distress, should be interpreted with caution. Forth, given that early trauma-related symptoms and sensory modulation were assessed concurrently at T1, conclusions regarding the direction of the associations between them are precluded. Therefore, although sensory modulation difficulties are regarded as a neurodevelopmental condition that generally appears in *early* childhood, the present findings may reflect the implications of distress during trauma exposure expressed in high sensory responsiveness. Fifth, this study focused on the implications of sensory modulation on trauma-related symptoms in the face of rocket attacks. It might be that the effects of sensory modulation differ as a function of trauma type. Future prospective studies should explore the relationship between sensory modulation and trauma-related symptoms among a variety of populations with diverse cultural backgrounds and with regard to other types of traumatic events, while incorporating data from clinical interviews.

Notwithstanding the aforementioned limitations, the current study contributes to our understanding of the repercussions of sensory modulation on trauma-related symptomatology during the peritraumatic and posttraumatic phases. Our findings suggest that high sensory responsiveness may serve as a risk factor for early symptomatology, as well as for PTSD, and that individuals with high sensory responsiveness may be susceptible to trauma-related symptoms during trauma exposure, which, in turn predict their PTSD symptomatology. These results have important clinical implications. They imply that assessment of sensory modulation may be beneficial for identifying individuals with high sensory responsiveness who may be particularly vulnerable to PTSD. Moreover, implementing sensory modulation strategies aimed at the individual and environmental dimensions developed for trauma survivors (Fraser et al., 2017) during the peritraumatic phase may be valuable, and may decrease the risk of future PTSD among this vulnerable population.

References

Agorastos, A., Nash, W. P., Nunnink, S., Yurgil, K. A., Goldsmith, A., Litz, B. T., Johnson, H., Lohr, J. B., & Baker, D. G. (2013). The Peritraumatic Behavior Questionnaire: Development and initial validation of a new measure for combat-related peritraumatic reactions. *BMC Psychiatry*, *13*(1), 9. https://doi.org/10.1186/1471-244X-13-9

Alfons, A., Ateş, N. Y., & Groenen, P. J. (2022). A Robust Bootstrap Test for Mediation Analysis. *Organizational Research Methods*, *25*(3), 591–617. https://doi.org/10.1177/1094428121999096

Alfons, A., Nüfer, Y. A., & Groenen, P. J. (2022). Robust Mediation Analysis: The R Package robmed. *Journal of Statistical Software*, *103*(13), 1–45. https://doi.org/10.18637/jss.v103.i13

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Health Disorders: DSM-5. 5th ed. American Psychiatric Association, Washington, DC.*

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, *51*(6), 1173.

Bar-Shalita, T., & Cermak, S. A. (2016). Atypical sensory modulation and psychological distress in the general population. *American Journal of Occupational Therapy*, *70*(4), 1–10.

Bar-Shalita, T., & Cermak, S. A. (2020). Multi-sensory Responsiveness and Personality Traits Predict Daily Pain Sensitivity. *Frontiers in Integrative Neuroscience*, *13*, 77. https://doi.org/10.3389/fnint.2019.00077

Bar-Shalita, T., Granovsky, Y., Parush, S., & Weissman-Fogel, I. (2019). Sensory modulation disorder (SMD) and pain: A new perspective. *Frontiers in Integrative Neuroscience*, *13*(27), 1–10.

Bar-Shalita, T., Seltzer, Z., Vatine, J.-J., Yochman, A., & Parush, S. (2009). Development and psychometric properties of the Sensory Responsiveness Questionnaire (SRQ). *Disability and Rehabilitation*, *31*(3), 189–201.

Bonanno, G. A. (2004). Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? *American Psychologist*, *59*(1), 20.

Bovin, M. J., Marx, B. P., Weathers, F. W., Gallagher, M. W., Rodriguez, P., Schnurr, P. P., & Keane, T. M. (2016). Psychometric properties of the PTSD checklist for diagnostic and statistical manual of mental disorders–fifth edition (PCL-5) in veterans. *Psychological Assessment*, *28*(11), 1379–1391. http://dx.doi.org/10.1037/pas0000254

Carpenter, K. L. H., Baranek, G. T., Copeland, W. E., Compton, S., Zucker, N., Dawson, G., & Egger, H. L. (2019). Sensory Over-Responsivity: An Early Risk Factor for Anxiety and Behavioral Challenges in Young Children. *Journal of Abnormal Child Psychology*, *47*(6), 1075–1088. https://doi.org/10.1007/s10802-018-0502-y

Charny, S., Cao, G., Gafter, L., Bar-Shalita, T., & Lahav, Y. (2023). Sensory modulation and trauma-related symptoms during rocket attacks. *European Journal of Psychotraumatology*, *14*(2), 2213110. https://doi.org/10.1080/20008066.2023.2213110

Christiansen, D. M., & Berke, E. T. (2020). Gender- and Sex-Based Contributors to Sex Differences in PTSD. *Current Psychiatry Reports*, *22*(4), 19. https://doi.org/10.1007/s11920-020-1140-y

Davison, A. C., & Hinkley, D. V. (1997). *Bootstrap Methods and their Application*. Cambridge University Press.

Dokkedahl, S. B., Charny, S., & Lahav, Y. (2024). Testing previously proposed models of the Tonic Immobility Scale in a peritraumatic sample of Israeli civilians: Support for a three-factor model. *Psychological Trauma: Theory, Research, Practice, and Policy, 16*(1), 21-29. https://doi.org/10.1037/tra0001499

Dokkedahl, S. B., & Lahav, Y. (2023a). A prospective study of the mediating role of tonic immobility and peritraumatic dissociation on the 4 DSM-5 symptom clusters of posttraumatic stress disorder. *The Journal of Clinical Psychiatry*, *84*(4), 47881.

Dokkedahl, S. B., & Lahav, Y. (2023b). A prospective study of the mediating role of tonic immobility and peritraumatic dissociation on the 4 DSM-5 symptom clusters of posttraumatic stress disorder. *The Journal of Clinical Psychiatry, 84*(4), 47881.

Dunn, W., Little, L., Dean, E., Robertson, S., & Evans, B. (2016). The state of the science on sensory factors and their impact on daily life for children: A scoping review. *OTJR: Occupation, Participation and Health*, *36*(2_suppl), 3S-26S.

Engel-Yeger, B., Palgy-Levin, D., & Lev-Wiesel, R. (2013). The sensory profile of people with post-traumatic stress symptoms. *Occupational Therapy in Mental Health*, *29*(3), 266–278.

Engel-Yeger, B., & Rosenblum, S. (2021). Executive dysfunctions mediate between altered sensory processing and daily activity performance in older adults. *BMC Geriatrics, 21*(1), 1–10.

Fraser, K., MacKenzie, D., & Versnel, J. (2017). Complex Trauma in Children and Youth: A Scoping Review of Sensory-Based Interventions. *Occupational Therapy in Mental Health*, *33*(3), 199–216. https://doi.org/10.1080/0164212X.2016.1265475

Gelkopf, M., Lapid Pickman, L., Carlson, E. B., & Greene, T. (2019). The Dynamic Relations Among Peritraumatic Posttraumatic Stress Symptoms: An Experience Sampling Study During Wartime. *Journal of Traumatic Stress*, jts.22374. https://doi.org/10.1002/jts.22374

Gelkopf, M., Lapid Pickman, L., Grinapol, S., Werbeloff, N., Carlson, E. B., & Greene, T. (2017). Peritraumatic reaction courses during war: Gender, serious mental illness, and exposure. *Psychiatry*, *80*(4), 382–398.

Geraci, M., & Farcomeni, A. (2022). Mid-quantile regression for discrete responses. *Statistical Methods in Medical Research*, *31*(5), 821–838. https://doi.org/10.1177/09622802211060525

Harricharan, S., McKinnon, M. C., & Lanius, R. A. (2021). How Processing of Sensory Information From the Internal and External Worlds Shape the Perception and Engagement With the World in the Aftermath of Trauma: Implications for PTSD. *Frontiers in Neuroscience*, *15*, 360.

Hayes, A. F., & Rockwood, N. J. (2017). Regression-based statistical mediation and moderation analysis in clinical research: Observations, recommendations, and implementation. *Behaviour Research and Therapy*, *98*, 39–57. https://doi.org/10.1016/j.brat.2016.11.001

Hoffman, M. A., & Kruczek, T. (2011). A Bioecological Model of Mass Trauma: Individual, Community, and Societal Effects. *The Counseling Psychologist*, *39*(8), 1087–1127. https://doi.org/10.1177/0011000010397932

Joseph, R., Van Der Linde, J., & Franzsen, D. (2022). Sensory modulation dysfunction in child victims of trauma from four residential care sites in southern Gauteng, South Africa. *South African Journal of Occupational Therapy*, *52*(2), 46–55. https://doi.org/10.17159/2310-3833/2022/vol52n2a6

Joseph, R. Y., Casteleijn, D., Van Der Linde, J., & Franzsen, D. (2021). Sensory Modulation Dysfunction in Child Victims of Trauma: A Scoping Review. *Journal of Child & Adolescent Trauma*, *14*(4), 455–470. https://doi.org/10.1007/s40653-020-00333-x

Kimball, J. G. (2022). Sensory Modulation Challenges: One Missing Piece in the Diagnosis and Treatment of Veterans with PTSD. *Occupational Therapy in Mental Health*, 1–18. https://doi.org/10.1080/0164212X.2022.2131695

Koenker, R., & Machado, J. A. F. (1999). Goodness of Fit and Related Inference Processes for Quantile Regression. *Journal of the American Statistical Association*, *94*(448), 1296–1310. https://doi.org/10.1080/01621459.1999.10473882

Kolltveit, S., Lange-Nielsen, I. I., Thabet, A. A. M., Dyregrov, A., Pallesen, S., Johnsen, T. B., & Laberg, J. C. (2012). Risk factors for PTSD, anxiety, and depression among adolescents in gaza. *Journal of Traumatic Stress*, *25*(2), 164–170. https://doi.org/10.1002/jts.21680

Lapid Pickman, L., Greene, T., & Gelkopf, M. (2017). Sense of threat as a mediator of peritraumatic stress symptom development during wartime: An experience sampling study. *Journal of Traumatic Stress*, *30*(4), 372–380.

Leys, C., Klein, O., Dominicy, Y., & Ley, C. (2018). Detecting multivariate outliers: Use a robust variant of the Mahalanobis distance. *Journal of Experimental Social Psychology*, *74*(October 2017), 150–156. https://doi.org/10.1016/j.jesp.2017.09.011

Luxton, D. D., Skopp, N. A., & Maguen, S. (2010). Gender differences in depression and PTSD symptoms following combat exposure. *Depression and Anxiety*, *27*(11), 1027–1033. https://doi.org/10.1002/da.20730

Machado, J. A. F., & Santos Silva, J. M. C. (2005). Quantiles for counts. *Journal of the American Statistical Association*, *100*(472), 1226–1237. https://doi.org/10.1198/016214505000000330

Miller, L. J., Anzalone, M. E., Lane, S. J., Cermak, S. A., & Osten, E. T. (2007). Concept evolution in sensory integration: A proposed nosology for diagnosis. *American Journal of Occupational Therapy*, *61*(2), 135–140.

Morina, N., Stam, K., Pollet, T. V., & Priebe, S. (2018). Prevalence of depression and posttraumatic stress disorder in adult civilian survivors of war who stay in war-afflicted regions. A systematic review and meta-analysis of epidemiological studies. *Journal of Affective Disorders*, *239*, 328–338. https://doi.org/10.1016/j.jad.2018.07.027

Murthy, R. S., & Lakshminarayana, R. (2006). Mental health consequences of war: A brief review of research findings. *World Psychiatry: Official Journal of the World Psychiatric Association (WPA)*, *5*(1), 25–30.

Neria, Y., Besser, A., Kiper, D., & Westphal, M. (2010). A longitudinal study of posttraumatic stress disorder, depression, and generalized anxiety disorder in Israeli civilians exposed to war trauma. *Journal of Traumatic Stress*, *23*(3), 322–330.

Porges, S. W. (2009). The polyvagal theory: New insights into adaptive reactions of the autonomic nervous system. *Cleveland Clinic Journal of Medicine*, *76*(4 suppl 2), S86–S90. https://doi.org/10.3949/ccjm.76.s2.17

R Core Team. (2022). *R: A language and environment for statistical computing* (4.1.3). R Foundation for Statistical Computing.

Reynolds, K., Pietrzak, R. H., Mackenzie, C. S., Chou, K. L., & Sareen, J. (2016). Post-Traumatic Stress Disorder Across the Adult Lifespan: Findings From a Nationally Representative Survey. *The American Journal of Geriatric Psychiatry*, *24*(1), 81–93. https://doi.org/10.1016/j.jagp.2015.11.001

Schoemann, A. M., Boulton, A. J., & Short, S. D. (2017). Determining Power and Sample Size for Simple and Complex Mediation Models. *Social Psychological and Personality Science*, *8*(4), 379–386. https://doi.org/10.1177/1948550617715068

Sievers, B., Lee, C., Haslett, W., & Wheatley, T. (2019). A multi-sensory code for emotional arousal. *Proceedings of the Royal Society B: Biological Sciences*, *286*(1906), 20190513. https://doi.org/10.1098/rspb.2019.0513

Sommer, J. L., Reynolds, K., El-Gabalawy, R., Pietrzak, R. H., Mackenzie, C. S., Ceccarelli, L., Mota, N., & Sareen, J. (2021). Associations between physical health conditions and posttraumatic stress disorder according to age. *Aging & Mental Health*, *25*(2), 234–242. https://doi.org/10.1080/13607863.2019.1693969

Tabachnick, B. G., Fidell, L. S., & Ullman, J. B. (2007). *Using Multivariate Statistics* (7th ed.). Pearson. https://doi.org/10.4324/9781315181158-21

Warner, E., Koomar, J., Lary, B., & Cook, A. (2013). Can the body change the score? Application of sensory modulation principles in the treatment of traumatized adolescents in residential settings. *Journal of Family Violence*, *28*(7), 729–738.

Weathers, F., Litz, B., Keane, T., Palmieri, T., Marx, B. P., & Schnurr, P. (2013). *The ptsd checklist for dsm-5 (pcl-5).*

Yochman, A., & Pat-Horenczyk, R. (2020). Sensory modulation in children exposed to continuous traumatic stress. *Journal of Child & Adolescent Trauma*, *13*(1), 93–102.

Yuan, Y., & Mackinnin, D. P. (2014). Robust Mediation Analysis Based on Median Regression. *Psychological Methods*, *19*(1), 1–7. https://doi.org/10.1037/a0033820.Robust

Table 1.
Inter-correlations the study variables and PTSD symptoms (n=209)

Measure	1	2	3	4	5	6	7	8	9	10	11	12
1. Age	-											
2. Gender	.18**	-										
3. High sensory responsiveness (T1)	-.03	.17*	-									
4. Low sensory responsiveness (T1)	-.07	.11	.20**	-								
5. ETRS - Intrusion (T1)	-.25***	.23***	.20**	.06	-							
6. ETRS - Avoidance (T1)	-.15*	.12	.20**	-.01	.68***	-						
7. ETRS - Negative alterations in mood and cognitions (T1)	-.19**	.07	.23***	.02	.68***	.69***	-					
8. ETRS - Hyperarousal (T1)	-.28***	.18*	.21**	.05	.69***	.59***	.72***	-				
9. PTSD symptoms - Intrusion (T2)	-.12	.18*	.22**	.11	.67***	.61***	.56***	.49***	-			
10. PTSD symptoms - Avoidance (T2)	-.11	.14	.20**	.04	.56***	.55***	.55***	.43***	.81***	-		
12. PTSD symptoms - Negative alterations in mood and cognitions (T2)	-.15*	.03	.25***	.09	.51***	.57***	.61***	.37***	.76***	.78***	-	
12. PTSD symptoms - Hyperarousal (T2)	-.09	.11	.34***	.10	.60***	.54***	.55***	.49***	.81***	.72***	.80***	-
M/%	37.7	80.9	1.8	1.7	3.7	1.3	5.4	8.5	6.8	2.8	9.3	9.2
(SD)/category	10.4	Female	0.4	0.4	4.3	1.9	5.7	5.3	3.1	1.6	4.3	3.9

Note. ETRS= Early trauma-related symptoms; PTSD = Posttraumatic Stress Disorder * $p < .05$; ** $p < .01$; *** $p < .001$

Table 2.

Regression coefficients, standard errors, model summary information, and indirect effects for the mediation model between early trauma-related symptoms, sensory modulation and PTSD.

	Hyperarousal			
	T1 $\hat{\beta}(SE)$	**T2** $\hat{\beta}(SE)$		
Total effects				
High sensory responsiveness (T1)		2.6 (0.74)***		
Low sensory responsiveness (T1)		0.27 (0.77)		
Direct effects				
(Intercept)	9.54 (3.58)**	1.25 (1.63)		
Age	-0.18 (0.05)***	0.03 (0.02)		
Gender (Male)	-1.61 (1.13)	0.32 (0.47)		
Hyperarousal (T1)	-	0.28 (0.06)***		
High sensory responsiveness (T1)	3.05 (1.11)**	1.73 (0.59)**		
Low sensory responsiveness (T1)	-0.03 (1.31)	0.29 (0.65)		
R^2	11%	15%		
Indirect Effects		$\hat{\beta}$	**BootCI**	$Min\ \alpha$
HSR→Etrs1→Post2		0.87	[-0.03,1.46]	6%
LSR→ Etrs1→Post2		-0.01	[-0.57,1]	72%
	Intrusion			
	T1 $\hat{\beta}(SE)$	**T2** $\hat{\beta}(SE)$		
Total effects				
High sensory responsiveness (T1)		0.74 (0.35)*		
Low sensory responsiveness (T1)		-0.14 (0.31)		
Direct effects				
(Intercept)	2.04 (1.65)	4.65 (0.5)***		
Age	-0.06 (0.02)*	0.01 (0.01)		
Gender (Male)	-1.5 (0.41)***	0.02 (0.09)		
Intrusion (T1)	-	0.36 (0.05)***		
High sensory responsiveness (T1)	1.61 (0.65)*	0.16 (0.2)		
Low sensory responsiveness (T1)	-0.1 (0.66)	-0.11 (0.19)		
R^2	7%	25%		
Indirect Effects		$\hat{\beta}$	**BootCI**	$Min\ \alpha$
HSR→Etrs1→Post2		0.58	[0.07,1.05]	3%
LSR→ Etrs1→Post2		-0.04	[-0.34,0.51]	57%
	Avoidance[1]			
	T1	**T2**		
Total effects	$\hat{\beta}(SE)$	$\hat{\beta}(SE)$		
High sensory responsiveness (T1)		0.08 (0.09)		
Low sensory responsiveness (T1)		-0.04 (0.1)		
Direct effects				
(Intercept)	0.24 (0.94)	1.95 (0.27)***		
Age	-0.02 (0.01)	0 (0)		
Gender (Male)	-0.21 (0.26)	-0.07 (0.06)		

Avoidance (T1)		0.26 (0.12)*		
High sensory responsiveness (T1)	0.54 (0.39)	0.08 (0.09)		
Low sensory responsiveness (T1)	-0.05 (0.36)	-0.04 (0.1)		
R^2	0.02%	6%		
Indirect Effects		$\hat{\boldsymbol{\beta}}$	**BootCI**	$\boldsymbol{Min\ \alpha}$
HSR→Etrs1→Post2		0.14	[0.01,0.6]	3%
LSR→ Etrs1→Post2		-0.01	[-0.09,0.49]	37%
	Negative Alterations in mood and cognitions			
	T1 $\hat{\boldsymbol{\beta}}(\boldsymbol{SE})$	**T2** $\hat{\boldsymbol{\beta}}(\boldsymbol{SE})$		
Total effects				
High sensory responsiveness (T1)		1.09 (0.44)*		
Low sensory responsiveness (T1)		-0.24 (0.46)		
Direct effects				
(Intercept)	3.53 (2.16)	6.13 (0.68)***		
Age	-0.07 (0.04)*	0 (0.01)		
Gender (Male)	-0.33 (1.04)	0.18 (0.17)		
Negative alteration in mood and cognitions (T1)	-	0.3 (0.08)***		
High sensory responsiveness (T1)	2.36 (0.82)**	0.38 (0.26)		
Low sensory responsiveness (T1)	-0.76 (1.4)	-0.01 (0.21)		
R^2	5%	18%		
Indirect Effects		$\hat{\boldsymbol{\beta}}$	**BootCI**	$\boldsymbol{Min\ \alpha}$
HSR→Etrs1→Post2		0.71	[0.14,1.46]	1%
LSR→ Etrs1→Post2		-0.23	[-1.38,0.4]	26%

*p<0.05, **p<0.01, ***p<0.001; [1]Data is jittered; HSR=high sensory responsiveness, LSR=low sensory responsiveness, Etrs1= Early trauma-related symptoms cluster at T1, Post2=PTSD symptoms cluster at T2

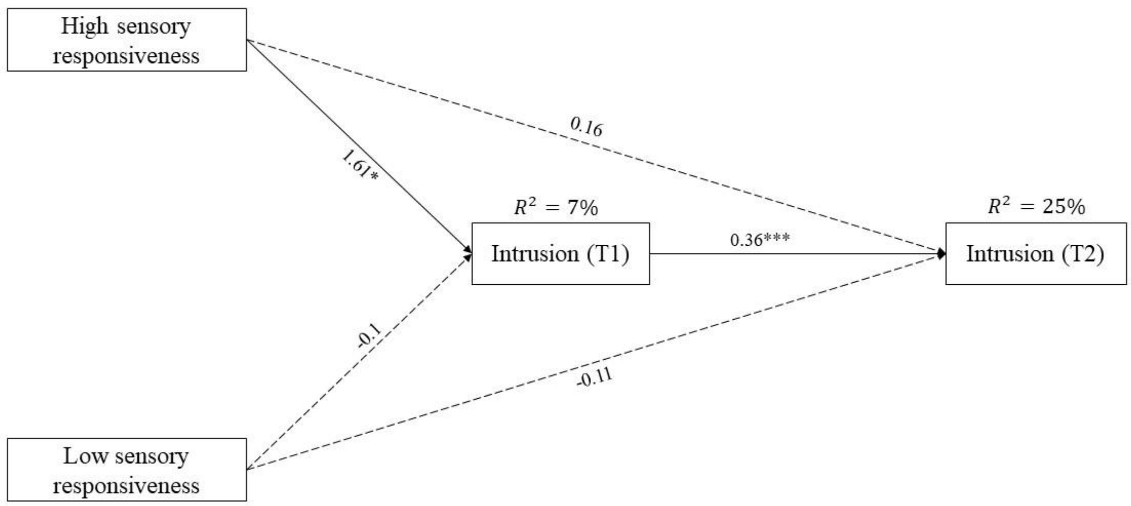

Figure 1. Implications of sensory responsiveness on intrusion PTSD symptoms through early trauma intrusion symptoms. Solid lines represent significant predictors. Values in brackets represent indirect effects. (*p<0.05, **p<0.01, ***p<0.001).

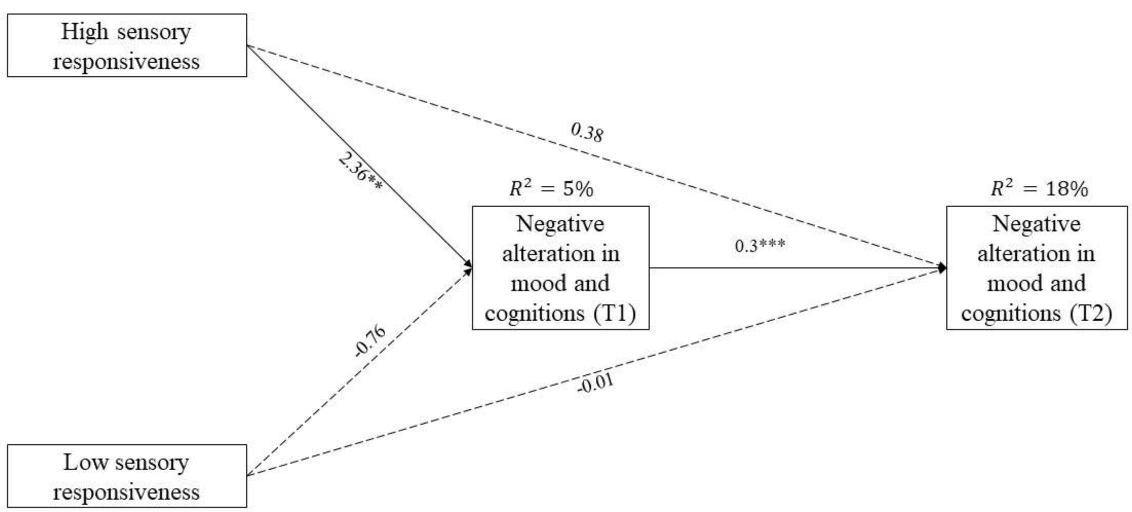

Figure 2. Implications of sensory responsiveness on negative alterations in mood and cognitions PTSD symptoms through early trauma negative alterations in mood and cognitions symptoms. Solid lines represent significant predictors. Values in brackets represent indirect effects. (*p<0.05, **p<0.01, ***p<0.001).

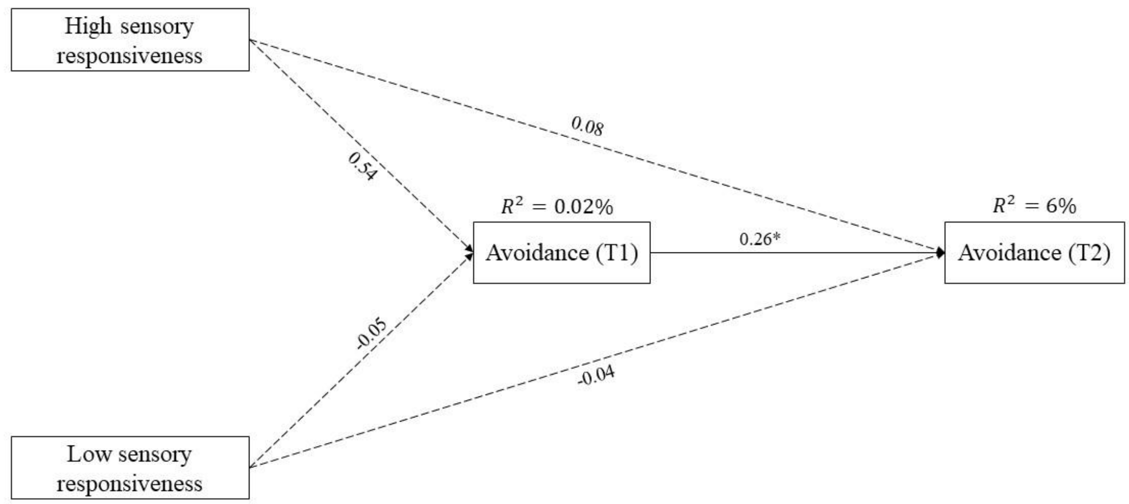

Figure 3. Implications of sensory responsiveness on avoidance PTSD symptoms through early trauma avoidance symptoms. Solid lines represent significant predictors. Values in brackets represent indirect effects. (*p<0.05, **p<0.01, ***p<0.001).